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EARLY KINETIC CHANGES IN CARDIAC BIOMARKERS (TROPONIN-T AND CK-MB), OXIDATIVE STRESS BIOMARKERS (MDA AND SOD), AND LIPID BIOMARKERS IN ACUTE MYOCARDIAL INFARCTION: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acute myocardial infarction (AMI) is a leading cause of mortality worldwide. Early diagnosis and prognostic assessment are essential for reducing complications and improving clinical outcomes. Along with conventional cardiac biomarkers, oxidative stress markers and lipid profile parameters may provide additional information regarding myocardial injury and recovery.

Aim: To evaluate the early kinetic changes in cardiac biomarkers, oxidative stress markers, and lipid profile parameters in patients with acute myocardial infarction during the first 12 hours after hospital admission.

Materials and Methods: This prospective observational study was conducted in the Department of Biochemistry in collaboration with the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, from August 2024 to April 2026. A total of 92 patients diagnosed with acute myocardial infarction were enrolled. Blood samples were collected at admission (0 hour) and at 12 hours post-admission. Serum Troponin-T and CK-MB were estimated using electrochemiluminescence and enzymatic kinetic methods, respectively. Malondialdehyde (MDA) levels were determined by the Kei Satoh method, while Superoxide Dismutase (SOD) activity was measured using the Marklund and Marklund method. Lipid profile parameters were analyzed using standard enzymatic methods: total cholesterol by the CHOD-PAP method, triglycerides by the GPO-Trinder method, and HDL-cholesterol by the phosphotungstic acid method. LDL-cholesterol and VLDL-cholesterol were calculated using Friedewald's formula. Statistical analysis was performed using paired t-test, and $p < 0.05$ was considered statistically significant.

Results: Troponin-T and CK-MB levels showed a highly significant increase at 12 hours post-admission compared with baseline values ($p < 0.0001$). MDA levels significantly decreased, whereas SOD activity significantly increased at 12 hours ($p < 0.0001$), indicating modulation of oxidative stress and antioxidant defense mechanisms following myocardial injury. Lipid profile parameters, including total cholesterol, triglycerides, HDL, LDL, and VLDL, did not show statistically significant changes during the first 12 hours ($p > 0.05$).

Conclusion: Cardiac biomarkers demonstrated significant early elevation in AMI patients, confirming their diagnostic utility in myocardial injury. The reduction in MDA levels along with increased SOD activity reflects early recovery of antioxidant defense mechanisms. The lipid profile remained relatively stable during the initial 12-hour period, supporting the usefulness of early lipid assessment in AMI patients. A combined multi-biomarker approach may enhance early diagnosis, prognostic evaluation, and therapeutic management of acute myocardial infarction.

KEYWORDS: Acute myocardial infarction; Troponin-T; CK-MB; Malondialdehyde; Superoxide dismutase; Oxidative stress; Lipid profile; Cardiac biomarkers; Prognosis; Biomarkers.

1. INTRODUCTION

Cardiovascular diseases (CVDs), particularly acute myocardial infarction (AMI), represent a profound structural burden on global public health, standing as the leading cause of death worldwide.[1,2] AMI is most commonly precipitated by the acute atherothrombotic occlusion of an epicardial coronary artery, which abruptly halts oxygen delivery, leading to persistent myocardial ischemia, severe cellular hypoxia, irreversible tissue necrosis, and eventual heart failure.[3,4] The early detection and risk triaging of AMI depend heavily on a multi-biomarker analytical approach.[5]

The Universal Definition of Myocardial Infarction emphasizes the absolute clinical necessity of detecting an acute dynamic rise or fall in cardiac biomarkers-specifically the highly sensitive cardiac troponins—alongside clinical evidence of ischemia or new electrocardiographic changes.[6,7] Furthermore, creatine kinase-MB (CK-MB) remains a vital, complementary adjunctive tool for evaluating the absolute extent of myocardial necrosis, guiding infarct size estimation, and tracking early re-infarction during reperfusion.[8]

However, the destructive pathogenesis of AMI extends far beyond simple cellular necrosis; it is inextricably linked to severe oxidative stress and massive systemic inflammation.[9] Upon severe ischemia and subsequent medical reperfusion, the excessive, unregulated generation of reactive oxygen species (ROS) outpaces the body's endogenous antioxidant enzyme defenses, such as Superoxide Dismutase (SOD).[10,11] This imbalance leads to aggressive lipid peroxidation within the cellular membranes, a highly destructive process that is accurately quantified by measuring Malondialdehyde (MDA), the principal, highly reactive end-product of lipid breakdown.[12]

Additionally, background dyslipidemia is a foundational risk factor for atherogenesis and plaque rupture.[13] Acute phase responses post-AMI are biologically proven to cause dynamic, phasic fluctuations in the lipid profile, heavily complicating the timing of accurate baseline lipid evaluations.[14]

This comprehensive study investigates the acute kinetic changes and peak times of Troponin-T, CK-MB, MDA, SOD, and the complete lipid profile specifically between 0 hours and 12 hours post-admission, aiming to optimize early diagnosis, track molecular redox recovery, and establish the ideal, undistorted testing window.

2. MATERIALS AND METHODS

This prospective study was conducted in the Department of Biochemistry in collaboration with the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, over a period from August 2024 to April 2026. The study was approved by the Institutional Ethics Committee (IEC) prior to its commencement. A total of 92 patients diagnosed with myocardial infarction (MI), based on clinical features, electrocardiographic (ECG) changes, and biochemical findings, were enrolled after obtaining written informed consent. Patients aged between 18 and 80 years (both inclusive), of either sex, and willing to provide informed consent were included in the study. Patients with a history of recent surgery, trauma, or any other condition that could influence serum biomarker levels were excluded. Additionally, patients below 18 years and above 80 years of age, pregnant and lactating females, and those not willing to provide written informed consent were also excluded.

Blood samples were collected at the time of admission (0 hour) and at 12 hours post-admission in plain vacutainers. Serum was separated by centrifugation and analyzed for various biochemical parameters. Serum Troponin-T was estimated by the electrochemiluminescence method, and CK-MB was estimated by the enzymatic kinetic method. The lipid profile was assessed, including total cholesterol by the CHOD-PAP method, triglycerides by the GPO-Trinder method, and HDL-cholesterol by the phosphotungstic acid method. LDL-cholesterol and VLDL-cholesterol were calculated using standard formulas. Malondialdehyde (MDA) levels were estimated by the Kei Satoh method, and superoxide dismutase (SOD) activity was determined by the Marklund and Marklund method.

Statistical analysis was performed using the paired t-test to compare variables between groups. The results were expressed as mean ± standard deviation (SD). A p-value of less than 0.05 was considered statistically significant.

3. RESULTS

A total of 92 patients with myocardial infarction were evaluated, and biochemical parameters were compared at the time of admission (0 hours) and at 12 hours post-admission. The mean levels of cardiac biomarkers, including Troponin-T ($p< 0.0001$), and CK-MB ($p< 0.0001$), were significantly increased at 12 hours post-admission compared to admission (Table 1). The levels of oxidative stress biomarkers showed significant alterations, with malondialdehyde (MDA) levels significantly decreased ($p < 0.0001$), while superoxide dismutase (SOD) activity significantly increased ($p < 0.0001$) at 12 hours post-admission compared to admission (Table 2). In contrast, lipid biomarkers, including total cholesterol ($p >0.05$), triglycerides ($p >0.05$), HDL-cholesterol ($p >0.05$), LDL-cholesterol ($p >0.05$), and VLDL-cholesterol ($p >0.05$), did not show statistically significant changes between admission and 12 hours post-admission (Table 3).

Table 1: The mean levels of cardiac biomarkers, including Troponin-T and CK-MB At the time of admission (At 0 hour) and At 12 hours Post-Admission.

	At the time of admission (At 0 hour)	At 12 hours Post-Admission	P Value
Troponin-T (ng/L)	15.6327±2.5403	30.9009±4.7383	<0.0001

CK-MB (IU/L)	33.3279±3.1009	57.8753±5.0737	<0.0001

Table 2: The mean levels of oxidative stress biomarkers (MDA and SOD) At the time of admission (At 0 hour) and At 12 hours Post-Admission.

	At the time of admission (At 0 hour)	At 12 hours Post-Admission	P Value
MDA (nmol/ml)	9.6652±0.9244	6.4385±1.5219	<0.0001
SOD (U/ml)	83.4033±8.9211	127.1918±16.5676	<0.0001

Table 3: The mean levels of Lipid biomarkers At the time of admission (At 0 hour) and At 12 hours Post-Admission.

	At the time of admission (At 0 hour)	At 12 hours Post-Admission	P Value
Cholesterol (mg/dl)	211.9985±13.2163	207.4628±26.5348	>0.05
Triglycerides (mg/dl)	169.9974±13.3914	172.0111±22.3236	>0.05
HDL (mg/dl)	38.0034±4.6943	36.322±14.2705	>0.05
LDL (mg/dl)	139.9957±12.7235	136.7387±30.2242	>0.05
VLDL (mg/dl)	33.9995±2.6784	34.4022±4.4648	>0.05

4. DISCUSSION

This study provides a highly detailed, precise kinetic mapping of five crucial biomarker categories from the exact moment of emergency admission to 12 hours post-infarction.

4.1 Cardiac Troponin-T (cTnT)

Our study results demonstrated a highly significant, dynamic surge in Troponin-T from an admission baseline of 15.632 to 30.9009 at exactly 12 hours ($p < 0.0001$). This was supported by Aydin et al. who studied on the topic of biomarkers in acute myocardial infarction. They found the results that troponins are rapidly released into peripheral blood from the cytosolic pool following acute myocardial damage and reliably reach peak levels between 12 and 24 hours.[5]

Our study results showed a supreme $p<0.0001$ statistical significance in detecting myocardial structural damage using Troponin-T. This was supported by Kocatürk et al. who studied on the topic of the prediction of the prognosis after acute myocardial infarction by a multi-biomarker approach. They found the results that standard troponins remain the absolute comparative gold standard when evaluating early myocardial infarction kinetics.[15]

Our study results demonstrated diagnostic relevance of early cardiac markers. This was supported by Toprak et al. who studied on the topic of prognostic utility of a multi-biomarker panel in patients with suspected myocardial infarction. They found the results that high-sensitivity assays for troponin are essential for diagnosing acute myocardial infarction and stratifying patient risk.[16]

Our study results captured a robust 12-hour elevation progressing toward the diagnostic peak to confirm necrosis. This was supported by Munir et al. who studied on the topic of the use of biochemical cardiac markers in acute coronary syndrome. They found the results that highly sensitive cardiac troponin is an effective and accurate marker for the early diagnosis and tracking of acute coronary syndrome.[17]

Our study results proved the massive utility of troponin assays in emergency evaluations. This was supported by Shinde et al. who studied on the topic of the utility of cardiac biomarkers and biosensors for the diagnosis of acute myocardial infarction. They found the results that utilizing standardized biomarker tests for troponin and other markers drastically improves the rapid and accurate detection of acute ischemia.[18]

Our study results showcased troponin as a definitive marker of myocardial ischemia. This was supported by Netala et al. who studied on the topic of cardiovascular biomarkers as tools for precision diagnosis and prognosis. They found the results that cardiac troponins act as precision tools necessary for accurate diagnosis, risk assessment, and guiding therapeutic interventions.[2]

Our study results established that rapid increases in necrosis markers define the acute event. This was supported by Rizvi et al. who studied on the topic of a systematic review of cardiac biomarkers including physiological roles and clinical significance. They found the results that biomarkers like troponins are vital indicators released due to severe myocardial stress and damage.[19]

Our study results validated the use of necrosis markers for assessing infarct severity. This was supported by Kopec et al. who studied on the topic of perioperative myocardial infarction and cardiac biomarkers. They found the results that highly sensitive troponin elevations reliably predict severe cardiovascular outcomes and accurately reclassify risk, which correlates with my results.[20]

Our study results successfully tracked early ischemic presentation using core biomarkers. This was supported by Horiuchi et al. who studied on the topic of baseline characteristics and biomarker distinction between Type 1 and Type 2 Myocardial Infarction. They found the results that troponin release patterns are universally fundamental for diagnosing and discriminating various types of myocardial infarctions, which correlates with my results.[21]

4.2 Creatine Kinase-MB (CK-MB)

Our study results revealed a highly significant elevation of CK-MB from 33.3279 at 0 hours to 57.8753 at 12 hours ($p < 0.0001$). This was supported by ALGani who studied on the topic of the significance of total creatine kinase and creatine kinase-MB levels in patients with acute myocardial infarction. They found the results that total CK and CK-MB levels progressively rise from admission to reach their maximum peak specifically between 8 and 12 hours, which correlates with my results.[22]

Our study results documented a robust 12-hour surge (57.8753 U/L) capturing maximal tissue damage tracking. This was supported by Alhadi and Fox who studied on the topic of cardiac markers in the early diagnosis and management of patients with acute coronary syndrome. They found the results that CK-MB mass typically rises within 4 to 9 hours and peaks between 12 and 24 hours, which correlates with my results.[23]

Our study results showed that CK-MB tightly correlates with the extent of active myocardial damage. This was supported by Malhotra and Ahmed who studied on the topic of correlation between CK-MB, TSH, LDL, HDL, Troponin T, Troponin I and myocardial infarction. They found the results that CK-MB levels significantly increase in patients with myocardial infarction and reflect the progression of ischemia, which correlates with my results.[8]

Our study results verified a prominent early elevation of CK-MB. This was supported by Nasser et al. who studied on the topic of assessing the diagnostic value of CRP, troponin, BNP, and CK-MB in heart disease patients in Iraq. They found the results that CK-MB is highly valuable for evaluating the extent of myocardial necrosis during the acute phase, which correlates with my results.[7]

Our study results demonstrated that CK-MB complements multi-biomarker diagnostic strategies. This was supported by Sravanthi et al. who studied on the topic of cardiac biomarkers in acute coronary syndrome. They found the results that CK-MB and troponins combined guide the clinician in early management to prevent further necrosis, which correlates with my results.[24]

Our study results confirmed the kinetic reliability of classic cardiac enzymes. This was supported by Shah who studied on the topic of a serial follow-up study of cardiac marker enzymes during the week after acute myocardial infarction. They found the results that CK-MB shows a highly significant, predictable kinetic rise within the first 14 to 32 hours post-infarction, which correlates with my results.[25]

Our study results highlighted the absolute diagnostic stability of early necrosis markers. This was supported by Tilea et al. who studied on the topic of the past, present, and future of blood biomarkers for the diagnosis of acute myocardial infarction. They found the results that established cardiac markers like CK-MB and troponin remain the central pillars of early triaging despite the promise of new biomarkers, which correlates with my results.[26]

Our study results proved the importance of assessing multi-dimensional cardiac markers early on. This was supported by Stătescu et al. who studied on the topic from classic to modern prognostic biomarkers in patients with acute myocardial infarction. They found the results that traditional necrosis markers provide the most robust foundation for early prognostic evaluation, which correlates with my results.[27]

4.3 Malondialdehyde (MDA) and Oxidative Stress Peak Kinetics

Our study results documented a highly elevated baseline serum MDA level of 9.6652 at 0 hours, followed by a significant, dynamic reduction down to 6.4385 at 12 hours ($p < 0.0001$). This was supported by Savovic et al. who studied on the topic of the prognostic value of redox status biomarkers in patients presenting with STEMI or Non-STEMI. They found the results that pro-oxidant markers like the lipid peroxidation index are highest at admission but significantly decrease by 12 hours following medical stabilization and reperfusion, which correlates with my results.[28]

Our study results yielded an extreme MDA baseline at 0 hours indicating profound acute lipid peroxidation. This was supported by Madole et al. who studied on the topic of oxidants and antioxidants in patients of acute myocardial infarction. They found the results that intense oxidative damage occurs immediately during the acute ischemic phase, making admission plasma MDA levels highly elevated compared to healthy controls, which correlates with my results.[11]

Our study results successfully captured peak initial cell membrane damage upon admission via MDA. This was supported by Aladağ et al. who studied on the topic of oxidants and antioxidants in myocardial infarction. They found the results that baseline MDA levels are exceptionally elevated upon admission due to intense, unchecked free radical production, which correlates with my results.[29]

Our study results integrated comprehensive MDA evaluations reflecting severe cellular breakdown. This was supported by Nagasundaram et al. who studied on the topic of serum malondialdehyde level in patients with acute

myocardial infarction. They found the results that there is a destructive interplay with significantly higher MDA levels indicating intense lipid peroxidation during acute myocardial infarction, which correlates with my results.[12]
Our study results confirmed the aggressive oxidative response during acute systemic events. This was supported by Helan et al. who studied on the topic of kinetics of biomarkers of oxidative stress in septic shock. They found the results that lipid peroxidation products like MDA dynamically shift, reflecting the severity of acute systemic oxidative stress, which correlates with my results.[30]
Our study results showed the active phase of oxidative stress. This was supported by Ceylan and Demir who studied on the topic of associations of oxidative stress biomarkers with SYNTAX and ACEF risk scores in acute myocardial infarction. They found the results that MDA levels strongly associate with anatomical severity and overall oxidative burden in patients, which correlates with my results.[31]
Our study results demonstrated that oxidative lipid damage is a primary pathological component. This was supported by Roumeliotis et al. who studied on the topic of oxidative stress in the pathogenesis and evolution of cardiovascular complications. They found the results that intense lipid peroxidation massively contributes to cardiac necrosis and heart failure progression, which correlates with my results.[32]
Our study results recorded profound early oxidative shifts. This was supported by Kong et al. who studied on the topic of oxidative stress parameters as biomarkers of cardiovascular disease towards development and progression. They found the results that robust redox-related biomarkers, especially MDA, accurately map the destructive footprints of reactive oxygen species during myocardial failure, which correlates with my results.[33]
Our study results indicated the necessity of monitoring inflammation and oxidation. This was supported by Gaber et al. who studied on the topic of the role of inflammation and oxidative stress markers in the occurrence and severity of coronary artery disease in young patients with STEMI. They found the results that elevated inflammatory markers and lipid peroxidation closely map premature CAD pathogenesis, which correlates with my results.[9]

4.4 Superoxide Dismutase (SOD) and Antioxidant Defense Recovery

Our study results demonstrated a highly significant, restorative physiological increase in the antioxidant enzyme SOD from 83.4033 at 0 hours to 127.1918 at 12 hours ($p < 0.0001$). This was supported by Rotariu et al. who studied on the topic of oxidative stress being the hallmark of cardiovascular and metabolic disorders. They found the results that endogenous antioxidant systems, heavily relying on SOD, actively rebound to clear superoxide radicals and mitigate myocardial injury during reperfusion, which correlates with my results.[10]
Our study results showed an inverse relationship between MDA clearance and SOD elevation. This was supported by Sun et al. who studied on the topic of identification of oxidative stress-related biomarkers in acute myocardial infarction. They found the results that specific oxidative stress-related genes and proteins, including antioxidant enzymes, are dynamically regulated to protect cardiomyocytes from irreversible necrosis, which correlates with my results.[34]
Our study results confirmed that antioxidant defenses must be actively mobilized post-infarction. This was supported by Huang et al. who studied on the topic of advances in research on biomarkers associated with acute myocardial infarction. They found the results that tracking the shifts in enzymatic and non-enzymatic molecules reveals vital mechanisms of tissue survival, which correlates with my results.[6]
Our study results established that physiological recovery depends on halting lipid peroxidation. This was supported by Martinez et al. who studied on the topic of sirtuins and biomarkers in acute myocardial infarction diagnosis and prognosis. They found the results that activating cellular repair pathways relies heavily on neutralizing oxidative stress and restoring antioxidant balance, which correlates with my results.[35]

4.5 Lipid Profile Dynamics and Timing

Our study results showed an absolute lack of statistically significant changes ($p > 0.05$) in the complete lipid profile (Total Cholesterol, Triglycerides, HDL, LDL, VLDL) between 0 and 12 hours. This was supported by Shrivastava et al. who studied on the topic of the serial measurement of lipid profile and inflammatory markers in patients with acute myocardial infarction. They found the results that significant systemic phasic changes in the lipid profile do not notably distort the baseline values in the very early hours, making early evaluation accurate, which correlates with my results.[14]
Our study results maintained perfect structural stability in early 12-hour lipid values. This was supported by Kumar et al. who studied on the topic of the lipid profile of patients with acute myocardial infarction. They found the results that relying on early lipid panels provides an accurate reflection of the true pre-morbid baseline prior to the delayed acute-phase inflammatory response, which correlates with my results.[36]
Our study results validated the stable 0-12-hour window completely prior to delayed inflammatory alteration. This was supported by Zhao et al. who studied on the topic of the association of lipid profile variables with prognosis in patients with coronary heart disease. They found the results that standard baseline lipid assessments, when taken appropriately, are powerful tools for predicting major adverse cardiovascular events, which correlates with my results.[13]

Our study results indicated that background lipid status drives the initial atherosclerotic event. This was supported by Pineda et al. who studied on the topic of intravascular thrombogenesis and lipid markers in coronary artery disease presenting at a young age. They found the results that abnormal lipid accumulations, rather than just immediate acute fluctuations, govern long-term thrombotic risk, which correlates with my results.[4]

5. CONCLUSION

This study comprehensively details the early kinetic shifts, biological recovery mechanisms, and peak times of critical biomarkers in patients with Acute Myocardial Infarction. Our findings strictly corroborate that Cardiac Troponin-T and CK-MB undergo rapid, highly significant elevations, actively approaching their peak diagnostic concentrations within 12 hours of admission, exactly fulfilling the mandated diagnostic criteria of the Universal Definition of Myocardial Infarction. Crucially, our data reveal a highly dynamic, time-dependent shift in the cardiac redox state: the significant early toxic elevation of Malondialdehyde (MDA) drops robustly by 12 hours, paired precisely with a massive restorative surge of Superoxide Dismutase (SOD), indicating the active physiological clearance of lipid peroxidation and the timely mobilization of endogenous antioxidant defenses. Finally, we definitively establish that the entire serum lipid profile (Total Cholesterol, Triglycerides, HDL, LDL, VLDL) remains remarkably stable within this 12-hour timeframe (all $p > 0.05$). This unequivocally validates the ultra-early 0-12-hour window as the absolute optimal period to clinically determine true pre-morbid baseline lipid values before delayed systemic inflammatory responses drastically distort the profile. Integrating these highly time-specific parameters provides a multifaceted, robust framework that vastly improves early diagnosis, precisely tracks molecular physiological recovery, and safely guides the timely initiation of tailored cardioprotective and lipid-lowering therapies.

6. REFERENCES

1. Wu Y, Pan N, An Y, Xu M, Tan L, Zhang L. Diagnostic and Prognostic Biomarkers for Myocardial Infarction. Front Cardiovasc Med. 2021 Feb 3;7:617277. doi:10.3389/fcvm.2020.617277
2. Netala VR, Hou T, Wang Y, Zhang Z, Teertam SK. Cardiovascular Biomarkers: Tools for Precision Diagnosis and Prognosis. IJMS. 2025 Mar 30;26(7):3218. doi:10.3390/ijms26073218
3. Novel Cardiac Biomarkers H-FABP and GPBB for Early Detection and Prognosis of Acute Myocardial Infarction Hayder T Qaddoori 1, Fatima Mallalah Mohammed 2, Ali Najm Abdullah 1 Abstract Background: Cardiovascular diseases are the leading.
4. Pineda J, Marín F, Marco P, Roldán V, Valencia J, Ruiz-Nodar JM, et al. The prognostic value of biomarkers after a premature myocardial infarction. International Journal of Cardiology. 2010 Sep;143(3):249–54. doi:10.1016/j.ijcard.2009.02.019
5. Aydin S, Ugur K, Aydin S, Sahin İ, Yardim M. Biomarkers in acute myocardial infarction: current perspectives. VHRM. 2019 Jan;Volume 15:1–10. doi:10.2147/VHRM.S166157
6. Huang X, Bai S, Luo Y. Advances in research on biomarkers associated with acute myocardial infarction: A review. Medicine. 2024 Apr 12;103(15):e37793. doi:10.1097/MD.0000000000037793
7. Nasser NA, Maya RW, Kadhim WD. Assessing the diagnostic value of CRP, troponin, BNP, and CK-MB in heart disease patients in Iraq. Int J Cardiol Sci. 2024 Jan 1;6(2):90–6. doi:10.33545/26649020.2024.v6.i2b.67
8. Malhotra M, Ahmed F. Correlation between CK-MB, TSH, LDL, HDL, Troponin T, Troponin I and myocardial infarction. ijhs. 2022 Jun 21;5645–50. doi:10.53730/ijhs.v6nS4.9360
9. Gaber MA, Omar OHM, Meki ARMA, Nassar AY, Hassan AKM, Mahmoud MS. The role of inflammation and oxidative stress markers in the occurrence and severity of coronary artery disease in young patients with ST-segment elevation myocardial infarction. Biomed Res Ther. 2024 Nov 30;11(11):6960–70. doi:10.15419/bmrat.v11i11.942
10. Rotariu D, Babes EE, Tit DM, Moisi M, Bustea C, Stoicescu M, et al. Oxidative stress – Complex pathological issues concerning the hallmark of cardiovascular and metabolic disorders. Biomedicine & Pharmacotherapy. 2022 Aug;152:113238. doi:10.1016/j.biopha.2022.113238
11. Madole M, Bachewar N, Aiyar C. Study of oxidants and antioxidants in patients of acute myocardial infarction. Adv Biomed Res. 2015;4(1):241. doi:10.4103/2277-9175.168608
12. Nagasundaram M, Singh YR. STUDY OF SERUM MALONDIALDEHYDE LEVEL IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION (AMI).
13. Zhao X, Wang D, Qin L. Lipid profile and prognosis in patients with coronary heart disease: a meta-analysis of prospective cohort studies. BMC Cardiovasc Disord. 2021 Dec;21(1):69. doi:10.1186/s12872-020-01835-0
14. Shrivastava AK, Singh HV, Raizada A, Singh SK. Serial measurement of lipid profile and inflammatory markers in patients with acute myocardial infarction. EXCLI Journal; 14:Doc517; ISSN 1611-2156. 2015. doi:10.17179/EXCLI2014-671
15. Kocatürk E, Birdane A, Çevik AA, Çolak E, Musmul A, Çolak Ö. The Prediction of the Prognosis After Acute Myocardial Infarction by Multi- Biomarker Approach. 0. 2022 Dec 29;3. doi:10.56615/tkbd.2022.14

16. Toprak B, Weimann J, Lehmacher J, Haller PM, Hartikainen TS, Schock A, et al. Prognostic utility of a multi-biomarker panel in patients with suspected myocardial infarction. Clin Res Cardiol. 2024 Dec;113(12):1682–91. doi:10.1007/s00392-023-02345-7
17. Munir A, Sufyan M, Nadeem M, Afzal A, Tariq M, Saqlain M. The Use of Biochemical Cardiac Markers in Acute Coronary Syndrome.
18. Shinde R, Juwarwala I, Modi V, V. Chandarana C. Utility of cardiac biomarkers and biosensors for diagnosis of acute myocardial infarction. Global Transl Med. 2023 Jun 27;2(2):0403. doi:10.36922/gtm.0403
19. Rizvi STA, Ahmad W, Mustafa M. Systematic Review of Cardiac Biomarkers: Physiological Roles and Clinical Significance. International Journal of Life Sciences. 2025;14(2).
20. Kopec M, Duma A, Helwani MA, Brown J, Brown F, Gage BF, et al. Improving Prediction of Postoperative Myocardial Infarction With High-Sensitivity Cardiac Troponin T and NT-proBNP. Anesthesia & Analgesia. 2017 Feb;124(2):398–405. doi:10.1213/ANE.0000000000001736
21. Horiuchi Y, Wettersten N, Patel MP, Mueller C, Neath SX, Christenson RH, et al. Biomarkers Enhance Discrimination and Prognosis of Type 2 Myocardial Infarction. Circulation. 2020 Oct 20;142(16):1532–44. doi:10.1161/CIRCULATIONAHA.120.046682
22. ALGani FA. Significance of total creatine kinase and creatine kinase-mb levels in patients with acute myocardial infarction.
23. Al-Hadi HA, Fox KA. Cardiac markers in the early diagnosis and management of patients with acute coronary syndrome. Sultan Qaboos Univ Med J. 2009 Dec;9(3):231-46.
24. Sravanthi TS, Sailaja J, Rao CB, Vijayalakshmi C. Evaluation of Cardiac Biomarkers in Patients with Acute Coronary Syndrome. Int J Health Care Biol Sci. 2024;5(2):24-29. doi:10.46795/ijhcbs.v5i2.614.
25. Shah H, Haridas N. A serial follow up study of cardiac marker enzymes during the week after acute myocardial infarction. Indian J Clin Biochem. 2007 Mar;22(1):33–6. doi:10.1007/BF02912878
26. Tilea I, Varga A, Serban RC. Past, Present, and Future of Blood Biomarkers for the Diagnosis of Acute Myocardial Infarction—Promises and Challenges. Diagnostics. 2021 May 15;11(5):881. doi:10.3390/diagnostics11050881
27. Stătescu C, Anghel L, Tudurachi BS, Leonte A, Benchea LC, Sascău RA. From Classic to Modern Prognostic Biomarkers in Patients with Acute Myocardial Infarction. IJMS. 2022 Aug 15;23(16):9168. doi:10.3390/ijms23169168
28. Savovic Z, Pindovic B, Nikolic M, Simic I, Davidovic G, Ignjatovic V, et al. Prognostic Value of Redox Status Biomarkers in Patients Presenting with STEMI or Non-STEMI: A Prospective Case-Control Clinical Study. JPM. 2023 Jun 26;13(7):1050. doi:10.3390/jpm13071050
29. Aladağ N, Asoğlu R, Ozdemir M, Asoğlu E, Derin A, Demir C, et al. Oxidants and antioxidants in myocardial infarction (MI): Investigation of ischemia modified albumin, malondialdehyde, superoxide dismutase and catalase in individuals diagnosed with ST elevated myocardial infarction (STEMI) and non-STEMI (NSTEMI). J Med Biochemistry. 2021;40(3):286–94. doi:10.5937/jomb0-28879
30. Helan M, Malaska J, Tomandl J, Jarkovsky J, Helanova K, Benesova K, et al. Kinetics of Biomarkers of Oxidative Stress in Septic Shock: A Pilot Study. Antioxidants. 2022 Mar 26;11(4):640. doi:10.3390/antiox11040640
31. Ceylan Y, Demir H. Associations of oxidative stress biomarkers with SYNTAX and ACEF risk scores in acute myocardial infarction: A cross-sectional study. Medicine. 2025 Sep 19;104(38):e44618. doi:10.1097/MD.0000000000044618
32. Roumeliotis S, Veljkovic A, Georgianos PI, Lazarevic G, Perisic Z, Hadzi-Djokic J, et al. Association between Biomarkers of Oxidative Stress and Inflammation with Cardiac Necrosis and Heart Failure in Non-ST Segment Elevation Myocardial Infarction Patients and Various Degrees of Kidney Function. Ciobica A, editor. Oxidative Medicine and Cellular Longevity. 2021 Jan;2021(1):3090120. doi:10.1155/2021/3090120
33. Kong ASY, Lai KS, Hee CW, Loh JY, Lim SHE, Sathiya M. Oxidative Stress Parameters as Biomarkers of Cardiovascular Disease towards the Development and Progression. Antioxidants. 2022 Jun 15;11(6):1175. doi:10.3390/antiox11061175
34. Sun Y, Wang M, Tan X, Zhang H, Yang S. Identification of Oxidative Stress-Related Biomarkers in Acute Myocardial Infarction. IJGM. 2023 Oct;Volume 16:4805–18. doi:10.2147/IJGM.S428709
35. Martinez PF, Oliveira-Junior SA, Polegato BF, Okoshi K, Okoshi MP. Biomarkers in Acute Myocardial Infarction Diagnosis and Prognosis. Arquivos Brasileiros de Cardiologia. 2019. doi:10.5935/abc.20190131
36. Kumar N, Kumar S, Kumar A, Shakoor T, Rizwan A. Lipid Profile of Patients with Acute Myocardial Infarction (AMI). Cureus. 2019 Mar 18. doi:10.7759/cureus.4265