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BIOCHEMICAL MARKERS IN ACUTE MYOCARDIAL INFARCTION PATIENTS WITH AND WITHOUT DIABETES MELLITUS: A COMPARATIVE STUDY

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ABSTRACT

Introduction: Acute myocardial infarction (AMI) is a leading cause of death, and diabetes mellitus is one of its strongest risk factors.

Aim: To compare biochemical markers, namely homocysteine, glycemic parameters, cardiac markers (troponin I, CK-MB, AST) and lipid profile, in AMI patients with and without diabetes mellitus.

Methods: A total of 94 AMI patients were studied: 50 with diabetes and 44 without.

Results: Mean age was similar in the two groups (53.2 ± 17.8 vs 54.6 ± 18.6 years), as was the sex distribution (66.0% vs 61.4% male). As expected, random blood sugar (237.6 ± 65.4 vs 111.2 ± 18.1 mg/dL) and HbA1c (9.8 ± 1.9 vs 5.2 ± 0.3%) were markedly higher in diabetic patients (both $P < 0.001$). Mean serum homocysteine was more than 80% higher in diabetic patients (25.3 ± 7.2 vs 13.8 ± 4.6 µmol/L, $P < 0.001$); hyperhomocysteinemia (≥15 µmol/L) was present in 84% of diabetic and 23% of non-diabetic patients, and severe hyperhomocysteinemia (≥30 µmol/L) in 36% and 5%, respectively (both $P < 0.001$). CK-MB (211.4 ± 141.8 vs 179.2 ± 108.6 U/L, $P = 0.024$) and AST (187.6 ± 109.8 vs 156.8 ± 101.4 U/L, $P = 0.034$) were higher in diabetic patients, whereas mean troponin I was 12.27 ± 16.68 ng/mL in diabetic and 14.15 ± 17.11 ng/mL in non-diabetic patients.

Conclusion: These findings indicate that AMI patients with diabetes carry a heavier burden of hyperglycemia, hyperhomocysteinemia and enzymatic evidence of myocardial injury than those without diabetes. Homocysteine estimation alongside conventional cardiac markers may help refine risk assessment in diabetic AMI patients, although prospective studies are needed to establish its prognostic value.

KEYWORDS: Acute myocardial infarction; Diabetes mellitus; Homocysteine; Troponin I; CK-MB; Biochemical markers.

INTRODUCTION

Cardiovascular diseases are the leading cause of death worldwide and in India, where coronary artery disease also tends to present at a younger age than in Western populations [1, 2]. Acute myocardial infarction (AMI) results from sudden interruption of coronary blood flow with myocardial ischemia and irreversible myocyte injury, and its diagnosis rests on a rise and fall of cardiac biomarkers in the appropriate clinical setting [3].

Diabetes mellitus is a major and independent risk factor for AMI. Diabetic patients have a risk of coronary events approaching that of non-diabetic patients with a previous infarction [4], and they have higher mortality after acute coronary syndromes [5]. Chronic hyperglycemia promotes endothelial dysfunction, oxidative stress, inflammation and accelerated atherosclerosis [6, 7]. Hyperglycemia at the time of infarction also carries an adverse prognosis in patients with and without diabetes [8].

Cardiac troponin I is the preferred marker of myocardial necrosis because of its sensitivity and specificity [3, 9]. Creatine kinase-MB (CK-MB) and aspartate aminotransferase (AST) are less specific but rise in infarction and are used to gauge the extent of myocardial damage [10, 11]. Dyslipidemia is characteristic of diabetes and further increases coronary risk [12].

Homocysteine, a sulfur-containing amino acid formed during methionine metabolism, has emerged as a risk marker for vascular disease. Meta-analyses of observational studies link higher homocysteine concentrations to ischemic heart disease [13, 14], and proposed mechanisms include endothelial dysfunction, oxidative stress and a prothrombotic state [15, 16]. Homocysteine concentrations are frequently raised in type 2 diabetes, and the combination of hyperhomocysteinemia and diabetes appears to carry a particularly high mortality risk [17, 18].

Few studies have compared homocysteine, glycemic indices, cardiac markers and lipids together in AMI patients with and without diabetes. The aim of the present study was therefore to measure and compare homocysteine, random blood sugar (RBS), glycated hemoglobin (HbA1c), lipid profile, troponin I, CK-MB and AST in AMI patients with and without diabetes mellitus.

MATERIAL AND METHODS

Study place: Tertiary care center
Study design: Cross sectional study
Study duration: 18 months
Study population: All the patients admitted in ICU for management of MI.
Study subjects: 94 Sample of MI patients

Study Setting

This was a hospital-based comparative study conducted in the Bharati Vidyapeeth (Deemed to be University) Medical College & Hospital, Sangli, Maharashtra, India. The study protocol was approved by the Institutional Ethics Committee BV(DU)MC&H/Sangli/IEC/Dissertation2023-24/543 Dated: 04/04/2024 and written informed consent was obtained from all participants.

Participants

Ninety-four patients with AMI were included, 50 with diabetes mellitus (diabetic group) and 44 without (non-diabetic group). AMI was diagnosed according to the Fourth Universal Definition of Myocardial Infarction [3]. Diabetes was defined as RBS ≥ 126 and and/or HbA1c ≥ 6.5% detected on more than one occasion as per ADA criteria.

Inclusion criteria: Subjects of all ages and both gender with of AMI were included. AMI diagnosis was based on history of chest pain, ECG changes and elevated cardiac enzymes. Diabetic and non-diabetic MI patients within 24 hours of diagnosis will be included in this study.

Exclusion criteria: Subjects who have the history congestive cardiac failure (CCF), valvular heart disease, patients with sever co-morbid condition (percutaneous coronary intervention, bypass grafting), patients already taking folic acid, vit B12 and vit B6 will be excluded from this.

Sample Collection and Biochemical Analysis

Venous blood was collected [timing relative to symptom onset]. Troponin I, CK-MB, AST, RBS, lipid profile (total cholesterol, triglycerides, LDL, HDL), HbA1c and serum homocysteine were measured on [analyzer, methods and kit manufacturers]. Reference ranges are given in the tables. Hyperhomocysteinemia was defined as homocysteine ≥ 15 μmol/L and severe hyperhomocysteinemia as ≥ 30 μmol/L.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation and categorical variables as number (percentage). The two groups were compared with independent samples t-test for continuous variables and the chi-square test for categorical variables. A P value < 0.05 was considered statistically significant. Analyses were done with GraphPad prism 5.0.

RESULTS

Characteristics of the study population

The mean age of the 94 patients was 53.8 ± 18.2 years (range 20-94 years). Age and sex distribution did not differ significantly between diabetic and non-diabetic patients (Table 1).

Table 1: Sociodemographic characteristics of AMI patients with and without diabetes mellitus

Characteristic	Diabetic (n = 50)	Non-diabetic (n = 44)	P value
Age (years), mean ± SD	53.2 ± 17.8	54.6 ± 18.6	0.342[a]
Male, n (%)	33 (66.0)	27 (61.4)	0.471[b]
Female, n (%)	17 (34.0)	17 (38.6)	

[a] Independent samples t-test; [b] chi-square test.

Glycemic parameters

RBS and HbA1c were markedly higher in diabetic than in non-diabetic AMI patients (P < 0.001 for both; Table 2). Mean values in non-diabetic patients were within the reference range.

Table 2: Glycemic parameters in AMI patients with and without diabetes mellitus

Parameter	Reference range	Diabetic (n = 50)	Non-diabetic (n = 44)	P value
RBS (mg/dL)	70-140	237.6 ± 65.4	111.2 ± 18.1	< 0.001
HbA1c (%)	< 5.7	9.8 ± 1.9	5.2 ± 0.3	< 0.001

Values are mean ± SD. RBS, random blood sugar; HbA1c, glycated hemoglobin.

Cardiac markers

Mean CK-MB and AST were significantly higher in diabetic patients (211.4 ± 141.8 vs 179.2 ± 108.6 U/L, P = 0.024; and 187.6 ± 109.8 vs 156.8 ± 101.4 U/L, P = 0.034, respectively; Table 3). Mean troponin I was 12.27 ± 16.68 ng/mL

in diabetic and 14.15 ± 17.11 ng/mL in non-diabetic patients (reported P < 0.001). All three markers were well above their reference ranges in both groups.

Table 3: Cardiac markers in AMI patients with and without diabetes mellitus

Parameter	Reference range	Diabetic (n = 50)	Non-diabetic (n = 44)	P value
Troponin I (ng/mL)	< 0.04	12.27 ± 16.68	14.15 ± 17.11	< 0.001
CK-MB (U/L)	0-25	211.4 ± 141.8	179.2 ± 108.6	0.024
AST (U/L)	0-40	187.6 ± 109.8	156.8 ± 101.4	0.034

Values are mean ± SD. CK-MB, creatine kinase-MB; AST, aspartate aminotransferase.

Homocysteine

Serum homocysteine was substantially higher in diabetic than in non-diabetic patients (25.3 ± 7.2 vs 13.8 ± 4.6 µmol/L, P < 0.001; Table 4). Hyperhomocysteinemia was present in 42 (84%) diabetic and 10 (23%) non-diabetic patients, and severe hyperhomocysteinemia in 18 (36%) and 2 (5%), respectively (P < 0.001 for both).

Table 4: Serum homocysteine in AMI patients with and without diabetes mellitus

Parameter	Diabetic (n = 50)	Non-diabetic (n = 44)	P value
Homocysteine (µmol/L), mean ± SD	25.3 ± 7.2	13.8 ± 4.6	< 0.001[a]
Hyperhomocysteinemia (≥ 15 µmol/L), n (%)	42 (84)	10 (23)	< 0.001[b]
Severe hyperhomocysteinemia (≥ 30 µmol/L), n (%)	18 (36)	2 (5)	< 0.001[b]

[a] Independent samples t-test; [b] chi-square / Fisher's exact test.

DISCUSSION

In this comparison of 94 AMI patients, those with diabetes had markedly higher homocysteine than those without, with a mean about 11 µmol/L higher and hyperhomocysteinemia in more than four of five diabetic patients. Diabetic patients also had higher CK-MB and AST, while age and sex were well balanced between the groups, so these differences are unlikely to be explained by demographic imbalance.

The strong association between diabetes and homocysteine agrees with earlier work reporting raised homocysteine in type 2 diabetes, particularly in those with macrovascular disease, nephropathy and insulin resistance [17]. It is also consistent with prospective data showing that hyperhomocysteinemia carries a greater mortality risk in diabetic than non-diabetic individuals [18]. Raised homocysteine predicts adverse outcomes in patients with established coronary disease [19] and may contribute to vascular injury through endothelial dysfunction, oxidative stress and thrombosis [15, 16]. In the present study the high prevalence of severe hyperhomocysteinemia among diabetic patients (36%) deserves attention. Factors that influence homocysteine, such as renal function, vitamin B12 and folate status and metformin use, and could contribute to the difference.

The higher CK-MB and AST in diabetic patients suggest a larger enzymatic footprint of myocardial injury, which fits the observation that diabetes is associated with worse outcomes after acute coronary syndromes [5]. Troponin I did not show the same pattern: its mean was numerically lower in diabetic patients, and the large standard deviations indicate a skewed distribution. The sampling time relative to symptom onset strongly affects troponin and CK-MB values [9, 11], so these results should be interpreted with care.

As expected, RBS and HbA1c were much higher in diabetic patients. The mean HbA1c of 9.8% indicates poor long-term glycemic control in this group, which is relevant because hyperglycemia in the setting of infarction is associated with increased mortality [8].

Whether homocysteine is a cause or only a marker of risk remains uncertain, because large trials of homocysteine lowering with B vitamins did not reduce cardiovascular events [20]. The present data therefore support homocysteine as a marker of risk in diabetic AMI patients rather than as evidence for treatment.

This study has limitations. It was conducted with a modest sample, and the design does not allow conclusions about causality or outcomes. Follow-up data were [not available], so the prognostic value of homocysteine could not be assessed. Confounders such as medication, renal function and nutritional status may not have been fully controlled.

FUTURE PERSPECTIVES

Larger, multicenter prospective cohorts with follow-up are the first priority. Hyperhomocysteinemia and diabetes together carry a high mortality risk in population studies [18], and diabetic patients fare worse after acute coronary

syndromes [1, 3, 5, 12, 15, 21–25]. Whether admission homocysteine predicts in-hospital and long-term mortality, heart failure or recurrent infarction in diabetic AMI patients has not been settled and should be tested with pre-specified outcomes.

Genetic studies would be a natural extension. Genotyping of the MTHFR C677T variant together with measured homocysteine, folate and vitamin B12 could show how much of the raised homocysteine is inherited, nutritional or related to diabetes treatment. Mendelian randomization data suggest that lifelong moderate elevation of homocysteine has little or no effect on coronary heart disease [26], so such work should aim to clarify whether homocysteine is a marker or a mediator of risk in diabetic patients [2, 9, 27–29].

Interventional questions remain open. B-vitamin supplementation lowered homocysteine but did not reduce cardiovascular events in patients with vascular disease [20]. Whether a benefit exists in the subgroup with diabetes and severe hyperhomocysteinemia (≥ 30 μmol/L) is unknown and could be examined in a targeted, adequately powered trial.

Standardized and serial biomarker sampling should be used. Troponin and CK-MB concentrations depend strongly on the time since symptom onset [3, 9], so future studies should record timing, use high-sensitivity troponin assays, and report medians with interquartile ranges, including peak values and infarct size, to allow fair comparison between diabetic and non-diabetic patients.

Finally, multi-marker panels combining homocysteine, glycemic indices, lipids and cardiac enzymes could be analysed with machine-learning methods. Artificial intelligence approaches are already being applied in pharmacokinetic and pharmacodynamic modelling [27] and in microbiome research [30], and similar approaches could be explored for risk stratification in AMI, provided that models are validated in independent cohorts [19, 30–38].

CONCLUSION

AMI patients with diabetes mellitus had significantly higher homocysteine, a much higher frequency of hyperhomocysteinemia, and higher CK-MB and AST than AMI patients without diabetes. Measuring homocysteine together with conventional cardiac markers may help to identify diabetic AMI patients at higher risk. Prospective, larger studies with outcome data are needed to establish its clinical value.

ACKNOWLEDGMENTS

No funding was received.

CONFLICTS OF INTEREST

The authors declare no conflict of interest – confirm.

REFERENCES

1. Yusuf S, Hawken S, Ounpuu S, Dans T, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364:937-952.
2. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. Circulation. 2016;133:1605-1620.
3. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, et al. Fourth universal definition of myocardial infarction (2018). Circulation. 2018;138:e618-e651.
4. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, et al. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med. 1998;339:229-234.
5. Donahoe SM, Stewart GC, McCabe CH, Mohanavelu S, et al. Diabetes and mortality following acute coronary syndromes. JAMA. 2007;298:765-775.
6. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. Diabetes. 2005;54:1615-1625.
7. Beckman JA, Creager MA, Libby P. Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. JAMA. 2002;287:2570-2581.
8. Capes SE, Hunt D, Malmberg K, Gerstein HC. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet. 2000;355:773-778.
9. Reichlin T, Hochholzer W, Bassetti S, Steuer S, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. N Engl J Med. 2009;361:858-867.
10. Ladue JS, Wroblewski F, Karmen A. Serum glutamic oxaloacetic transaminase activity in human acute transmural myocardial infarction. Science. 1954;120:497-499.
11. Jaffe AS, Babuin L, Apple FS. Biomarkers in acute cardiac disease: the present and the future. J Am Coll Cardiol. 2006;48:1-11.
12. Taskinen MR. Diabetic dyslipidaemia: from basic research to clinical practice. Diabetologia. 2003;46:733-749.
13. Boushey CJ, Beresford SA, Omenn GS, Motulsky AG. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. Probable benefits of increasing folic acid intakes. JAMA. 1995;274:1049-1057.
14. Homocysteine Studies Collaboration. Homocysteine and risk of ischemic heart disease and stroke: a meta-analysis. JAMA. 2002;288:2015-2022.

15. Welch GN, Loscalzo J. Homocysteine and atherothrombosis. N Engl J Med. 1998;338:1042-1050.
16. Lentz SR. Mechanisms of homocysteine-induced atherothrombosis. J Thromb Haemost. 2005;3:1646-1654.
17. Buysschaert M, Dramais AS, Wallemacq PE, Hermans MP. Hyperhomocysteinemia in type 2 diabetes: relationship to macroangiopathy, nephropathy, and insulin resistance. Diabetes Care. 2000;23:1816-1822.
18. Hoogeveen EK, Kostense PJ, Jakobs C, Dekker JM, et al. Hyperhomocysteinemia increases risk of death, especially in type 2 diabetes: 5-year follow-up of the Hoorn Study. Circulation. 2000;101:1506-1511.
19. Nygard O, Nordrehaug JE, Refsum H, Ueland PM, et al. Plasma homocysteine levels and mortality in patients with coronary artery disease. N Engl J Med. 1997;337:230-236.
20. Lonn E, Yusuf S, Arnold MJ, Sheridan P, et al. Homocysteine lowering with folic acid and B vitamins in vascular disease. N Engl J Med. 2006;354:1567-1577.
21. Shinde KS, Jangme CM, Patil AR. Nanoemulsion-based delivery system for naftifine hydrochloride: a promising approach for solubility enhancement. Indian J Pharm Educ Res. 2026;60(3):S944-S953.
22. Shingade JA, Padalkar NS, Park JP, Patil AR. Biocompatible nanobiohybrid of maghemite nanoparticle-coated probiotics: a synergistic platform for enhanced antibacterial, antibiofilm, and antioxidant activities. New Biotechnol. 2026.
23. Survase K, Patil AR, Pawar MN, Jangme C. Preparation, optimization, and characterization of eugenol oil-based phytosomes as therapeutic agent. J Mol Sci. 2025;35(3):1182-1188.
24. Walvekar M, Patil A, Pawar SH. Climate change and human health. In: Climate Change and Sustainable Development. 2025:148-167.
25. Wang J, Yuan F, Kendre M, He Z, Dong S, Patil A, Padvi K. Rational design of allosteric inhibitors targeting C797S. In: Novel Molecular Targets in Cancer Therapy, Volume I. 2026.
26. Clarke R, Bennett DA, Parish S, Verhoef P, et al. Homocysteine and coronary heart disease: meta-analysis of MTHFR case-control studies, avoiding publication bias. PLoS Med. 2012;9:e1001177.
27. Pawar V, Patil A, Tamboli F, Gaikwad D, et al. Harnessing the power of AI in pharmacokinetics and pharmacodynamics: a comprehensive review. Int J Pharm Qual Assur. 2023;14:426-439.
28. Phalake SS, Patil AP, Patil AR, Salunkhe AB, Park JP, Thorat ND, et al. Synergistic magnetic nano-chemotherapy overcomes chemoresistance in 3D breast cancer models. Microchim Acta. 2026;193(8):593.
29. Sharma P, Sharma V, Jaswal S, Patil AR, Jha AN, Ramachandiran S. Personalized medicine in the era of predictive and preventive healthcare. In: Modern Biological Applications in the Age of AI. 2027:1-36.
30. Patil A, Singh N, Patwekar M, Patwekar F, et al. AI-driven insights into the microbiota: figuring out the mysterious world of the gut. Intell Pharm. 2025;3:46-52.
31. Memon S, Rukade S, Patil A, Patvegar S. Managing: new molecular markers to differentiate carbon dioxide intoxication from asphyxia due to oxygen deficiency. Forensic Sci Med Pathol. 2026.
32. Patil A. Age prediction of hematoma using hyperspectral imaging. Forensic Sci Med Pathol. 2026.
33. Patil A. The relationship between quality of discharge teaching and oral nutritional supplementation adherence in postoperative patients with gastric cancer: a chain mediated role of ... J Clin Nurs. 2026 Jul;35(7):3258-3260.
34. Patil A, Mali V. Assessment of learning aptitudes: the case for self-regulated and self-directed integration. Med Sci Educ. 2026:1-2.
35. Patil AR. Prognostic factors in neurofibromatosis type 1 (NF1)-associated malignant peripheral nerve sheath tumors: clinical implications beyond NF1 status. Cancer. 2026;132(8):e70399.
36. Patil AR, Ballari S, Bagwan A, Chougule A, Chougale H, Chougale S, et al. Nutraceuticals in colon cancer: emerging evidence, mechanistic insights and clinical applications. J Chem Health Risks. 2025;15(6):3621.
37. Patil AR, Diwate MSK, Memon S, Patil PS. Environmental management for raising resilient kids: a practical plan. Literatureslight Publishing; 2025.
38. Patil S, Powar GS, Harale S, Galatage ST, Liu S, Lopes BS, et al. Formulation and evaluation of a licorice-resveratrol lollipop for targeting Streptococcus mutans biofilm and antimicrobial resistance. Infect Drug Resist. 2025.
39. Cheng Z, Kandekar U, Ma X, Bhabad V, Pandit A, Liu L, Luo J, Munot N, et al. Optimizing fluconazole-embedded transfersomal gel for enhanced antifungal activity and compatibility studies. Front Pharmacol. 2024;15:1353791.
40. Dhane K, Gupta M, Hyam S, Patil A. Systemic review on polyherbal formulation. Int J Res Pharm Chem. 2023;13(1):29-37.
41. Diwate S, Chougale S, Chougule P, Latkar S, Bhandari S, Ballari S, et al. Drug utilization patterns in orthopaedic practice: a systematic literature review and meta-analysis of present-day evidence. J Chem Health Risks. 2025;15(6):3638.
42. Gaikwad A, Harshad R, Patil SB. Formulation, evaluation and antioxidant properties of herbal soap for anti-acne treatment. Int J Novel Res Dev. 2024;9(8). ISSN: 2456-4184.
43. Konduskar RR, Patil AR, Bhinge SD, Chawla GR, Yadav YB. Phytochemical and nanoparticle-based therapeutic potential of Sphaeranthus indicus against hepatocellular carcinoma via cryptomeridiol targeting. Front Oncol. 2025;15:1691905.