(International Print/Online Journal)

SJIF IMPACT FACTOR: 6.829
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 9, Issue 4, Page No: 491-501
July-August 2026

IJMSCR

Correlation Of Serum Prolactin As Prognostic Indicator Of Cardiovascular Mortality In Type-2 Diabetes Mellitus Patients

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Type of Publication: Original Research Paper
Conflicts of Interest: Nil

Abstract

Background: Cardiovascular disease continues to be the leading cause of morbidity and mortality in patients with T2DM. It has been scientifically proved that PRL can serve as a cardiometabolic regulator; however, the importance of PRL in predicting the risk of development of cardiovascular diseases associated with T2DM has not yet been well-defined. The current study is designed to examine the association between PRL levels and cardiac biomarkers (Troponin-I, CK-MB) and HbA1c.

Methods: The study was conducted cross-sectionally during the period November 2024 to October 2025, using 92 participants with acute myocardial injury who were also diagnosed with T2DM in a tertiary care hospital. The parameters measured included demographic information, clinical history, HbA1c, Troponin I, CK-MB, and PRL levels in blood. The correlation analysis used was Spearman's rank correlation coefficient.

Results: However, the group (66.3% males; average age 59.54 ± 9.54 years) revealed poor glycemic control (average HbA1c 8.68 ± 1.96%). Though the cardiac injury biomarkers indicated a strong and significant positive association between each other (Troponin-I and CK-MB: $\rho = 0.692$, $p < 0.001$), PRL values did not reveal any statistically significant association with Troponin-I ($\rho = 0.108$, $p = 0.305$), CK-MB ($\rho = 0.125$, $p = 0.235$), or HbA1c ($\rho = -0.008$, $p = 0.938$).

Conclusion: PRL serum levels are not influenced by myocardial injury or glycemic control during the period of the acute cardiac event. The current observations fail to establish the utility of PRL as a marker of myocardial injury.

Keywords: Type 2 Diabetes Mellitus, Serum Prolactin, Troponin-I, CK-MB, Cardiovascular Disease, Acute Myocardial Injury, Biomarkers

Introduction

T2DM still remains one of the most widespread diseases across the world. Taking into account the current state of affairs regarding this disease as described in the data obtained from IDF Diabetes Atlas 11th Edition for 2025, it should be noted that there are 589 million people suffering from this disease, and 90% (or 853 million people in 2050) have T2DM [1]. It should be also mentioned that cardiovascular diseases remain the leading cause of morbidity and mortality among T2DM patients according to the recent statistics provided by IDF, the chance of developing heart failure in T2DM patients is 84% higher compared to non-diabetic patients [1]. Moreover, disease burden studies demonstrated an increase in the incidence of the complications associated with T2DM that are connected with the functioning of cardiovascular, renal and nervous systems [2].

The relationship between T2DM and CVD from a pathophysiological perspective is mainly through the chronic state of hyperinsulinemia, insulin resistance, endothelial dysfunction, and low-level inflammatory status which promote atherosclerosis and increase the risk for acute coronary complications [3]. With this understanding, the need for biomarkers that can reflect both metabolic dysfunction and myocardial damage comes into play.

Traditionally considered a regulator of lactation, prolactin (PRL), a hormone which mediates through extra-pituitary receptors, is now considered as a pleiotropic mediator of glucose homeostasis and vasculature biology by virtue of its wide distribution in tissues like pancreatic β-cells, adipose tissue, and vascular endothelial cells [4]. In a cross-sectional study conducted in South India, the physiological level of prolactin was found to be inversely proportional to the prevalence of T2DM and was also inversely correlated with glycemic and lipids levels [5].

Nonetheless, the present state of evidence on the association between PRL and cardiovascular events in T2DM is inconclusive. A population-based study among individuals with T2DM demonstrated an association between high levels of serum PRL and both all-cause and cardiovascular mortality in a dose-response manner [6]. However, a more recent study showed that mildly increased levels of PRL were associated with decreased carotid intima-media thickness, thus suggesting that PRL had a possible vasculoprotective role [7]. The editorial accompanying this study explained the discrepancies due to design and population differences and also in the technique used for PRL measurement [4].

Considering the discrepancy, and in light of the proven role of Troponin-I and CK-MB in diagnosing myocardial damage, the current study was conducted to look into the relation between prolactin levels in the blood and the mentioned cardiac markers to be used as a predictor of cardiovascular mortality among T2DM patients.

Materials And Methods

Study Design and Setting

The study was conducted during an 11 month time period beginning in November 2024 and ending in October 2025. The study was conducted in the OPD and IPD of the Department of General Medicine and Cardiology of Sri Manakula Vinayagar Medical College & Hospital (SMVMCH), a tertiary care hospital situated in the rural area of Pondicherry, India. Prior approval for the study was taken from the Institutional Research Committee and the Institutional Ethics Committee.

Study Population and Eligibility Criteria

The study comprised adults diagnosed with T2DM and who had developed acute cardiovascular problems. The use of convenience sampling was done in selecting participants. Convenience sampling is a type of non-probability sampling method. Consent was obtained from all participants.

Inclusion Criteria:

1. Age > 20 years.
2. Confirmed diagnosis of Type 2 Diabetes Mellitus for a duration of ≥ 2 years.
3. Diagnosed with an acute cardiac event: Acute Coronary Syndrome (ACS), myocarditis, or cardiomyopathy.
4. Evidence of myocardial injury: 2D Echocardiogram showing regional wall motion abnormality (RWMA) and elevated cardiac biomarkers (CK-MB, Troponin-I).

Exclusion Criteria:

1. Age < 20 years.
2. T2DM diagnosis for < 2 years.
3. Current treatment with corticosteroids, Antipsychotics, Antiemetics and Antidepressants.
4. Pregnancy or gestational diabetes mellitus (GDM).
5. Known cases of valvular heart disease or a history of previous myocardial infarction (MI).
6. Other conditions known to cause isolated Troponin-I elevation (e.g., severe sepsis, chronic kidney disease, malignancy).
7. Patients who refused consent.

Sample Size Calculation

The sample size was calculated using Cochran's formula. Based on an expected cardiovascular disease prevalence in T2DM patients of 32.2% (Einarson et al. [8]), a 95% confidence level, and an 8% margin of error, the initial target was 132 subjects. Ultimately, 92 subjects met all the strictly defined eligibility criteria.

Clinical and Demographic Assessment

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Having obtained consent from the patients, clinical evaluations were carried out in order to establish any diseases that would influence the results of the experiment. The demographics involved age, gender, height measured using the measuring tape, and body weight established using the weighing scale. To establish the hemodynamic values (mean arterial pressure, heart rate, and oxygen saturation), electronic instruments were used, while to measure the systolic and diastolic blood pressures, sphygmomanometer was used. Patients were also evaluated in terms of having other disease conditions such as systemic hypertension and thyroid disease.

Data Collection and Biochemical Analysis

The clinical and laboratory parameters were gathered from the patients' hospital files, while the endocrine profile was collected from the Department of Biochemistry. The proforma was used to ensure uniformity in the data collected. FBS and PPBS were done through the use of the GOD-POD method together with HbA1c test which was quantified through Nephelometry. Cardiac function assessment was performed using 12-lead ECG machine to detect any electrical irregularities in the heart together with 2D ECHO (Philips). Besides that, there was the cardiac marker analysis in which CK-MB was determined using the IFCC method and Troponin-I using the rapid card tests. There was also determination of the serum prolactin (PRL) using the Uricase method with the help of an automated clinical chemistry analyzer (Cobas Integra/Cobas C System). Additionally, there were Renal Function Tests (RFT) which were conducted using BOILIS 50i Autoanalyzer.

Bias Mitigation and Quality Control

Data consistency and avoidance of research bias were ensured through consistent training of all medical personnel who conducted assessments and data entry. In addition, measurement bias was avoided by consistently using calibrated and standardized clinical instruments and automated biochemical analyzers in all laboratory tests.

Statistical Analysis

Raw data were first entered and arranged in Epi Info (Version 7.2.2.6). Complete statistical analysis was done using SPSS Version 24.0. Descriptive statistics were used to present categorical data, such as gender and hypertensive, in frequencies and percentages. On the other hand, continuous data, such as age, HbA1c, TROP-I, CK-MB, and PRL, were presented using mean and standard deviation (SD). For inferential statistics, Spearman's rank correlation coefficient (ρ) was used for analyzing the relationship between continuous non-normally distributed data. In particular, the analysis was used to check the concurrent validity of the cardiac enzymes and also to determine the relationship between serum prolactin and glycemic control (HbA1c) and myocardial injury. The level of significance was taken at <0.05. Figure 1 shown the Flowchart of the Study Design and Data Analysis.

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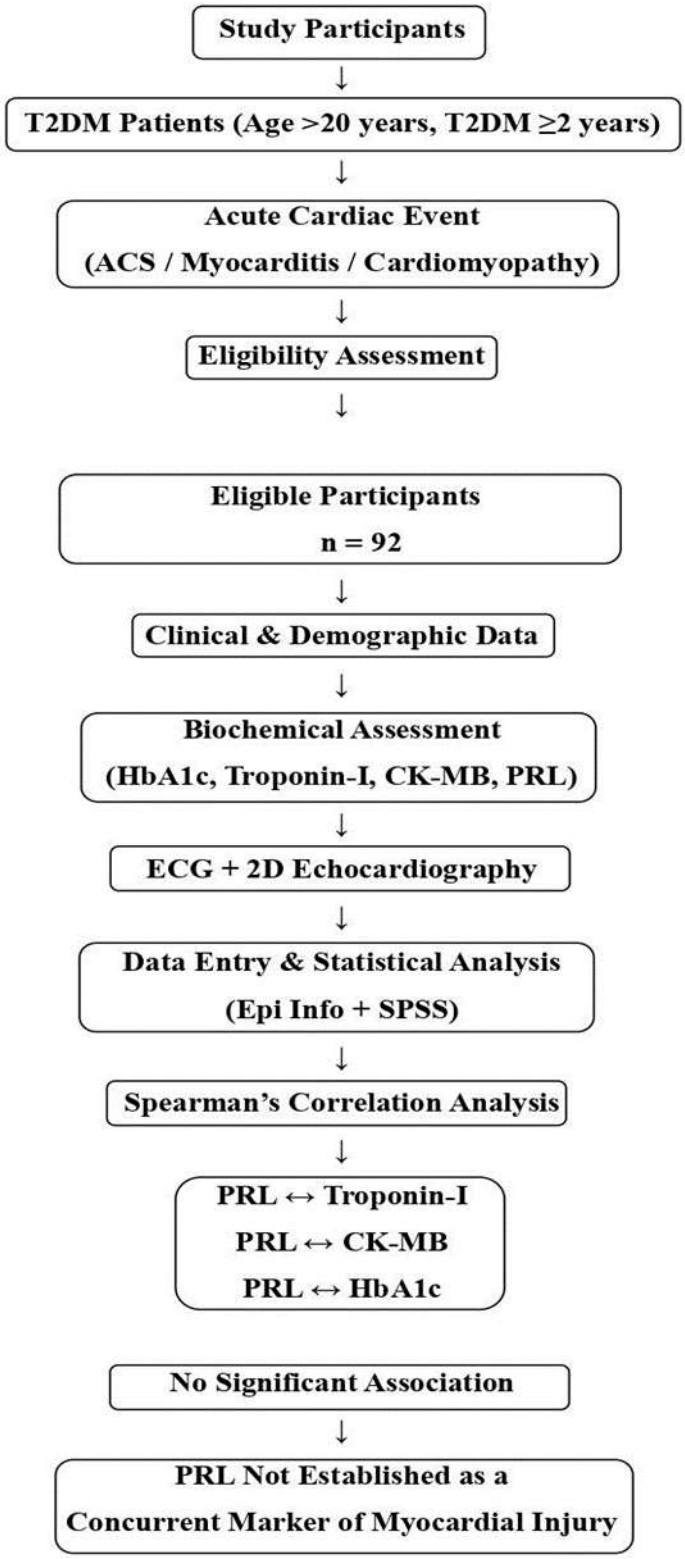
Figure 1: Flowchart of the Study Design and Data Analysis



Results

Among the 132 patients estimated to have enough power, 92 diabetic patients meeting all eligibility criteria were recruited from the outpatient and inpatient clinics of General Medicine and Cardiology during the period November 2024 through October 2025; this became the final sample used in this study.

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All 92 patients had complete data on all four primary biochemical parameters considered in this study and were thus retained for the analysis below.

Baseline Demographic And Clinical Characteristics

Males formed the majority of this sample group, with two males to each female; 61 (66.3%) were males while 31 (33.7%) were females (Figure 2). The mean age of onset was 59.54 years (standard deviation of 9.54); hence, the participants of the study were within their fifties and sixties, age group where chronic T2DM and cardiovascular risks are often associated. The above age group is also consistent with the usual demographic makeup of T2DM patients who have acute cardiovascular problems.

Distribution Of Comorbid Conditions

The prevalence of systemic hypertension was seen in more than half of the patients, that is 48 patients (52.2%) whereas the other 44 patients (47.8%) did not have any such history (Figure 3). The rest of the comorbid conditions were very few. Only nine patients were found with chronic kidney disease (9.8%), whereas thyroid dysfunction occurred rarely with only one case of hypothyroidism and hyperthyroidism each (1.1%). There were no other comorbid conditions present in the other 81 patients (88.0%) (Figure 4). Thus, the presence of hypertension was the primary comorbid condition in this population while the rest of the comorbid conditions like chronic kidney disease and thyroid disease were minimal.

Figure 2: Gender distribution of study participants (N = 92)

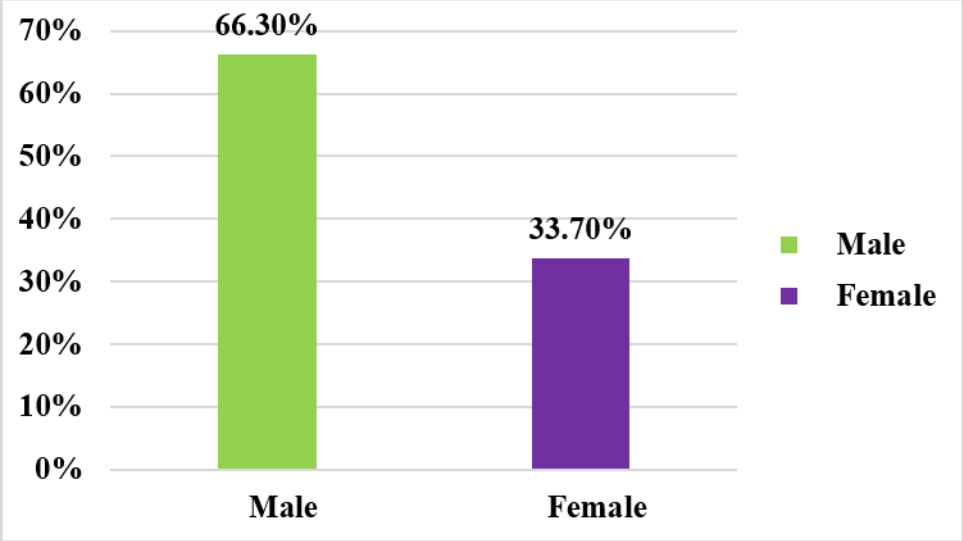

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Figure 3: Distribution of systemic hypertension among study participants

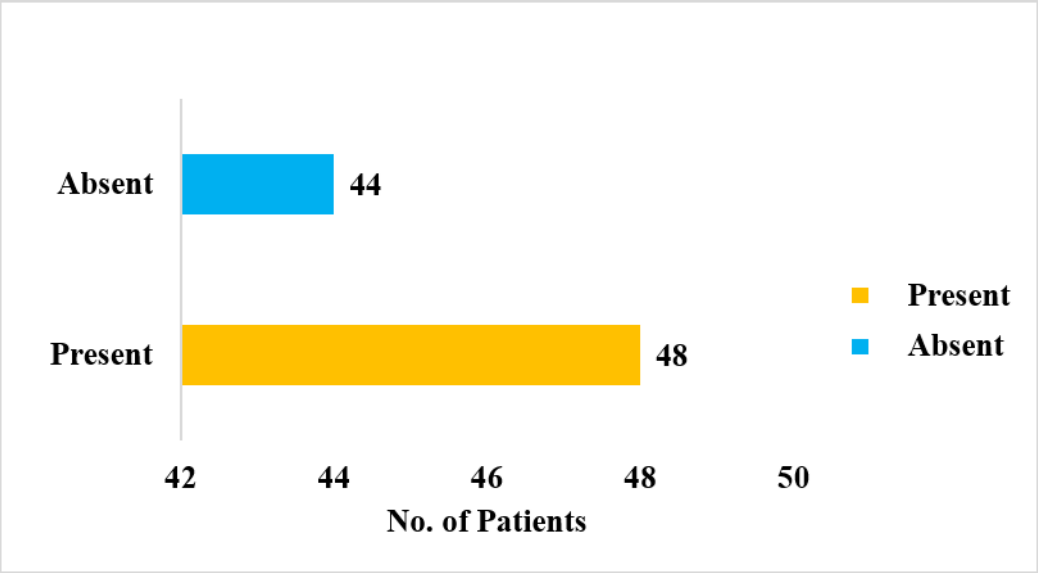
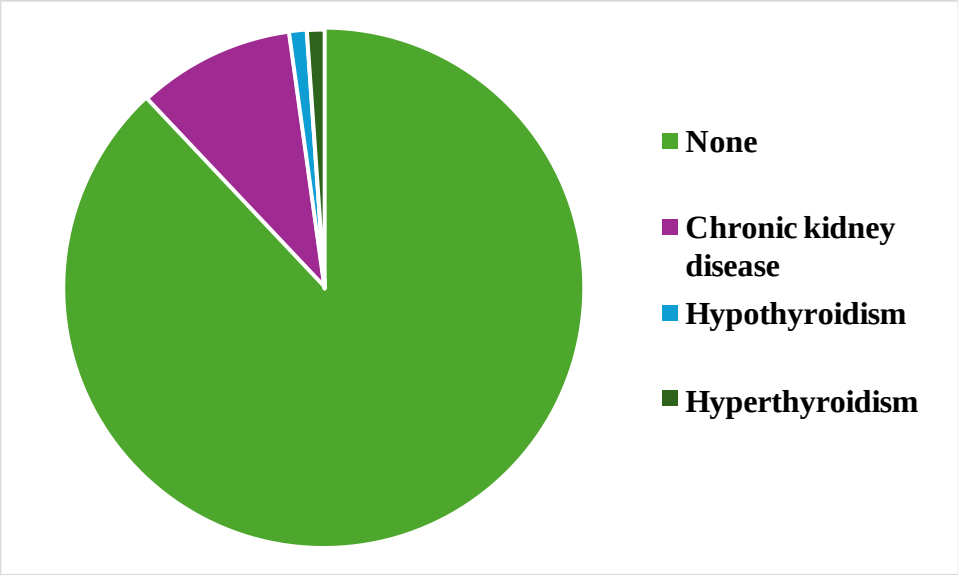


Figure 4: Distribution of other comorbid conditions among study participants



Glycemic Control and Cardiac Biomarker Profile

Table 1 provides a summary of the biochemical markers that were measured in the sample, apart from age and demographic factors that were described earlier in order not to repeat the same data in different tables. Overall, glycemic control in this sample was suboptimal, as indicated by the mean HbA1c concentration of 8.68% (SD 1.96%), which is much higher than what would be expected in patients with T2DM.

The two parameters used for assessment of myocardial injury were distributed widely about relatively small means. The mean value of troponin-I was 4.37 ng/mL, whereas its standard deviation of 10.58 was more than twice as large as the mean itself, suggesting a highly skewed distribution where the majority of participants had low or almost normal values while the minority had high ones. This variance in the level of troponin is understandable as a specific selection of the participants who suffer from acute coronary syndrome, myocarditis, and cardiomyopathy, different presentations with respect to the degree of acute myocardium involvement. The

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same could be said about CK-MB that was distributed similarly, having a mean value of 26.56 U/L with a standard deviation of 21.66. Serum prolactin demonstrated the same pattern of distribution, having a mean of 21.75 ng/mL and a standard deviation of 24.33, and this suggests that the secretion of prolactin among the selected group could be affected by some individual-specific factors like stress or acute illness.

Table 1: Descriptive statistics of biochemical parameters (N = 92)

Parameter	Mean	SD
HbA1c (%)	8.68	1.96
Troponin-I (ng/mL)	4.37	10.58
CK-MB (U/L)	26.56	21.66
Prolactin (ng/mL)	21.75	24.33

Correlation Between Serum Prolactin and Cardiac and Glycemic Biomarkers

Pairwise association among the variables like HbA1c, troponin I, CK-MB, and prolactin in 92 participants was analyzed using Spearman's rank correlation coefficient (ρ).

HbA1c and the other biomarkers: The level of HbA1c was unrelated to any of the other variables considered. It had only a small correlation to troponin-I, which was negative ($\rho = -0.113$, $p = 0.284$), small to CK-MB, which was positive ($\rho = 0.068$, $p = 0.521$), and essentially zero to prolactin ($\rho = -0.008$, $p = 0.938$). None of these correlations were close to being statistically significant, suggesting that blood glucose regulation, as measured by HbA1c, was unrelated to cardiac enzyme or prolactin levels in this particular sample population.

Troponin-I and CK-MB: However, the single correlation that was significant for all the data set was that existing between the two cardiac injury markers ($\rho = 0.692$, $p < 0.001$). Patients with high levels of troponin I were found to have high levels of CK-MB, which is not surprising because both enzymes enter the blood stream following myocardial damage and hence they should be increasing simultaneously.

Prolactin and the cardiac markers: There was no statistically significant relationship between prolactin and either troponin-I ($\rho = 0.108$, $p = 0.305$) or CK-MB ($\rho = 0.125$, $p = 0.235$).

Table 2: Spearman's correlation matrix between biochemical parameters (N = 92)

Variable pair	P	p value
HbA1c vs Troponin-I	-0.113	0.284
HbA1c vs CK-MB	0.068	0.521
HbA1c vs Prolactin	-0.008	0.938
Troponin-I vs CK-MB	0.692	<0.001*
Troponin-I vs Prolactin	0.108	0.305
CK-MB vs Prolactin	0.125	0.235

*Statistically significant, $p < 0.05$.

In all the six possible pairwise combinations, only the one between troponin-I and CK-MB exceeded the level of statistical significance, while the other five, in which one or the other of the two factors - HbA1c or prolactin - took part, had weak correlations that might have happened by chance. This observation remains true irrespective of whether prolactin is paired with either troponin-I or CK-MB.

Taken together, these observations confirm once again troponin I and CK-MB as contemporaneous

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biomarkers of mycardial damage in this population, in keeping with their previously recognized medical importance. On the contrary, prolactin in the blood did not vary depending on the degree of glycaemia or either of the two cardiac biomarkers, indicating thus that, at least in the acute presentation of cardiovascular disease, the prolactin levels in T2DM

subjects are independent of myocardial damage or glycaemic control. Survival and mortality analyses cannot be conducted on the basis of the current cross-sectional information, and the correlation results described above should be considered therefore as an observation regarding contemporaneous biomarkers' association and not as proof of any prognosis or causal relationship between prolactin and cardiovascular outcome.

DISCUSSION

The primary result of the current research can be defined as a negative one: the levels of prolactin in the blood serum neither correlated with the patients glycemic status nor with any of the markers of myocardial injury in 92 patients with chronic type 2 diabetes with documented myocardial infarction. On the other hand, troponin-I and CK-MB correlated perfectly as one would expect from a course on physiological ($\rho = 0.692$, $p < 0.001$). This gives us confidence in our measurements of myocardial injury as the two factors we set out to study, prolactin and glycaemia, did not cooperate at all.

This negative finding is somewhat uncomfortable but not surprising considering the existing literature which does not provide a unified view of the physiological role of prolactin in cardiovascular disease development. Shen et al. conducted a study of general T2DM population and discovered that there was a dose-response association between high prolactin levels and increased risk of both all-cause and cardiovascular mortality [6]; conversely, Zhang et al. who used carotid intima-media thickness as an alternative endpoint observed an opposite trend where mildly elevated prolactin had a beneficial effect [7]. In between these two studies, several large population-based cohorts, the Framingham hormones sub-study, EPIC-Norfolk case-control analysis among others, demonstrated lack of association between prolactin and cardiovascular disease [9]. According to Glezer et al., analyzing the very same literature, the association between prolactin and cardiometabolic status may have more of a U-shape to it, with a certain physiological optimal window lying somewhere around 25-100 ng/mL, while the abnormal metabolic profile emerges only when prolactin levels become significantly higher or lower than this window [9]. If such a thing were true, the use of just a single snapshot prolactin level measurement from a mixed group of patients presenting with their first-time cardiac disease and showing an enormous variability in this biomarker (SD 24.33 versus mean 21.75 ng/mL) does not seem to be the approach that should have revealed the effect. Even the presence of several patients with very high prolactin levels (possibly due to stress) among many other individuals with normal prolactin might mask any existing effect.

This underscores an inherent limitation of the use of prolactin as an acute-phase marker in such cases. The response of prolactin to physical stress, venipuncture, food intake, and sympathetic reaction following ischemia is very sensitive, which increases prolactin levels regardless of cardiometabolic abnormalities [9,10]. This concept is supported by another recent review by Auriemma et al., who highlight the fact that chronic hypoprolactinemia may be associated with adverse cardiometabolic outcomes, including insulin resistance and dyslipidemia [10]. However, this type of cross-sectional design study would not be able to differentiate between a patient with a temporarily raised level of prolactin because he/she was undergoing a cardiac event and another with a permanently elevated prolactin due to endocrinological reasons. In both cases, this would result in a tendency towards blurring the relationship of such patients with either troponin-I or CK-MB measured simultaneously.

The demographics and co-morbidities of the cohort are also interesting to note because they may themselves provide a partial explanation for the unusual prolactin profile. There were twice as many men as women in the cohort, which is a fairly common demographic distribution in acute cardiac cases but is also important since the literature regarding prolactin and cardiovascular risk is predominantly separated by sex — some studies, like the Danish registry study and the UK prolactinoma cohort study, that found a positive association did so in males only and not in the general population [9]. An analysis of a cohort that contains predominantly males and without any form of stratification of results could very well be diluting a

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potential sex-dependent effect rather than finding no effect at all. At the same time, there is also evidence that T2DM females have a greater relative risk of developing cardiovascular disease than men [11], bringing into question whether the presence of about one-third females in the cohort is sufficient to reveal a sex-dependent prolactin effect.

In fact, the lack of association between HbA1c levels and the cardiac enzymes is not so unexpected, at least in the context of the very difference in timescales in which chronic glycemic burden and acute myocardial damage operate. Glycosylated hemoglobin measures average blood sugar levels in the past three months, whereas troponin-I and CK-MB peak and trough over a period of hours to days around an isolated ischemic event [12,13]. It has been shown by the UKPDS legacy studies that the effect of glycemic control on cardiovascular outcomes manifests itself over years, sometimes even becoming evident after ten years from the end of the exposure period [14], and it has been documented in the pathophysiological literature on diabetic cardiomyopathy and endothelial dysfunction that hyperglycemia causes vascular damage in a progressive manner [15-19].

An average HbA1c level of 8.68% in this population indicates that the glycemic control was, on the whole, suboptimal, but the suboptimal glycemic control only signifies accumulated risk and not the myocardial damage sustained in any single acute attack. The comparatively lower prevalence of chronic kidney disease (9.8%) and thyroid disorders (2.2%) is even lower than the common prevalence rate of 20-30% in T2DM cases seen in other populations [20]. This could be due to their healthier renal status or even under-ascertainment during a cross-sectional record review.

There are several limitations to this conclusion. The recruitment process did not meet the required number of 132 participants; the number was only 92, rendering the study poorly powered for a moderate-to-strong effect size since a real yet weak association between prolactin and T2DM may not be picked up, and instead, the absence of such an association may be falsely concluded. The study design involves a cross-sectional, single-center and convenience sampling approach such that the sample consists of cases admitted to one tertiary care hospital in Pondicherry, and not the entire population of T2DM patients with cardiac disease. In addition, the study title suggests a prognostic analysis of the mortality rate of T2DM patients with cardiac disease, while, in reality, no follow-up period was included in the study and the death event was not identified. Similarly, the prolactin testing procedure was not well-defined in the methods section of the paper, and the troponin-I test was done using a qualitative rapid card test as opposed to a quantitative, high sensitivity assay, reducing accuracy at the low range, where most of the values in this study were. However, this does not cast any doubt on the troponin and CK-MB relationship, which is very reliable and expected. Nevertheless, the results obtained from this study concerning prolactin should be considered as hypothesis-generating and not conclusive.

CONCLUSION

In this sample of 92 patients with established T2DM and acute myocardial injury, serum prolactin levels showed no statistically significant correlations with troponin-I, CK-MB, or HbA1c. As would be expected, troponin-I and CK-MB showed highly statistically significant correlations with each other, indicating that the biological markers measured in this study truly reflected myocardial damage. In contrast to these results, serum prolactin seemed independent

of both the glycemic state and the degree of myocardial damage at the time of sample collection, which is similar to other population studies while contradicting other studies that have found either negative or positive associations. Thus, these results do not confirm the role of serum prolactin as a concurrent marker of myocardial damage in T2DM patients. It should be noted that since this study design is cross-sectional and does not contain any mortality follow-up, any conclusions about the prognostic or predictive power of serum prolactin in terms of mortality cannot be made, although it is clear from the framing of the title of this study. What has been shown here are just concurrent biochemical relations and not predictive relations. An adequately powered prospective study with sex-stratification and survival outcomes needs to be conducted in order to make such conclusions.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas. 11th ed. Brussels: International Diabetes Federation; 2025.

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2. He KJ, Wang H, Xu J, Gong G, Liu X, Guan H. Global burden of type 2 diabetes mellitus from 1990 to 2021, with projections of prevalence to 2044: a systematic analysis across SDI levels for the global burden of disease study 2021. Front Endocrinol (Lausanne). 2024;15:1501690.
3. Ikmal SISS, Huri HZ, Vethakkan SR, Wan Ahmad WA. Potential biomarkers of insulin resistance and atherosclerosis in type 2 diabetes mellitus patients with coronary artery disease. J Diabetes Res. 2013;2013:698567.
4. Papazoglou AS, Leite AR. Prolactin levels and cardiovascular disease: a complicate relationship or a confounding association? Eur J Prev Cardiol. 2023;32(7):612-615.
5. Jayashankar CA, Manohar A, Joshi A, Dwarakanathan V, Pinnelli VBK, Sarathi V, Gada LM. Association of serum prolactin with type 2 diabetes mellitus: a comparative cross-sectional study from South India. Cureus. 2022;14(4):e23721.
6. Shen Y, Yang Q, Hu T, Wang Y, Chen L, Gao F, et al. Association of prolactin with all-cause and cardiovascular mortality among patients with type 2 diabetes: a real-world study. Eur J Prev Cardiol. 2023;30(14):1439-1447.
7. Zhang B, Ke J, Zhang N, Zhao W, Zhong L. Mildly elevated serum prolactin level may be a protective factor for preventing thickening of the carotid intima-media in patients with type 2 diabetes mellitus. Endocr Connect. 2025;14(1):e240285.
8. Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017. Cardiovasc Diabetol. 2018;17(1):83.
9. Glezer A, Santana MR, Bronstein MD, Donato J Jr, Jallad RS. The interplay between prolactin and cardiovascular disease. Front Endocrinol (Lausanne). 2023;13:1018090.
10. Auriemma RS, Scairati R, Pirchio R, Del Vecchio G, Di Meglio S, Menafra D, Pivonello R, Colao A. Cardiometabolic effects of hypoprolactinemia. Rev Endocr Metab Disord. 2024;25(6). doi:10.1007/s11154-024-09891-z.
11. Peters SA, Huxley RR, Sattar N, Woodward M. Sex differences in the excess risk of cardiovascular diseases associated with type 2 diabetes: potential explanations and clinical implications. Current cardiovascular risk reports. 2015 Jul;9(7):36.
12. Yiu KH, Lau KK, Zhao CT, Chan YH, Chen Y, Zhen Z, Wong A, Lau CP, Tse HF. Predictive value of high-sensitivity troponin-I for future adverse cardiovascular outcome in stable patients with type 2 diabetes mellitus. Cardiovasc Diabetol. 2014;13:63.
13. Kurapati R, Zubair M. Creatine Kinase MB: Diagnostic Utility and Limitations. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan.
14. Stratton IM, Adler AI, Neil HAW, Matthews DR, Manley SE, Cull CA, Hadden D, Turner RC, Holman RR. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. BMJ. 2000;321(7258):405-412.
15. Salvatore T, Pafundi PC, Galiero R, Albanese G, Di Martino A, Caturano A, Vetrano E, Rinaldi L, Sasso FC. The diabetic cardiomyopathy: the contributing pathophysiological mechanisms. Front Med (Lausanne). 2021;8:695792.
16. Kaur R, Kaur M, Singh J. Endothelial dysfunction and platelet hyperactivity in type 2 diabetes mellitus: molecular insights and therapeutic strategies. Cardiovasc Diabetol. 2018;17:121.
17. Iglesias P, Moure Rodríguez MD, Guerrero-Pérez F, Simó Servat A, González Fernández L, Fernández-Rodríguez E, et al. Sex differences in cardiovascular risk factors and cardiovascular disease in patients with hyperprolactinemia: a multicenter cross-sectional study. Endocrine. 2026;91(1):65.
18. Dhondge RH, Agrawal S, Patil R, Kadu A, Kothari M. A comprehensive review of metabolic syndrome and its role in cardiovascular disease and type 2 diabetes mellitus: mechanisms, risk factors, and management. Cureus. 2024;16(8):e67428.
19. Raveendran A, Ragunathan S, Neto JF, Mohamed MM, Chen YMK, Baral S, et al. Thyroid disorders and their impact on metabolic syndrome and cardiovascular risk: a narrative review. Cureus. 2025;17(10):e93669.

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20. Fenta ET, Eshetu HB, Kebede N, Bogale EK, Zewdie A, Kassie TD, Anagaw TF, Mazengia EM, Gelaw SS. Prevalence and predictors of chronic kidney disease among type 2 diabetic patients worldwide, systematic review and meta-analysis. Diabetology & Metabolic Syndrome. 2023 Nov 28;15(1):245.

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