Academic Journal of International University of Erbil

Journal Homepage: https://ojs.cihanrtv.com/index.php/public

PRINT-ISSN: 2519-6928

Online-ISSN: 3080-7174

Elevated Blood Cardio-Markers and its Relation to Diabetes Duration in T2DM Patients

Dilaram Bakir Faraj[1]*, Ismail Salih Ibrahim Kakey[2], Rezhna Adil Rashee[3]

[1,2]Department of Biology, Faculty of Science & Health, Koya University, Koya 44023, Kurdistan Region- F.R. IRAQ

[3]Department of medical laboratory technology, Koya Technical Institute, Erbil Polytechnic University, Kurdistan Region F.R. IRAQ

DOI: https://doi.org/10.63841/iue24515

Received 17 May 2025; Accepted 28 Jun 2025; Available online 25 Oct 2025

ABSTRACT

The relationship between diabetes mellitus type 2 and cardiovascular complications stands as a primary cause of patient morbidity and mortality. The myocardial remodeling process caused by hyperglycemia leads to chronic changes which can be identified through specific cardiac biomarkers. The research investigated changes in type 2 diabetic patients regarding cardiac function biomarkers together with their relationship to disease duration and patient age and sex.

The research employed a cross-sectional design at Shahid Dr. Khalid Teaching Hospital located in Koya, Erbil. The research included 90 participants where 60 subjects had diabetes while 30 participants served as controls. The research team measured serum random blood sugar together with lipid profiles and cardiac markers Troponin I and CK-MB and Myoglobin. The analysis between groups and diabetes duration was performed through SPSS version 27.

The cardiac biomarker and lipid abnormality levels in diabetic patients exceeded those of controls at a statistical significance of $p < 0.05$. Patients with diabetes duration exceeding five years demonstrated worse blood sugar management and higher cholesterol levels. Diabetic patients showed elevated levels of Troponin I and CK-MB and myoglobin which indicated subclinical myocardial injury. The triglyceride and LDL and VLDL levels were elevated in diabetic patients, but HDL levels showed no significant differences.

The second form of diabetes mellitus produces significant changes in cardiac biomarkers which indicate early myocardial stress and elevated cardiovascular risk. The combination of inadequate blood sugar management with extended disease duration intensifies these effects which demonstrates the necessity for ongoing cardiac evaluations and strict metabolic management in diabetic patients.

Keywords: Type 2 Diabetes, Cardiac Biomarkers, Hyperglycemia, Cardiovascular Risk, Troponin I, CK-MB, Myoglobin

1 INTRODUCTION

Diabetes mellitus (DM) and especially type 2 diabetes is one of the world's diseases characterized by hyperglycemia and metabolic disturbance [1]. Diabetes has been well established to significantly increase the likelihood of developing cardiovascular disease (CVD) that is responsible for more than 32% of diabetic deaths [2] as well as the diabetic patients have much more risk to have complications of cardiovascular diseases [3]. Perhaps the most important way through which diabetes results in cardiac disease is by prolonging the cardiac exposure to hyperglycemia that results in what is called diabetic cardiomyopathy, structural and functional myocardial remodeling. These changes can be detected at an early stage through cardiac biomarkers, which may be a window for therapeutic intervention and the prevention of harmful cardiovascular events [4]. Growing evidence suggests that higher fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) are associated with subclinical myocardial damage, usually indicated by elevated

*Corresponding author: dlaram.faraj@epu.edu.iq

circulating cardiac troponins [5], [6]. These biomarkers have been of interest not only for their use in the diagnosis of acute coronary syndromes but also as markers of mild cardiac injury in asymptomatic diabetic patients [7].

Notably, diabetic ketoacidosis (DKA) hyperglycemic complications, hyperosmolar hyperglycemic state (HHS) and chronic microvascular complications of nephropathy and neuropathy can also lead to the release of cardiac markers through multi-organ stress reactions [8], [9]. These conditions can lead to transient myocardial ischemia or fluid and electrolyte imbalance which increases the workload of the heart. In such cases, elevated BNP and troponin may indicate both metabolic stress and underlying cardiac disease, which makes clinical decision making challenging unless made in relation with the context [10], [11].

Therefore, it is important to investigate dynamic changes in markers of cardiac function with respect to glycemic control and hyperglycemic crises in the rising global burden of diabetes and its complications. Such info decisions and potential to improve risk stratification, inform treatment decisions, and allow for early intervention in diabetic patients before the onset of irreversible cardiac damage.

This study aims to evaluate and detect biomarkers and cardio marker changes in diabetic patients with cardiovascular disorders. As well as want to find the Prevalence of cardiac disorders in relation to age and gender among type 2 diabetic patients. Then finally, clarify Diabetes onset and disease duration effects on cardiac marker changes.

2 METHODOLOGY

STUDY LOCATION AND POPULATION

This cross-sectional study was conducted in Shahid Dr Khalid teaching hospital, located in Koya, Erbil, Kurdistan region- Iraq. The research involved type 2 diabetic patients with cardiovascular disorders who were being treated at Koya's Shahid Dr. Khalid Teaching Hospital. A total of 60 patients were enrolled for biochemical and cardiac biomarker testing. As well as another 30 controls were used to have comparison between groups.

SAMPLE COLLECTION AND PROCEDURES

A 5 mL venous blood sample was obtained from each patient through a 5cc syringe. The collected blood samples were placed in two separate types of tubes. Gel Tube (Clot Activator), For serum separation also, EDTA K3 Tube, For plasma and whole blood-based tests. After blood collection the gel tubes underwent centrifugation to separate the serum from the other components for biochemical analysis. The following biochemical tests were done for the participants.

1-Random Glucose Testing

Serum glucose was estimated by the enzymatic colorimetric method for the quantitative determination using automatic analyzer (ACCENT 200, CORMAY) diagnostic kit (Chania). which use reddish-violet quinonimine as an indication when glucose is enzymatically converted by glucose oxidase.

2-Lipid Profile Testing

Lipid profile was determined by enzymatic colorimetric method using automatic analyzer (cobas c111, Roche) kits (Germany) Based on the enzymatic conversion of cholesterol to quinonimine pigment and the hydrolysis of triglycerides with lipases, the enzymatic total serum concentration of both cholesterol and triglycerides was calculated. A homogenous enzymatic colorimetric assay was used to measure the amounts of LDL-HDL cholesterol. Triglyceride / 5 = VLDL-c (mg/dl). where VLDL is represented by (TG/5). Every value is expressed in mg/dL.

3-Cardiac Biomarkers (Troponin I, CK-MB, Myoglobin)

The cTnl/CK-MB/Myo Rapid quantitative test was estimated by using Wondfo finecare plus diagnostic kit (Netherlands), with immunofluorescence quantitative analyzer. The finecare TM cTnl/CK-MB/Myo rapid quantitative test is based on florescence immunoassay technology, specifically the sandwich immunodetection method, add the specimen to detection buffer and mix well

signal intensity of detector cTnl/CK-MB/Myo antibodies reflect the amount of antigens and finecare TM FIA meter show cTnl/CK-MB/Myo-concentration in blood specimen.

CHARACTERISTICS OF THE PARTICIPANTS

The research was conducted in tertiary care centers (Shahid Dr Khalid teaching hospital) and targeted adult patients aged 18 years and older who had a confirmed diagnosis of Type 2 Diabetes Mellitus (T2DM) by specialist physicians.

Convenience sampling technique was used to select participants. Participants were recruited consecutively during routine follow-up visits or upon hospitalization due to diabetic-related complications.

SAMPLE SIZE AND SAMPLING TECHNIQUE

A total of 90 patients were selected using purposive sampling and categorized into two subgroups:

Group A: Patients diagnosed with T2DM without any evident cardiovascular or hyperglycemic complications.

Group B: Patients diagnosed with T2DM who presented with hyperglycemic complications such as diabetic ketoacidosis or hyperosmolar hyperglycemic state, and/or cardiovascular conditions such as arrhythmias or ischemic heart disease.

Inclusion Criteria

Participants were eligible for inclusion if they met the following criteria:

- Aged 18 years or older.
- Diagnosed with T2DM for a duration of at least one year.
- Presence or absence of hyperglycemic complications.
- Provided informed consent to participate in the study.

Exclusion Criteria

Participants were excluded from the study if they:

- Had cardiovascular diseases not related to diabetes, regardless of the presence of hyperglycemia.
- Were pregnant or lactating at the time of recruitment.
- Were diagnosed with end-stage chronic kidney disease classified as stage 4 or above.
- Were undergoing chemotherapy or receiving immunosuppressive therapy.

DATA COLLECTION TOOLS AND PROCEDURES

Eligible patients were identified from hospital records and outpatient diabetes clinics. During their scheduled appointments or hospital stays, they were approached by the research team and provided with detailed information about the study. After obtaining informed consent, participants were interviewed face-to-face using a structured questionnaire. Clinical information was collected through direct patient interviews and review of their medical records. Patients were then allocated to either Group A or Group B based on their medical history and current clinical presentation. All data were anonymized to ensure confidentiality and stored securely for analysis.

ETHICAL CONSIDERATIONS

Ethical clearance was acquired from the scientific board in the faculty of health and science, Biology department. The ethical approval issued by Ethical committee in faculty of science, biology department. Informed consent was provided to all participants orally. All the data were anonymized and treated confidentially in line with research ethics and data protection protocols.

STATISTICAL ANALYSIS

The following analyses performed, descriptive statistics to summarize patient characteristics and biomarker levels. Since the data were different for the cases independent simple t test was used, for the controls non-parametric tests were used. While for the comparing both groups due to non-distribution normally, Mann–Whitney U test was used. A p-value ≤ 0.05 was used to determine statistical significance. SPSS version 27 was used to analyze the data.

3 RESULTS

Table 1 summarizes the demographic data of the participants in the case and control groups. As for gender distribution, most participants in both groups were female, with 60% being cases and 66.7% being controls, while males were 40% and 33.3% respectively. In terms of the duration of type 2 diabetes mellitus (T2DM), all case group members were diabetic, 55% for over five years and 45% for five years or less. The control group had no diabetic members and therefore no data about duration of diabetes were relevant. Based on age analysis, case group respondents were on

*Corresponding author: dlaram.faraj@epu.edu.iq

average older than control group. Mean age in case group was 58.13 years (±10.43), and control group showed a lower mean age of 51.53 years (±10.1). Likewise, median age in case group was 57 years with IQR 18.75, while the control group showed a median of 51 years and an IQR of 18.25. These results indicate that the patients with T2DM in this research are older than their respective non-affected counterparts.

Table 1. Characteristics of the participants in both groups.

Variables	Cases No. (%)	Controls No. (%)
Gender		
Male	24 (40)	10 (33.3)
Female	36 (60)	20 (66.7)
Duration of T2 DM		
≤5 years	27 (45)	
> 5 years	33 (55)	
(Age) Mean ± S.D	58.13 ± 10.43	51.53 ± 10.1
(Age) Median ± IQR	57 ± 18.75	51 ± 18.25

Comparative comparison of cardiovascular biomarkers between the two groups revealed statistically significant differences in two most important parameters: random blood sugar and total cholesterol level. Diabetes subjects for over five years had significantly worse random blood glucoses mean (273.66 mg/dL) than diabetes duration of up to a maximum of five years mean (217.81 mg/dL) with a p-value of 0.009 and a difference in means of 55.85 mg/dL. The implication is that longer diabetes duration is a predictor of worse glycemia control. Likewise, overall cholesterol levels were also significantly higher in the longer-duration group mean (195.51 mg/dL) than in the shorter-duration group mean (173.18 mg/dL) and had a p-value of 0.038. This indicates an increase in lipid profile with passage of time, which may be one of the causations for the rise in risk of cardiovascular complications in diabetic patients having chronic diabetes. The other biomarkers, troponin, triglycerides, and very-low-density lipoprotein (VLDL), were also greater in mean values in the group with longer duration but their p-values (0.07 in the case of troponin and 0.088 for triglycerides and VLDL) were not significant. Also, markers like CK-MB, myoglobin, HDL, and LDL had no differences between the two groups. Although these results were not statistically significant, they indicate trends which may be examined with greater populations (Table 2).

Table 2. Cardiovascular biomarkers of the cases relation with the duration of diabetes

Biomarkers	Duration	N	Mean	SD	P value	Mean difference	95% Confidence Interval of the Difference	
							Lower	Upper
Random blood sugar	> 5 years	33	273.66	86.09	0.009	55.85	14.40	97.29
	≤5 years	27	217.81	71.25				
Troponin	> 5 years	33	1.15	1.64	0.07	0.65	-0.05	1.37
	≤5 years	27	0.49	0.94				
CK-MB	> 5 years	33	8.86	8.85	0.378	-3.83	-12.47	4.81
	≤5 years	27	12.7	22.82				
Myoglobin	> 5 years	33	51.37	52.48	0.392	11.57	-15.29	38.45
	≤5 years	27	39.79	50.79				
Cholesterol	> 5 years	33	195.51	39.01	0.038	22.32	1.32	43.33
	≤5 years	27	173.18	42.12				
Triglyceride	> 5 years	33	206.66	80.01	0.088	33.29	-5.12	71.72
	≤5 years	27	173.37	65.77				
HDL	> 5 years	33	48.39	13.13	0.701	1.32	-5.57	8.22
	≤5 years	27	47.06	13.46				
LDL	> 5 years	33	105.13	48.17	0.5	8.2	-16	32.42
	≤5 years	27	96.9296	44.63802				
VLDL	> 5 years	33	41.32	16.12	0.088	6.7	-1.01	14.41
	≤5 years	27	34.62	13.13				

Table3 presents levels of different cardiovascular and metabolic biomarkers in control patients. Random blood sugar averaged at (107.56 mg/dL ±15.12) and was median (106.5 mg/dL with interquartile range (IQR) 25.25), demonstrating normoglycemia among non-diabetic patients. Troponin was extremely low at an average of (0.05 ng/mL ±0.032) and a median of (0.04 ng/mL), representing lack of acute cardiac injury in the controls.

For cardiac enzymes, CK-MB had an average value of (3.11 U/L ±0.63) and median value of (2.94 U/L), while the range observed for myoglobin was higher with an average value of (15.96 ng/mL ±16.31) and median value of 10.3 ng/ml. The lipid profile markers showed an average value for cholesterol was (154.7 mg/dL ±29.77), and with a median of (152.5 mg/dL). Triglycerides averaged (146.64 mg/dL ±41.35) with the median being greater at (161.5 mg/dL), showing mild variation in lipid level in controls.

High-density lipoprotein (HDL) reflected fairly consistent values with a mean of (48.33 mg/dL ±6.48) and a median of (47.55 mg/dL). Low-density lipoprotein (LDL) values reflected a mean of (142.9 mg/dL ±33.94) and a much higher median of (158 mg/dL), reflecting skewness. Very low-density lipoprotein (VLDL) was (21.37 mg/dL 6.66) and the median was (20.25 mg/dL).

Table 3. Biomarkers for the control group

Biomarker	Mean ± SD	Median ± IQR	SEM
Random blood sugar	107.56 ± 15.12	106.5 ± 25.25	2.76
Troponin	0.05 ± 0.032	0.04 ± 0.06	0.0058
CK-MB	3.11 ± 0.63	2.94 ± 0.7	0.115
Myoglobin	15.96 ± 16.31	10.3 ± 7.68	2.98
Cholesterol	154.7 ± 29.77	152.5 ± 41.25	5.44
Triglyceride	146.64 ± 41.35	161.5 ± 60	7.55
HDL	48.33 ± 6.48	47.55 ± 9.58	1.18
LDL	142.9 ± 33.94	158 ± 60.75	6.20
VLDL	21.37 ± 6.66	20.25 ± 12.08	1.22

Table 4 shows the comparison of the control and case groups for a series of cardiovascular and metabolic biomarkers using the Mann-Whitney U test. The data show a series of statistically significant differences between the two groups, which reflects the effect of diabetes on these biomarkers.

Random blood glucose level recorded much higher median in cases (60.5) compared to controls (15.5) with Z score 7.704 and p value < 0.001 strongly indicative of impaired glycemic control in diabetic patients. Triglycerides also recorded much higher (median: cases = 50.84 and controls = 34.82, Z = 5.881, p = 0.006), which indicates changed lipid metabolism in diabetics.

Both LDL and total cholesterol were significantly greater in cases than controls, with $p < 0.001$ for both, reflecting higher cardiovascular risk in the diabetic group. VLDL was also significantly greater in cases (median = 56.22) than controls (median = 24.07), with Z score = 2.743 and $p < 0.001$.

Troponin I, cardiac injury marker, had a median value in the case group higher than that for the control group (56.88 vs 22.73) with the p-value having a significant magnitude < 0.001 whereas the Z score is negative at -0.535, reflecting heterogeneity rather than direction of values. So, CK-MB and myoglobin, the two myocardial stress markers, were found to be markedly raised in diabetics with Z values of -3.933 and 5.504 and p-values of 0.004 and < 0.001, respectively.

HDL did not, however, reveal any statistically significant disparity between groups ($p = 0.593$), although the median was minutely lower in cases (44.46) than in controls (47.58).

Table 5 results show differences in the rate of abnormal biomarker levels between the case and control groups. Cases showed 35% of the participants having elevated levels of Troponin compared to the controls level while the controls had all within normal Troponin levels, which is a possible indicator of cardiac injury. For CK-MB ,43.3% 0f the diabetics had elevated levels, whereas all control subjects (100%) were within normal levels of CK-MB, reaffirming this result again. In the case of Myoglobin, 30% of the cases were elevated versus only 3.3% of the controls. The cholesterol levels where 40% of the cases have more than the normal cholesterol levels while all the controls were normal in the case of cholesterol levels. In terms of triglycerides (TG), 50% of the patients had increased values versus 3.3% of controls only, indicating an apparent metabolic disorder in the population of patients.

Table 4. Association between controls and cases

Tested Variable	Median (Controls)	Median (Cases)	Z Score	p-value
Random Blood Sugar	15.5	60.5	7.704	< 0.001
Triglycerides (TG)	34.82	50.84	5.881	0.006
Total Cholesterol	31.63	52.43	2.898	< 0.001
HDL	47.58	44.46	4.121	0.593
LDL	60.82	37.84	3.561	< 0.001
VLDL	24.07	56.22	2.743	< 0.001
Troponin I (cTnI)	22.73	56.88	-0.535	< 0.001
CK-MB	34.22	51.14	-3.933	0.004
Myoglobin	29.45	53.53	5.504	< 0.001

In the case of HDL, 21.7% of the cases were elevated versus only 6.7% of the controls. For LDL, 5% of the cases showed elevated levels while the controls had all within normal LDL levels. A striking result is observed with VLDL levels, which have 68.3% of diabetic patients being elevated from normal values compared to only 16.7% of controls.

In general, the case group tended to show a significantly higher frequency of abnormal levels of biomarkers across all groups compared to the control group, which indicates a higher frequency of cardiovascular and metabolic risk factors among the patients. The findings imply that these biomarkers would be valuable for distinguishing between at-risk and healthy controls.

Table 5. Prevalence of biomarkers in both groups

Biomarkers	Cases No. (%)	Control No. (%)
Troponin		
Normal	39 (65)	30 (100)
Higher than normal	21(35)	0
CK-MB		
Normal	34(56.7)	30 (100)
Higher than normal	26(43.3)	0
Myoglobin		
Normal	42 (70)	29 (96.7)
Higher than normal	18(30)	1 (3.3)
Cholesterol level		
Normal	36(60)	30 (100)
Higher than normal	24(40)	0
Tg Level		
Normal	30 (50)	29 (96.7)
Higher than normal	30 (50)	1 (3.3)
HDL		
Normal	47(78.3)	28(93.3)
Higher than normal	13 (21.7)	2 (6.7)
LDL		
Normal	57 (95)	30 (100)
Higher than normal	3 (5)	0
VLDL		
Normal	19(31.7)	25(83.3)
Higher than normal	41(68.3)	5(16.7)

4 DISCUSSION

Type 2 diabetic patients underwent a study to analyze the connection between their elevated blood glucose levels and chronic disease progression and the increase in cardiac biomarkers relative to non-diabetic controls. The study demonstrated how poor blood sugar control and illness duration influence cardiovascular risks because of elevated cardiac injury parameters and blood lipid changes.

Random blood sugar levels together with triglycerides, total cholesterol, LDL, VLDL and cardiac biomarkers troponin I, CK-MB and myoglobin were higher in diabetic patients than in non-diabetic controls according to our study findings. This supported by previous findings, it was claimed that hyperglycemia combined with metabolic disturbances in diabetes causes subclinical myocardial damage which leads to cardiovascular risk [3], [12], [13]. The elevated troponin I levels in diabetic patients which affected most of them indicate silent myocardial damage that is characteristic of diabetic cardiomyopathy [14].

The research data showed clear connections between biomarker progression and diabetes duration length. Patients who had diabetes for more than five years exhibited elevated random blood sugar and total cholesterol levels indicating that long-term hyperglycemia progressively damages vascular endothelium and myocardium thus increasing cardiovascular risk over time [15], [16]. No significant differences existed in troponin and triglyceride levels between diabetics with different disease duration lengths because the study had a small participant count. The existing research demonstrates how diabetic cardiac damage progresses forward and these trends align with this established pattern.

The elevated levels of CK-MB and myoglobin in diabetic patients support the existence of continuous mild myocardial stress which leads to tissue damage even without clear ischemic symptoms. This observation matches previous research which shows that hyperglycemia enhances oxidative stress and endothelial dysfunction and fibrosis to make the myocardium vulnerable to damage [17], [18], [19]

Type 2 diabetes-related dyslipidemia causes elevated LDL and VLDL levels which contribute to atherosclerosis development and coronary heart disease formation [20]. The study revealed no significant difference between diabetics and controls regarding their HDL levels which contradicts typical expectations but could stem from the small control group size or differences in lifestyle factors such as diet and physical exercise.

The study did not measure body weight or BMI but future research should include these parameters to understand obesity-diabetes-cardiovascular biomarker interactions. The parameters must be included in future studies to achieve better risk profiling.

Our research findings demonstrated the heart function effects of hyperglycemic crises specifically diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS). Research demonstrates their ability to cause brief myocardial ischemia or increase cardiac stress [21], [22]. The observed increases of cardiac markers in patients with diabetic complications in this study point to acute metabolic disturbances as the cause of subclinical and overt myocardial damage.

Recent publications show that subclinical inflammation plays a major role in diabetic cardiovascular pathology according to recent research as well as the addition of inflammatory markers in upcoming investigations would help improve diabetes patient cardiovascular risk assessment [23].

The research benefits from using an easily distinguishable diabetic group together with non-diabetic controls and precise measurements of multiple cardiac biomarkers. The study includes several limitations which require mention. The research included a limited number of participants who were further divided into subgroups according to diabetes duration length. The study failed to include factors that could act as confounders including smoking status along with weight and exercise and medication use which includes statins and antihypertensives. The study's cross-sectional nature makes it impossible to determine how cardiovascular results and biomarker changes and hyperglycemia relate to each other.

CLINICAL IMPLICATIONS

The high rate of abnormal cardiac biomarkers in type 2 diabetics demonstrates that cardiovascular screening should be routine for all diabetic patients regardless of symptoms. The use of troponin I and NT-proBNP biomarkers to identify early subclinical myocardial injury enables prompt treatment that could prevent heart failure and ischemic complications from occurring. Blood glucose and lipid control remain essential for preventing cardiovascular disease in diabetic patients.

CONCLUSION

The study results demonstrate that type 2 diabetes mellitus causes significant changes in cardiac biomarkers and lipid parameters which result in early myocardial stress and elevated cardiovascular risk. The study found that elevated troponin I and CK-MB and myoglobin levels and dyslipidemia strongly linked to both high blood sugar levels and longer diabetes duration. The research demonstrates the necessity of immediate heart monitoring and strict metabolic management for diabetic patients to prevent permanent cardiovascular damage. Future longitudinal research should be

conducted to establish the progression of subclinical cardiac injury and develop preventive interventions for high-risk diabetic populations.

FUNDING

The authors claimed no funding source

ACKNOWLEDGEMENT

Thanks for the faculty of health and science especially department of biology.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

DATA AVAILABILITY

The data is available on request.

REFERENCES

[1] R. Goyal, M. Singhal, and I. Jialal, 'Type 2 Diabetes', in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2025. Accessed: Apr. 19, 2025. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK513253/

[2] T. R. Einarson, A. Acs, C. Ludwig, and U. H. Panton, 'Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017', *Cardiovasc. Diabetol.*, vol. 17, no. 1, p. 83, Dec. 2018, doi: 10.1186/s12933-018-0728-6.

[3] N. H. Siam, N. N. Snigdha, N. Tabasumma, and I. Parvin, 'Diabetes Mellitus and Cardiovascular Disease: Exploring Epidemiology, Pathophysiology, and Treatment Strategies', *Rev. Cardiovasc. Med.*, vol. 25, no. 12, Art. no. 12, Dec. 2024, doi: 10.31083/j.rcm2512436.

[4] T. Salvatore *et al.*, 'The Diabetic Cardiomyopathy: The Contributing Pathophysiological Mechanisms', *Front. Med.*, vol. 8, p. 695792, Jun. 2021, doi: 10.3389/fmed.2021.695792.

[5] J. Chen, D. Yin, and K. Dou, 'Intensified glycemic control by HbA1c for patients with coronary heart disease and Type 2 diabetes: a review of findings and conclusions', *Cardiovasc. Diabetol.*, vol. 22, no. 1, p. 146, Jun. 2023, doi: 10.1186/s12933-023-01875-8.

[6] J. Yang *et al.*, 'Fasting Blood Glucose and HbA1c Correlate With Severity of Coronary Artery Disease in Elective PCI Patients With HbA1c 5.7% to 6.4', *Angiology*, vol. 71, no. 2, pp. 167–174, Feb. 2020, doi: 10.1177/0003319719887655.

[7] X.-Y. Wang, F. Zhang, C. Zhang, L.-R. Zheng, and J. Yang, 'The Biomarkers for Acute Myocardial Infarction and Heart Failure', *BioMed Res. Int.*, vol. 2020, p. 2018035, Jan. 2020, doi: 10.1155/2020/2018035.

[8] A. R. Gosmanov, E. O. Gosmanova, and A. E. Kitabchi, 'Hyperglycemic Crises: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State', in *Endotext*, K. R. Feingold, S. F. Ahmed, B. Anawalt, M. R. Blackman, A. Boyce, G. Chrousos, E. Corpas, W. W. de Herder, K. Dhatariya, K. Dungan, J. Hofland, S. Kalra, G. Kaltsas, N. Kapoor, C. Koch, P. Kopp, M. Korbonits, C. S. Kovacs, W. Kuohung, B. Laferrère, M. Levy, E. A. McGee, R. McLachlan, R. Muzumdar, J. Purnell, R. Rey, R. Sahay, A. S. Shah, F. Singer, M. A. Sperling, C. A. Stratakis, D. L. Trence, and D. P. Wilson, Eds., South Dartmouth (MA): MDText.com, Inc., 2021. Accessed: Apr. 19, 2025. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK279052/

[9] G. E. Umpierrez *et al.*, 'Hyperglycaemic crises in adults with diabetes: a consensus report', *Diabetologia*, vol. 67, no. 8, pp. 1455–1479, Aug. 2024, doi: 10.1007/s00125-024-06183-8.

[10] A. Chauin, 'The Main Causes and Mechanisms of Increase in Cardiac Troponin Concentrations Other Than Acute Myocardial Infarction (Part 1): Physical Exertion, Inflammatory Heart Disease, Pulmonary Embolism, Renal Failure, Sepsis', *Vasc. Health Risk Manag.*, vol. 17, pp. 601–617, Sep. 2021, doi: 10.2147/VHRM.S327661.

[11] A. Taghdiri, 'Cardiovascular biomarkers: exploring troponin and BNP applications in conditions related to carbon monoxide exposure', *Egypt. Heart J.*, vol. 76, no. 1, p. 9, Jan. 2024, doi: 10.1186/s43044-024-00446-w.

[12] Y. Fan *et al.*, 'Primordial Drivers of Diabetes Heart Disease: Comprehensive Insights into Insulin Resistance', *Diabetes Metab. J.*, vol. 48, no. 1, pp. 19–36, Jan. 2024, doi: 10.4093/dmj.2023.0110.

[13] W. Widiarti, P. B. T. Saputra, C. G. Savitri, J. N. E. Putranto, and F. F. Alkaff, 'The impact of cardiovascular drugs on hyperglycemia and diabetes: a review of 'unspoken' side effects', *Hellenic J. Cardiol.*, p. S1109966624002082, Oct. 2024, doi: 10.1016/j.hjc.2024.09.007.

[14] E. P. Odum and E. E. Young, 'Elevated cardiac troponin I, creatine kinase and myoglobin and their relationship with cardiovascular risk factors in patients with type 2 diabetes', *Diabetes Metab. Syndr. Clin. Res. Rev.*, vol. 12, no. 2, pp. 141–145, Apr. 2018, doi: 10.1016/j.dsx.2017.12.001.

[15] Y. Li *et al.*, 'Diabetic vascular diseases: molecular mechanisms and therapeutic strategies', *Signal Transduct. Target. Ther.*, vol. 8, no. 1, p. 152, Apr. 2023, doi: 10.1038/s41392-023-01400-z.

[16] A. V. Poznyak, L. Litvinova, P. Poggio, V. N. Sukhorukov, and A. N. Orekhov, 'Effect of Glucose Levels on Cardiovascular Risk', *Cells*, vol. 11, no. 19, p. 3034, Sep. 2022, doi: 10.3390/cells11193034.

[17] A. Caturano *et al.*, 'Oxidative Stress and Cardiovascular Complications in Type 2 Diabetes: From Pathophysiology to Lifestyle Modifications', *Antioxidants*, vol. 14, no. 1, p. 72, Jan. 2025, doi: 10.3390/antiox14010072.

[18] P. González, P. Lozano, G. Ros, and F. Solano, 'Hyperglycemia and Oxidative Stress: An Integral, Updated and Critical Overview of Their Metabolic Interconnections', *Int. J. Mol. Sci.*, vol. 24, no. 11, p. 9352, May 2023, doi: 10.3390/ijms24119352.

[19] M. Wang, Y. Li, S. Li, and J. Lv, 'Endothelial Dysfunction and Diabetic Cardiomyopathy', *Front. Endocrinol.*, vol. 13, p. 851941, Apr. 2022, doi: 10.3389/fendo.2022.851941.

[20] S. Chakraborty, A. Verma, R. Garg, J. Singh, and H. Verma, 'Cardiometabolic Risk Factors Associated With Type 2 Diabetes Mellitus: A Mechanistic Insight', *Clin. Med. Insights Endocrinol. Diabetes*, vol. 16, p. 11795514231220780, Jan. 2023, doi: 10.1177/11795514231220780.

[21] A. R. Gosmanov, E. O. Gosmanova, and A. E. Kitabchi, 'Hyperglycemic Crises: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State', in *Endotext*, K. R. Feingold, S. F. Ahmed, B. Anawalt, M. R. Blackman, A. Boyce, G. Chrousos, E. Corpas, W. W. de Herder, K. Dhatariya, K. Dungan, J. Hofland, S. Kalra, G. Kaltsas, N. Kapoor, C. Koch, P. Kopp, M. Korbonits, C. S. Kovacs, W. Kuohung, B. Laferrère, M. Levy, E. A. McGee, R. McLachlan, R. Muzumdar, J. Purnell, R. Rey, R. Sahay, A. S. Shah, F. Singer, M. A. Sperling, C. A. Stratakis, D. L. Trence, and D. P. Wilson, Eds., South Dartmouth (MA): MDText.com, Inc., 2000. Accessed: Apr. 26, 2025. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK279052/

[22] E. M. Hassan *et al.*, 'Overlap of diabetic ketoacidosis and hyperosmolar hyperglycemic state', *World J. Clin. Cases*, vol. 10, no. 32, pp. 11702–11711, Nov. 2022, doi: 10.12998/wjcc.v10.i32.11702.

[23] A. V. Mureșan *et al.*, 'Inflammatory Markers Used as Predictors of Subclinical Atherosclerosis in Patients with Diabetic Polyneuropathy', *Life*, vol. 13, no. 9, p. 1861, Sep. 2023, doi: 10.3390/life13091861.