KARBALA JOURNAL OF MEDICINE

Research Article

Association of Lipid Profile, high-Sensitivity Cardiac Troponin and Haemoglobin A1c in Diabetic Patients with and without ischemic heart disease

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Article information:
Received: 14-05-2025
Accepted: 20-06-2025
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https://doi.org/10.70863/karbalajm.v18i1.3846

Abstract

Background: Type 2 diabetes mellitus (T2DM) increases oxidative stress, with dyslipidemia, specifically uncontrolled T2DM, along with increased body mass index (BMI), all these factors increasing the risk of cardiovascular disease. The interplay between lipid profile, high-sensitivity cardiac troponin I (hs-cTnI), Hemoglobin A1c (HbA1c), and BMI in diabetic patients with is-chemic heart disease IHD remains critical for risk stratification and management. The study aimed measurement of lipid profile, hs-cTnI, HbA1c levels, and BMI as predictive biomarkers for ischemic heart disease in diabetic patients.

Methods: This case-control study was conducted at the Department of Microbiology, College of Medicine at the University of Kerbala in collaboration with the Cardiology Unit at Karbala Teaching Hospital. A total of 180 individuals aged 35–65 years were enrolled, including 120 patients with T2DM (60 with myocardial ischemia and 60 without) and 60 age- and sex-matched healthy controls. Blood samples were collected to measure HbA1c and lipid profile.

Results: Patients with T2DM and ischemia showed significantly higher levels of HbA1c, low-density lipoprotein cholesterol (LDL-C), triglycerides, and very low-density lipoprotein cholesterol (VLDL-C), and lower high-density lipoprotein cholesterol (HDL-C) than non-ischemic diabetic patients and controls ($p < 0.05$). HbA1c positively correlated with lipid abnormalities. There are no significant differences in hs-cTnI levels between diabetic patients without ischemia com-pared to controls

Conclusions: Elevated HbA1c and lipid derangements are strongly associated with myocardial ischemia in T2DM patients. These biomarkers can serve as early indicators of cardiovascular risk. Tight glycemic and lipid control should be emphasized to prevent ischemic complications in diabetic populations.

Keywords: Type 2 diabetes mellitus, Ischemic heart disease, HbA1c, Lipid profile, BMI, hs-cTnI

Introduction

Myocardial ischemia is a transient and reversible reduction in oxygen supply to the myocardium, resulting in an imbalance between myocardial oxygen demand and supply. It is responsible for the initial hemodynamic (elevated end diastolic left ventricular pressure and kinetic change), and change in the metabolic (lactate production), electric (repolarisation), and clinical (chest pain) [1]. Myocardial ischemia is a condition in which cardiomyocytes suffer as a result of a decrease in coronary blood flow in comparison to their metabolic demands, and it can manifest as a variety of clinical disorders [2]. Coronary artery disease (CAD) has a complex aetiology and other significant risk factors, including diabetes, which is one of the most modifiable risk factors. The prognosis is bleak, and diabetes increases the risk of developing heart disease. Cardiovascular disease (CVD)-related mortality is four times higher in diabetic women than in diabetic men [3].

The relation between DM and ischemic heart disease is that DM significantly increases the risk of ischemic heart disease (IHD), with both conditions being tightly interlinked through shared pathophysiological mechanisms such as

atherosclerosis, endothelial dysfunction, and chronic inflammation. The relationship between DM and ischemic heart disease (IHD) is multifaceted, in which DM serves as a significant risk factor for IHD. DM contributes to IHD development through atherosclerosis, chronic inflammation, and endothelial dysfunction [4]. Globally, the prevalence of ischemic heart disease continues to rise, particularly in low- and middle-income countries, largely driven by increasing rates of diabetes, obesity, and sedentary lifestyles. As such, the intersection of metabolic disorders and cardiovascular pathology represents a major public health challenge. In particular, type 2 diabetes mellitus (T2DM) has emerged as a dominant modifiable risk factor that accelerates atherosclerosis and contributes to the development of cardiovascular complications [5]. T2DM is a chronic metabolic disorder characterized by insulin resistance, relative insulin deficiency, and persistent hyperglycemia. It is well-established that T2DM significantly elevates the risk of atherosclerotic cardiovascular disease, including myocardial infarction and stroke. The Framingham Heart Study and other large-scale cohort investigations have demonstrated that diabetic individuals are at a two- to four-fold increased risk of developing cardiovascular diseases compared to non-diabetic individuals [6-7]. Moreover, diabetic patients often exhibit a constellation of metabolic disturbances, including poor glycemic control, obesity, dyslipidemia, and systemic inflammation, all of which accelerate atherogenesis and endothelial dysfunction [8]. Among these risk factors, HbA1c reflects long-term glycemic control and is widely considered a predictor of microvascular and macrovascular complications. Elevated HbA1c levels are associated with increased oxidative stress and inflammation, which contribute to vascular damage [9]. Simultaneously, diabetic dyslipidemia, characterized by elevated triglycerides, high-density lipoprotein (HDL), and increased small, dense low-density lipoprotein (LDL) particles, exacerbates cardiovascular risk [10]. Additionally, body mass index (BMI), particularly when reflecting central obesity, serves as a modifiable predictor of both T2DM and IHD [11].

Despite the established individual associations of HbA1c, lipid profile, and BMI with cardiovascular disease, few studies have simultaneously assessed their interrelationship in the context of myocardial ischemia in diabetic patients. Therefore, the current study aims to evaluate the association of lipid profile with HbA1c levels and BMI in diabetic patients suffering from ischemic heart disease compared to diabetic patients without ischemia and healthy controls. Understanding these interrelationships is essential for refining cardiovascular risk assessment and developing targeted interventions for high-risk diabetic populations. The study seeks to answer the following question: Can a combination of HbA1c, lipid profile, hs-cTnI, and BMI improve the early detection and risk stratification of ischemic heart disease in diabetic patients?

Materials and Methods

Patients

This case-control study was conducted at the Department of Microbiology, College of Medicine, University of Karbala, in collaboration with the Cardiology Unit at Imam AL-Hussein Teaching Medical City Hospital in Karbala City in Iraq, between November 19, 2024, and February 18, 2025. A total of 180 individuals were enrolled in the study, with ages ranging from 35 to 65 years, divided into two groups: 120 patients with T2DM (subdivided into two subgroups, 60 samples with ischemic heart disease (IHD) along with T2DM and 60 samples with T2DM only without ischemic heart disease). The second group consisted of 60 healthy individuals as a control group, that age and sex-matched. Diagnosis of T2DM and IHD was based on American Diabetes Association (ADA) and American Heart Association (AHA) criteria, respectively, supported by clinical presentation and electrocardiogram (ECG) findings. Anthropometric and clinical data were recorded, including age, sex, residence, smoking status, height, weight, and body mass index (BMI). BMI was classified according to WHO criteria as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30 kg/m²).

Inclusion criteria: All patients with T2DM and IHD were enrolled, and also healthy individuals as a control group.

Exclusion criteria: Excluded persons include those who are smokers and alcoholics, and patients with chronic disease, such as chronic kidney disease, autoimmune and inflammatory diseases (systemic lupus erythematosus, rheumatoid arthritis, and psoriatic arthritis), infection cases, and myocardial infarction.

Sample collection and analysis

Blood was collected from participants. Samples were collected in gel tubes, allowed to clot at room temperature for 30 minutes, and then centrifuged at 3000 rpm for 10 minutes. The resulting sera were

stored at –20°C until analysis. All procedures were strictly followed according to the manufacturer's protocol. Samples were analyzed by biochemical tests, which were included lipid profile parameters, including serum total cholesterol (normal range: <200 mg/dL), high-density lipoprotein (HDL; normal: >40 mg/dL for males and >50 mg/dL for females), triglycerides (normal: <150 mg/dL), and low-density lipoprotein (LDL), calculated using the Friedewald equation. The analyses were conducted using the Dimension-RXL fully automated chemistry autoanalyzer (Siemens Healthcare Diagnostics Inc., USA). Fasting blood glucose (FBG; normal range: 70–99 mg/dL) levels and symptoms of chest pain were also assessed.

High-sensitivity cardiac troponin I assay

High-sensitivity cardiac troponin I (hs-cTnI) levels were determined using a quantitative sandwich ELISA kit supplied by Bioassay Technology Laboratory (Shanghai, China). The assay detection range was 0.1–100 ng/mL, with a sensitivity of <0.05 ng/mL, as per the manufacturer's datasheet. Absorbance was measured at 450 nm using a microplate reader (Biotek ELx800, USA). A standard curve was generated using the provided calibrators, and sample concentrations were calculated accordingly. The upper reference limit (URL) was considered <0.04 ng/mL, consistent with current clinical guidelines for hs-cTnI interpretation.

Ethical Consideration

All research participants were informed and allowed to give their consent before sample collection. By document number 103 on 16 January 2025, a local college and hospital ethics committee reviewed and approved the protocol, subject information, and consent form. All participants provided written informed consent before their inclusion in the study. In the forms, the patients have given their consent for their clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity.

Statistical analysis

The statistical analysis was carried out using the Statistical Package for Social Sciences (SPSS, IBM, version 26.0). The data distribution's normality was assessed using the Shapiro-Wilk and Kolmogorov-Smirnov tests. Mean ± standard deviation (SD) is a representation of continuous data having a normal distribution. A one-way ANOVA test was used to compare the means of each group. Spearman correlation was used to perform correlations between variables. A value of $P \leq 0.05$ is deemed significant.

Results

A statistically significant difference was found in mean age or BMI between the patient and control groups (Table 1). The level of HbA1c was significantly elevated in diabetic patients compared to control groups, as shown in Table 2. The level of hs-cTnI is elevated in the diabetic patient group with ischemia in comparison with diabetic patients without ischemia and a higher in diabetic patients than in healthy controls. However, there is a non-significant difference between diabetic patients without ischemia and controls, as shown in Table 3. The results also showed a statistically significant increase ($p<0.01$) in total cholesterol (TC), TG, LDL-C, and VLDL-C, while serum HDL-C level was found to a significantly decreased ($p<0.01$) (Table 4).

Table 1: Mean ± Standard Deviation of age and BMI in diabetic patients and control groups

Parameter	Groups	NO.	Mean ± SD	Multiple comparison	p-value
Age (years)	Diabetic patients with ischemia	60	57.49 ±7.3	Diabetic patients with ischemia+, Diabetic patients without ischemia	0.003
	Diabetic patients without ischemia	60	53.22 ±9.05	Diabetic patients with ischemia+ Controls	<0.001
	controls	60	50.31± 6.32	Diabetic patients without ischemia + controls	0.039
BMI	diabetic patients with ischemia	60	24.69 ± 2.03	Diabetic patients with ischemia, diabetic patients without	0.001
	Diabetic patients without ischemia	60	31.27 ± 8.76	Diabetic patients with ischemia+ Controls	0.059
	controls	60	26.56 ± 2.04	Diabetic patients without ischemia + Controls	<0.001

BMI: Body mass index

Table 2: Mean ± Standard Deviation HbA1C in diabetic patients and control groups

Parameter	Groups	No.	Mean ± SD	Multiple comparison	p-value
HbA1c mmol/mol	Diabetic patients with ischemia	60	8.19 ±1.46	Diabetic patients with ischemia+, Diabetic patients without ischemia	0.02
	Diabetic patients without ischemia	60	9.05 ± 2.05	Diabetic patients with ischemia+ Controls	<0.05
	controls	60	5..24 ± 0.39	Diabetic patients without ischemia + controls	<0.05

HbA1c: Hemoglobin A1c

Table 3: Mean ± Standard Deviation of High Sensitive Troponin in patients and control groups

Parameter	Groups	No.	Mean ± SD	Multiple comparison	p-value
hs-cTnI (ng/mL)	Diabetic patients with ischemia	60	9.2 ± 1.86	Diabetic patients with ischemia+, Diabetic patients without ischemia	<0.05
	Diabetic patients without ischemia	60	5.1 ± 0.63	Diabetic patients with ischemia+ Controls	<0.05
	controls	60	5.6 ± 2.0	Diabetic patients without ischemia + controls	0.094

hs-cTnI: High-sensitivity cardiac troponin I

Table 4: Mean ± Standard Deviation of Lipid Profiles in patients and control groups

Parameter	Groups	No.	Mean ± SD	Multiple comparison	p-value
Cholesterol (mg/dL)	Diabetic patients with ischemia	60	187.96 ± 17.7	Diabetic patients with ischemia+, Diabetic patients without ischemia	<0.05
	Diabetic patients without ischemia	60	215.5 ± 11.8	Diabetic patients with ischemia+ Controls	<0.05
	controls	60	167.2 ± 18.7	Diabetic patients without ischemia + controls	<0.05
TG (mg/dL)	Diabetic patients with ischemia	60	143.3 ± 41.2	Diabetic patients with ischemia, diabetic patients without M-I	<0.05
	Diabetic patients without ischemia	60	185.9 ± 22.19	Diabetic patients with ischemia+ Controls	<0.05
	controls	60	103.1 ± 16.4	Diabetic patients without ischemia + controls	<0.05
LDL (mg/dl)	Diabetic patients with ischemia	60	99.8 ± 25.8	Diabetic patients with ischemia+, diabetic patients without M-I	<0.05
	Diabetic patients without ischemia	60	138.7± 20.5	Diabetic patients with ischemia+ Controls	<0.05
	Controls	60	83.15± 12.8	Diabetic patients without ischemia + controls	<0.05
HDL (mg/dL)	Diabetic patients with ischemia	60	47.6 ± 8.6	Diabetic patients with ischemia, diabetic patients without M-I	0.061
	Diabetic patients without ischemia	60	49.8± 4.6	Diabetic patients with ischemia+ Controls	0.093
	Controls	60	49.59± 4.9	Diabetic patients without ischemia + controls	0.874
VLDL (mg/dL)	Diabetic patients with ischemia	60	28.6± 8.2	Diabetic patients with ischemia, diabetic patients without M-I	<0.05
	Diabetic patients without ischemia	60	40.8± 8.1	Diabetic patients with ischemia+ Controls	<0.05
	Controls	60	20.6± 3.3	Diabetic patients without ischemia + controls	<0.05

TG: Triglycerides, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, VLDL: Very Low Density Lipoprotein

Discussion

BMI in the present study had significantly increased in diabetic patients without ischemia, and this is important because of obesity, particularly central obesity, linked with diabetes, along with ischemia progression [12-13]. This study showed an increase in HbA1c in diabetic patients, indicating that the patient has uncontrolled diabetes, which results in several problems. Macrovascular disease, which ultimately leads to myocardial infarction, is one of the most significant consequences. High HbA1c should thus be regarded as a risk factor for death; therefore, every 1% increase in HbA1c, the risk of ischemia rises by approximately 10%, and aggressive care should be

implemented to ensure that this population's HbA1c level stays precisely below 7% [14].

The results of this study agree with Sonia Butalia *et al.* (2024), who mentioned in their study that both men and women with HbA1c ≥6.0% were associated with an increased risk of CVD and mortality outcomes [15]. Diabetes mellitus is considered a condition that strongly affects the vascular system, leading to both microvascular and macrovascular complications [16]. Microvascular problems begin to occur long before the patient has obvious diabetes, as is widely recognized. Hyperglycemia is one independent risk factor for cardiovascular diseases. By creating glycated proteins and products, hyperglycemia accelerates the atherosclerosis process by inducing endothelial dysfunction, leading to macrovascular problems [17]. In acute metabolism of coronary syndromes, altered glucose stress hyperglycemia usually occurs owing to elevated catecholamine levels [18]. An approach looking just at plasma glucose levels at the time of an acute myocardial infarction (AMI) cannot be utilized to predict the outcome, as stress hyperglycemia. Glycosylated hemoglobin (HbA1c) readings might thus indicate diabetes in AMI situations [19].

A recent study found that higher HbA1c levels forecast cardiovascular disease and death in non-diabetic patients independent of fasting glucose levels, suggesting that long-term glycol-metabolic disturbance in the sub-diabetic range also increases cardiovascular disease risk [20]. Over 7% HbA1c is linked to a significant increase in the risk of cardiac events and fatalities [21]. Reddy *et al.* (2024) concluded that significant correlation between high-sensitivity C-reactive protein (hs-CRP) and HbA1c levels in patients with AMI and T2DM, with both biomarkers serving as strong predictors of six-month mortality. HbA1c, because of its positive correlation with hs-CRP, could be used as an independent marker for assessing the risk of adverse outcomes in these patients [22].

The cardiac troponin is raised when the heart becomes affected either by angina or myocardial infarction, so in this study, its level was elevated in patients with ischemia compared with other groups. This is due to the blockage of blood vessels that supply the heart with oxygen, leading to tissue death in the heart muscle. This can be detected by particular cardiac markers, especially troponin, due to their release into the bloodstream upon cardiac muscle damage. Most of the labs across the globe now routinely evaluate the usual chest discomfort using hs-cTnI tests. An early increase in hs-cTnI could shorten the time required to identify myocardial infarctions, which is crucial for patients arriving in emergency rooms with chest discomfort [23]. The most recent European Society of Cardiology Guidelines not only acknowledged their key importance in the diagnostic algorithm but also advised their usage for quick rule-in/rule-out of ischemia [24]. Not only in a broad range of different cardiovascular diseases (CVD) but also in several non-CVD disorders. High-sensitivity cardiac troponins have strong predictive indicators for long-term events and death. Furthermore, these biomarkers turned into a potent weapon in certain groups, such as pediatric patients and, most recently, COVID-19 sufferers [25]. Though much studied, the evaluation and reading of the hs-cTnI changes remain difficult in patients with baseline elevation, like CKD or severely sick conditions [26-27]. The early identification of myocardial damage has been greatly improved by the hs-cTnI test, which has also helped to diagnose myocardial infarction [28]. Through expedited diagnostic methods, these tests enable quicker clinical decision-making and have raised awareness of all kinds of cardiac damage. Based just on troponin findings, difficulties still exist in distinguishing between different causes of myocardial damage; the adoption of high-sensitivity testing has not always resulted in better clinical results [29-30].

Lipid metabolism is known to be dysregulated in T2DM, and traditional lipid markers, such as total cholesterol, high-density lipoprotein cholesterol, triglycerides, and low-density lipoprotein cholesterol, are used to assess the risk of future cardiovascular events. In this study, the increased concentrations of lipid profiles in patients in comparison with controls, except HDL-C, were lower in patients. Since lipids are essential for the formation of coronary atherosclerosis lesions, achieving a significant decrease in lipid-related risk has long been a key component of primary and secondary prevention. Frequently display an atherogenic lipid profile, which elevates their risk for cardiovascular disease [31]. This is due to heightened oxidation susceptibility. So, the diabetic dyslipidemia is a major cause of oxidative stress, which promotes and accelerates atherosclerosis and thus, end-organ damage AMI [32].

This study has several limitations that should be noted. First, the cross-sectional design limits the ability to establish causal relationships between the investigated biomarkers and ischemic heart disease. Second, as the research was conducted at a single centre, the findings may not be generalizable to broader populations with differing demographic

or clinical characteristics. Additionally, the possibility of selection bias cannot be excluded, as participants were selected based on specific inclusion criteria, which may not reflect the full representation of diabetic patients in the general population. Future multicenter, longitudinal studies are recommended to validate these findings and explore causality.

Conclusions

This study demonstrates that there are statistically significant differences in clinical and biochemical parameters between diabetic patients with ischemia, diabetic patients without ischemia, and healthy controls. Elevated HbA1c levels were strongly associated with the presence of ischemia in diabetic patients, highlighting its role as a predictor for cardiovascular complications, as in high-sensitivity cardiac troponin (hs-cTnI). Lipid profile abnormalities, including increased TC, TG, LDL-C, and VLDL-C levels, along with decreased HDL-C, further emphasize the dyslipidemia burden in diabetic individuals, contributing to ischemic risk. These findings support the use of HbA1c and hs-cTnI as valuable biomarkers in assessing cardiovascular risk in diabetic patients and suggest that aggressive glycemic and lipid control should be prioritized to prevent ischemic events. Future studies are recommended to validate these biomarkers in larger populations and to explore interventional strategies targeting early metabolic and cardiac markers in diabetic patients.

Funding: There is no funding for this research.
Conflict of interest: The authors state that there is no conflict of interest.
Author contributions: Conceptualization; D.M.H., Methodology: D.M.H., Formal analysis; A.H.A., Investigation: A.H.A., Writing; A.M.A.; Resource: A.M.A., Supervision: A.S.A.

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