ORIGINAL RESEARCH

Exploring the Correlation Between Lipid Profiles and Diabetes Mellitus Among Individuals Affected by Acute Myocardial Infarction

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ABSTRACT

Background:This research aimed to explore the correlation between lipid levels and diabetic indices among confirmed patients experiencing myocardial infarction, comparing those with and without type II diabetes mellitus.**Methods**:A total of 400 individuals experiencing acute myocardial infarction with ST-segment elevation were included in this study. The participants were divided into two groups: those with diabetes mellitus type II and those without. Lipid levels and diabetic indices were assessed, and the relationships between these variables were thoroughly examined.**Results**:Triglycerides demonstrated positive correlations with fasting blood sugar, while low-density lipoprotein showed positive correlations with insulin levels when assessed across myocardial infarction patients. In the subgroup analysis comparing myocardial infarction patients with and without diabetes mellitus type II, negative correlations emerged between triglycerides and glycosylated hemoglobin (HbA1C), low-density lipoprotein and glycosylated hemoglobin, as well as glycosylated hemoglobin and insulin. **Conclusion**:Positive associations were observed between triglycerides and fasting blood sugar, as well as between low-density lipoprotein and insulin levels in the overall assessment of myocardial infarction patients. In the subgroup analysis, comparing patients with and without diabetes mellitus type II, inverse relationships were identified between triglycerides and glycosylated hemoglobin (HbA1C), low-density lipoprotein and glycosylated hemoglobin, and glycosylated hemoglobin and insulin.

Keywords: Myocardial infarction, Lipid profile, Diabetes Mellitus

INTRODUCTION

The onset of acute myocardial infarction (AMI) is attributed to the disruption in coronary artery blood supply, primarily arising from underlying coronary artery diseases. This interruption leads to a state of serious and sustained ischemia. The clinical presentation of AMI is characterized by persistent and severe pain located in the posterior aspect of the chest, alongside elevated levels of myocardial enzymes detected in the bloodstream.[1] Furthermore, discernible changes in the electrocardiogram are observed, potentially progressing to severe arrhythmias, shock, heart failure, or, in extreme cases, even death.In the context of China, the prevalence of AMI is noteworthy. The admission rate for individuals experiencing AMI is reported at 6%, indicating a substantial impact on healthcare resources. Moreover, the long-term mortality rate among these patients stands at 12%, underscoring the gravity of AMI as a significant health concern in the Chinese population. These statistics emphasize the critical importance of understanding and addressing the factors contributing to AMI for both immediate medical interventions and long-term preventive strategies.

Coronary artery diseases (CAD) arise from a multitude of risk factors, with advancing age, high blood pressure, cigarette smoking, hyperlipidemia, diabetes mellitus, inflammatory reactions, and other variables contributing to their development.[2] Diabetes Mellitus Type II (DMT2) is particularly noteworthy in this context, as it is intricately connected to dyslipidemia – a condition characterized by altered lipid levels.Examining the lipid profile in individuals with pre-diabetes in comparison to those without

diabetes reveals a distinctive pattern. Pre-diabetic individuals exhibit heightened levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), coupled with diminished levels of high-density lipoprotein cholesterol (HDL-C). This aberrant lipid profile suggests an atherogenic tendency, indicating an increased susceptibility to cardiovascular complications.[3]

In cases of insulin resistance, a hallmark of DMT2, disturbances extend to the levels of very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). The intricate interplay between diabetes and dyslipidemia is further substantiated by numerous studies, revealing a compelling association between morbidity linked to DMT2 and cardiovascular diseases. Dyslipidemia-induced changes in the size or density of low-density lipoprotein (LDL) emerge as a pivotal factor, underscoring the complexity of these metabolic interactions and their profound implications for cardiovascular health. Understanding these intricacies is crucial for developing effective preventive strategies and interventions aimed at reducing the risk of coronary artery diseases in individuals with diabetes mellitus type II.[4]

The formation of small dense LDL (low-density lipoprotein) particles is intricately tied to the clearance of larger very low-density lipoprotein (VLDL) from the bloodstream. This process unfolds through a series of metabolic events, primarily involving lipolysis – the enzymatic breakdown of lipids. Notably, lipolysis operates through the breakdown of specific larger VLDL precursors, ultimately yielding smaller, denser LDL particles.[5]The intricacies of lipoprotein metabolism are underscored by the identification of seven distinct LDL subspecies, each characterized by unique metabolic behaviors and implicated roles in pathological processes. This diversity highlights the complexity of lipid transport and metabolism within the body.Hepatic lipase, an enzyme with significant regulatory functions, plays a crucial role in this lipid metabolism cascade. As part of its lipolytic action, hepatic lipase facilitates the breakdown of phospholipids and triglycerides (TGs) present in larger VLDL particles. This breakdown results in the generation of smaller, denser LDL particles, contributing to the intricate dynamics of lipoprotein metabolism.This process of lipolysis not only influences the catabolism of larger VLDL precursors but also induces a transformation in their composition, leading to the production of small dense LDL particles. Understanding these molecular processes is pivotal in unraveling the complex interplay between lipoprotein subspecies, their metabolic pathways, and their implications for cardiovascular health. This knowledge serves as a foundation for developing targeted interventions and therapeutic strategies to address lipid-related disorders and mitigate cardiovascular risks associated with the presence of small dense LDL particles.[6,7]

One crucial characteristic that renders high-density lipoprotein (HDL) cardio-protective lies in its anti-inflammatory and anti-oxidative properties, along with its ability to enhance the efflux of cellular cholesterol. This unique combination of attributes positions HDL as a significant factor in mitigating cardiovascular risk.[8] Notably, decreased levels of HDL-cholesterol often coincide with elevated triglyceride (TG) levels, thereby amplifying the likelihood of coronary heart disease (CHD).The relationship between HDL, insulin, and TGs adds another layer of complexity. Reduced HDL levels are frequently associated with increased levels of insulin and TGs, forming an interrelated triad that contributes to cardiovascular risk. The intricate balance of these lipid and metabolic factors plays a pivotal role in shaping the overall cardiovascular profile.Furthermore, the atherogenic potential of small dense low-density lipoprotein (LDL) adds to the complexity of lipid-related cardiovascular risks. Small dense LDL particles exhibit a tendency to invade the sub-endothelial space, rendering them more susceptible to oxidative modifications. Additionally, their increased binding to arterial wall proteoglycans and reduced affinity for LDL receptors contribute to their atherogenic potential.[9] This intricate interplay highlights the multifaceted nature of lipid metabolism and its impact on cardiovascular health, emphasizing the need for comprehensive strategies that address not only cholesterol levels but also the inflammatory and oxidative components associated with atherogenesis.

MATERIALS AND METHODS

The criteria employed for the selection of patients in this study were rigorously defined based on the diagnostic parameters indicative of acute myocardial infarction (AMI). Specifically, individuals were included if they exhibited ST-segment elevation and elevated levels of Troponin T, two key markers associated with myocardial infarction. This stringent approach aimed to ensure the homogeneity of the study cohort, enhancing the reliability and specificity of the findings related to this critical cardiac event.Equally important were the exclusion criteria, meticulously designed to eliminate potential confounding factors that could influence the study outcomes. Patients presenting with acute metabolic complications, including hypoglycemia, diabetic ketoacidosis, and hyperglycemic states, were excluded. Likewise, individuals with a history of inherited disorders or a family background of dyslipidemia, those with a documented history of cerebrovascular accidents, individuals exhibiting deranged liver functions, and those currently undergoing acute infections were not considered for inclusion. By excluding individuals with these specific conditions, the study sought to isolate and analyze the impact of AMI within a more controlled

and defined patient population.In addition to these clinical considerations, the ethical aspect of the research was emphasized through the mandatory requirement of written informed consent from each participant. This ethical safeguard ensured that individuals fully understood the nature and objectives of the study and voluntarily agreed to participate, underscoring the paramount importance of respecting participants' autonomy and protecting their rights throughout the research process. This rigorous and ethical approach to patient selection and study conduct serves as a foundation for generating meaningful and reliable insights into the relationship between AMI, diagnostic criteria, and associated factors.

Various biochemical parameters were assessed in this study using specific methodologies to obtain accurate and comprehensive data. The determination of fasting blood glucose levels was carried out through the glucose oxidase method, employing a commercial kit from Merck. HbA1C levels were measured using an automated kit on Cobas Integra, provided by Roche, offering a standardized and efficient approach to assess long-term glycemic control. Serum insulin levels were quantified through the radioimmunoassay (RIA) method, utilizing a kit from Merck. For triglycerides, an enzymatic kit method from Merck was employed, ensuring precise measurements of this crucial lipid parameter. Serum cholesterol levels were determined using a similar enzymatic kit method from Merck, maintaining consistency in the analytical approach. Furthermore, HDL-cholesterol levels were measured using an enzymatic kit method from Merck, providing insights into the protective lipoprotein fraction.Calculation of LDL-cholesterol was performed using the Friedewald formula, a widely accepted method for estimating this critical lipid parameter. The utilization of standardized kits and well-established formulas ensures accuracy and comparability of results across different measurements. In terms of diagnostic imaging, angiography was conducted using a TOSHIBA Infinix 2000 system, with the procedure overseen by a consultant cardiologist. This advanced imaging technique allowed for a detailed assessment of coronary arteries, aiding in the diagnosis and characterization of cardiovascular conditions. Overall, the combination of precise biochemical assays and advanced diagnostic imaging techniques contributes to a robust and comprehensive evaluation of the participants in the study.

RESULTS

The study cohort consisted of individuals aged between 40 and 65 years, with an average age of 56±3 years. Among the participants, there were 262 males and 138 females, creating a diverse sample to examine the association between myocardial infarction (MI) and diabetes mellitus type 2 (DMT2). The study further categorized the participants into two groups: 200 individuals with both MI and DMT2 and another 200 with MI but without DMT2.

Table 1 presents a comprehensive overview of the baseline and physical parameters for the two groups. The statistical analysis revealed that age and the duration of diabetes, as well as diastolic blood pressure (DBP), did not show significant differences between the two groups. However, notable distinctions were observed in body mass index (BMI) and systolic blood pressure (SBP). MI patients with DMT2 exhibited significantly higher levels of BMI and SBP ($P<0.001$) compared to their counterparts without DMT2.Additionally, the distribution of gender and smoking habits did not exhibit significant differences when subjected to the Chi-Square test, ensuring a balanced representation of these factors across both groups. These baseline and physical parameters provide a valuable foundation for assessing the impact of diabetes mellitus type 2 on individuals experiencing myocardial infarction, offering insights into potential associations and risk factors.

Table 1: Baseline and Physical Parameters of Patients with and without diabetes mellitus type II (T2DM)

	Without T2DM	**with T2DM**	**P-Value**
N	**200**	**200**	**-**
Gender (Male /Female)	136 / 64	126 / 74	0.457
Age (Years)	55 ± 4	56 ± 3	0.079
Height (m)	1.55 ± 0.01	1.54 ± 0.01	0.201
Weight (Kg)	68.22 ± 1.29	70.47 ± 2.49	0.001
Body Mass Index (BMI) (kg/m2)	27.57 ± 2.2	29.47 ± 4.71	0.001
Duration of Diabetes Mellitus (Years)	-	12 ± 3	-
Smoking	40	50	0.404
Systolic Blood Pressure (mmHg)	130 ± 5	129 ± 8	0.001
Diastolic Blood Pressure (mmHg)	80 ± 4	81 ± 6	0.012

Figure 1: Baseline and Physical Parameters of Patients with and without diabetes mellitus type II (T2DM)

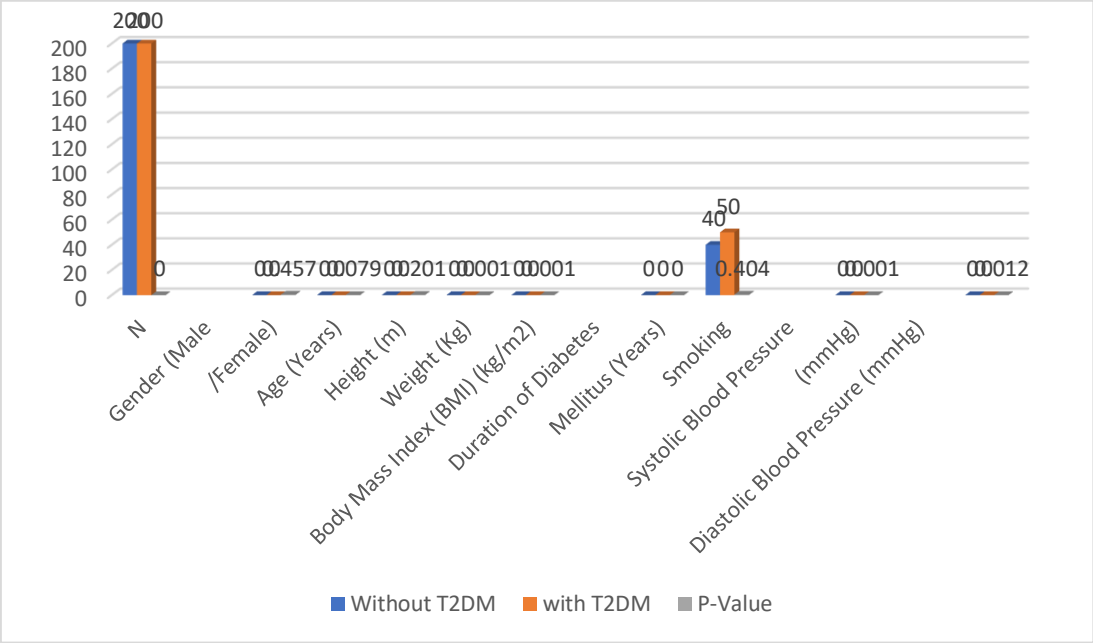
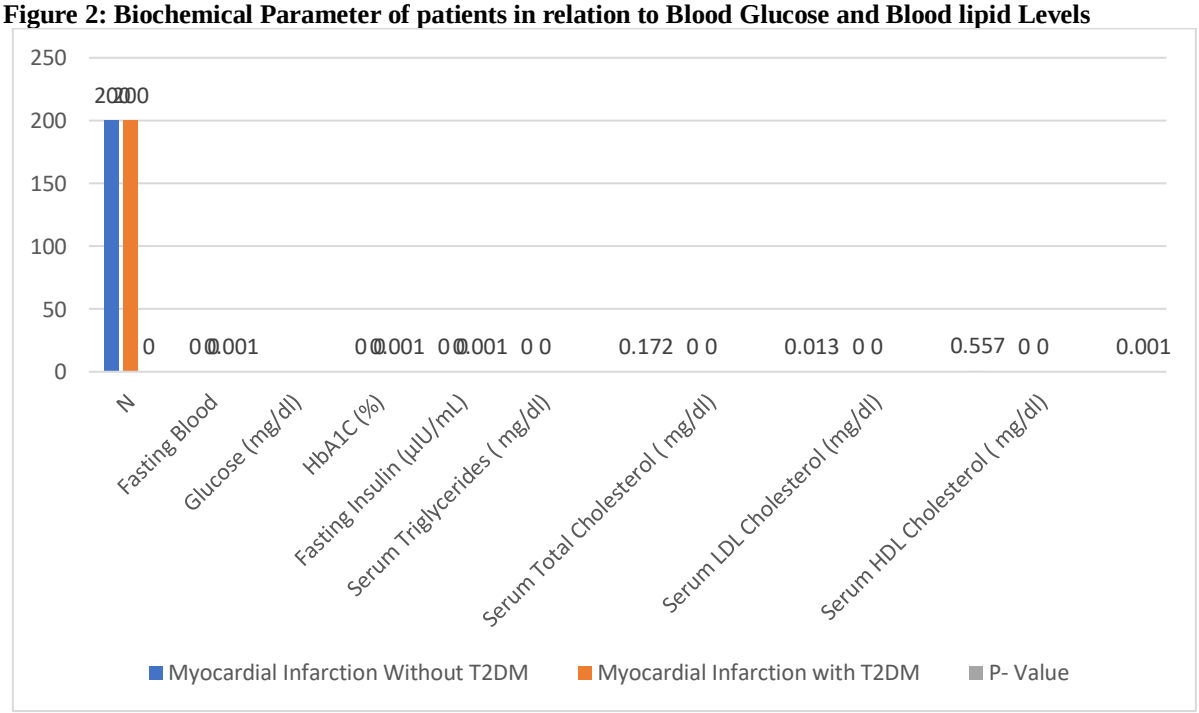


Table 2: Biochemical Parameter of patients in relation to Blood Glucose and Blood lipid Levels

	Myocardial Infarction Without T2DM	Myocardial Infarction with T2DM	P-Value
N	**200**	**200**	**-**
Fasting Blood Glucose (mg/dl)	80 ±4	132±16	0.001
HbA1C(%)	5±0.70	7±0.81	0.001
Fasting Insulin (µIU/mL)	11 ±2	19 ±4	0.001
Serum Triglycerides (mg/dl)	190±25	185±26	0.172
Serum Total Cholesterol(mg/dl)	204±22	196±24	0.013
Serum LDL Cholesterol (mg/dl)	147±30	144±37	0.557
Serum HDL Cholesterol(mg/dl)	24 ±5	27 ±7	0.001

Figure 2: Biochemical Parameter of patients in relation to Blood Glucose and Blood lipid Levels



DISCUSSION

Cardiovascular diseases, recognized as one of the leading causes of mortality globally, have prompted extensive research over the years. This emphasis on detailed investigation aims to comprehend the severity of damage and mortality caused by cardiovascular diseases, particularly focusing on finding ways to control their effects and prevent their occurrence. Diabetic dyslipidemia, often associated with insulin resistance, plays a pivotal role in this context.[10]Insulin resistance leads to alterations in very low-density lipoprotein (VLDL) metabolism, resulting in plasma lipid and lipoprotein abnormalities that increase the likelihood of atherosclerosis, ultimately contributing to coronary heart disease (CHD) in patients with diabetes mellitus (DMT). Various therapeutic approaches have been identified to improve dyslipidemia, including lifestyle interventions such as physical activity and weight loss, as well as the use of medications like fibrates, nicotinic acid, statins, and thiazolidinediones (TZDs). These treatments have demonstrated efficacy in reducing small dense low-density lipoprotein (LDL) particles, showing promise in slowing the progression of coronary artery disease.Historically, studies such as the one conducted by Biorck et al. in 1957 were among the first to report modifications in lipid levels after acute myocardial infarction (AMI). Subsequent research has shown significant alterations, including a 47% reduction in total cholesterol (TC), a 39% reduction in LDL cholesterol (LDL-C), an 11% reduction in high-density lipoprotein cholesterol (HDL-C), and a 50% increase in triglycerides (TG) in serum following AMI. These changes typically manifest within 24-48 hours of AMI, peak at 4-7 days, and gradually subside over several months.[11]The severity of the infarction, the extent of tissue necrosis, and their duration before the event can be assessed through the changes in lipid levels. However, it remains unclear whether therapeutic interventions, including percutaneous coronary interventions and thrombolytic treatment, can modify lipid levels. Protocols for managing acute coronary syndrome patients in hospitals recommend checking lipid levels within 24 hours and monitoring them regularly until a stable healthy state is achieved. Even a minimal change within the first 24 hours can provide valuable information for selecting lipid-lowering therapy. Initiating such therapy in the early days post-MI, even if lipid levels are decreasing periodically, is considered crucial for optimal management.

In our research, the primary focus was to scrutinize the intricate interplay between lipid levels and diabetes mellitus in distinct cohorts, specifically individuals with myocardial infarction (MI) but without diabetes mellitus type 2 (DMT2) and those presenting with both MI and DMT2.[12] The comprehensive examination of lipid profiles revealed intriguing findings.Triglycerides and low-density lipoprotein (LDL) levels, fundamental components of lipid metabolism, did not exhibit significant differences between the two groups. This suggests that, at the lipid level, there was no pronounced distinction between MI patients with and without DMT2. However, high-density lipoprotein (HDL), a key player in cholesterol transport, presented noteworthy differences. HDL levels were notably lower ($P<0.001$) in patients with MI but without DMT2, emphasizing the potential implications of reduced HDL in the context of atherosclerosis, especially within the arteries of the heart, ultimately contributing to the development of coronary artery diseases.The significance of lower HDL levels in the MI without DMT2 group lies in the understanding that HDL acts as a protective factor, facilitating the removal of cholesterol from vessels. Consequently, reduced HDL levels may heighten the risk of cholesterol deposition in arteries, exacerbating the potential for atherosclerotic complications.[13]

Moving beyond lipid parameters, our investigation extended to diabetic indices, particularly fasting blood sugar and insulin levels. The outcomes revealed statistically significant disparities ($P<0.001$) in these indices between MI patients with and without DMT2. Intriguingly, triglycerides, LDL, and total cholesterol did not show considerable differences between the two groups. On the contrary, HDL and glycosylated hemoglobin exhibited significant reductions ($P<0.001$) in individuals with myocardial infarction, particularly when compared to those with both myocardial infarction and diabetes mellitus type 2.[14,15]These nuanced findings underscore the complexity of the relationships among lipid metabolism, diabetes mellitus, and cardiovascular health. The observed distinctions in HDL levels and diabetic indices not only contribute to a deeper understanding of these interconnected factors but also pave the way for potential therapeutic interventions. Further exploration of these associations may uncover targeted strategies to mitigate the risk of coronary artery diseases in individuals with myocardial infarction, with and without diabetes mellitus type 2. [16,17]

CONCLUSION

In the subset of individuals experiencing myocardial infarction with diabetes mellitus type II, our analysis revealed positive correlations among key biochemical parameters. Specifically, there was a positive correlation observed between triglycerides and fasting blood sugar levels, suggesting a concurrent increase in both variables. Similarly, low-density lipoproteins (LDL) demonstrated a positive correlation with insulin levels, indicating a coordinated rise in these two parameters within the same group of patients.Contrastingly, in the subset of myocardial infarction patients without diabetes, our findings unveiled negative correlations between several pairs of biochemical markers. Notably, there was a negative correlation between triglycerides and glycosylated

hemoglobin (HbA1C), suggesting an inverse relationship between these variables. Additionally, low-density lipoproteins exhibited a negative correlation with HbA1C, implying that as HbA1C levels decrease, LDL levels tend to increase in this particular subset. Furthermore, a negative correlation was found between HbA1C and insulin levels, indicating an inverse relationship between glycemic control (HbA1C) and insulin levels in myocardial infarction patients without diabetes.These correlation patterns shed light on the intricate relationships between lipid metabolism, glycemic control, and insulin dynamics in distinct patient groups. Understanding these correlations is vital for unraveling the complex metabolic interactions that occur during myocardial infarction and can inform targeted interventions for specific subsets of patients based on their diabetic status.

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