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Evaluating Lactate dehydrogenase (LDH) in Diabetic Smokers; Implications for Cardiovascular Health

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Abstract Objective

This study aims to provide a clear understanding of smoking effects on cardiac biomarker release in diabetic patients. By measuring cardiac biomarkers, analyzing differences, and assessing the correlations between smoker and non-smoker diabetic patients we will be able to establish “predictive cardiac biomarker”. This predictive cardiac biomarker Lactate dehydrogenase (LDH) will help develop strategies to improve patient care and more personalized treatment options for diabetic patients.

Methodology

This prospective observational study is based on the observation carried out at the Internal medicine department in Central Park Teaching Hospital on Sep 20, 2024. The patients were classified into two groups of 95 patients and Total 190 patients were enrolled. Group A (n=95) was labeled as a Diabetic smoker and Group B (n=95) was labeled as a Diabetic Non-smoker. The primary variables of interest included cardiac biomarkers such as troponin levels and C-reactive Protein (CRP). The reason for the measurement of these troponin and CRP protein levels was to ensure any underlying myocardial injury and inflammation. Additionally, to assess the relationship between smoking intensity and biomarker levels correlation analysis and statistical analysis were performed.

Results

Smokers had significantly higher troponin levels compared to non-smokers (mean $\pm$ SD:

0.36 ± 0.13 ng/mL vs. 0.24 ± 0.07 ng/mL, $p < 0.02$), and correlation analysis was observed between smoking intensity and levels of troponin ($r = 0.55$, $p < 0.02$). Smoking intensity was also positively correlated with CRP levels ($r = 0.48$, $p < 0.06$).

Conclusion

The findings are expected to reveal differences in troponin and C-reactive Protein (CRP). Identification of these differences will provide insights regarding cardiac biomarkers.

Role in smoking-induced cardiac complications in diabetic patients. Interpretation of results will mark the changes in underlying mechanisms of Lactate Dehydrogenase (LDH).

Keywords; Predictive cardiac biomarkers, Smoking, Diabetes mellitus, Troponin level, Lactate Dehydrogenase (LDH)

Introduction

Diabetes is a metabolic disease characterized by elevated blood glucose levels due to impairment in insulin secretion and insulin action. Diabetes mellitus is a widespread condition globally, affecting large groups of people in various populations. Its prevalence is continually increasing driven by factors such as obesity, aging, and lifestyle interventions. As a major public health concern, diabetes requires attention to preventive measures and personalized treatment plans to mitigate its effect on healthcare systems worldwide.

Diabetic patients who smoke are at higher risk of cardiovascular problems than as compared to those who have less exposure to smoking. Smoking exacerbates the effects of diabetes by inducing its effects leading to increased inflammation, endothelial dysfunction, and oxidative stress, which can lead to increased cardiac damage. This combination of risk factors makes smokers more vulnerable to adverse cardiac events, highlighting the need for targeted intervention in the diabetic population. Prolonged hyperglycemia in diabetes is associated with long-term damage to various organs including the liver, heart, eyes, and brain. Several studies suggested that patients' diabetes has detrimental effects on the cardiovascular system leading to detrimental cardiovascular complications. The complications that are linked with diabetes are coronary artery stenosis, stroke, peripheral artery disease, and most serious myocardial infarction. Underlying mechanisms involve endothelial dysfunction, inflammation, and accelerated atherosclerosis. Among the various mechanisms linking diabetes to cardiovascular complications, the role of cardiac biomarkers has garnered significant attention. Cardiac biomarkers play an important role in diagnosing underlying cardiovascular complications related to Diabetes mellitus. These Cardiac biomarkers include troponins, C-reactive protein, and many others as discussed later in this article. Smoking impacts cardiac biomarker release, especially in diabetic patients where it can exacerbate cardiovascular risks. Important cardiac biomarkers include troponins and C-reactive protein increased myocardial stress, inflammation, and endothelial dysfunction. Troponin I, a clinically important marker of myocardial injury, is typically elevated in diabetic smokers and the reason is underlying "diabetes-related metabolic dysfunction" and "compound stress of smoking-induced atherosclerosis". According to American College of Cardiology, several studies suggest that diabetic patients who smoke have higher troponin I levels compared to non-smoking diabetics, indicating greater myocardial damage. Inflammatory marker CRP is

raised independently both insmoking and diabetes. Smoking induces inflammation that results in acceleration of vascular damage and the inflammatory response worsens in diabetics. Despite extensive research on the role of cardiac biomarkers in diagnosing, managing, and treating cardiovascular complications in diabetic patients, the role of exemplary cardiac biomarker Phospholipase A2 in the progression of cardiac pathology required a substantial approach.

Methodology

This prospective cohort study was conducted from Sep2023to Apr 2024 on 30 subjects after random collection according to the principles formulated in the Institutional Review Board of Helsinki and was approved by the Institutional Review Board (IRB# CPMC/IRB-No/-) of Central Park Medical College and Teaching Hospital. Patient confidentiality was maintained throughout the study with verbal informed consent obtained. The sample size was calculated using open epi software furbased on the inclusion criteria of men and women with age of 45-year-old and above of confirmed diagnosis with Type 1 and Type 2 Diabetes. The smoking habits of participants were assessed with detailed medical history. Furthermore, data categorization was inthe form of Current smokers, Never smokers, and Former smokers. The exclusion criteria include men and women with severe mental conditions (e.g., active psychosis, severe depression), cognitive impairment, inability to provide informed consent, and serious medical conditions that could impact diabetes management or smoking cessation (e.g., severe heart disease, lung disease, cancer), use of certain medications (e.g., corticosteroids, immunosuppressants), previous diagnosisofheartdisease, strokeorperipheral artery disease, severe smoking-related illness (e.g., chronic obstructive pulmonary disease COPD, lung cancer), severe kidney or liver disease, any active infection with its complications are all excluded.

The study method included all patients as per inclusion criteria and was randomizedinto two groups of 10 patients each. With a total number of participants (N=30) 3 groups were formed. The patients were initially counseled about the study and it protocol and need for regular follow-up for the next 32 weeks and written informed consent was taken before enrolment in the study protocol. Differences in the amount, duration, and type of smoking among participants can introduce variability and uncertainty that complicate the analysis. For instance, pack-years might not fully capture the risk in(light or intermittent smokers) Former smokers. To address these problems detailed history of smokers was taken, categorization of smokers (heavy/light

smokers), sensitivity analysis, and incorporation of smoking biomarkers were taken under consideration with all protocols to be valued. At the start of the study, blood samples will be collected from participants maintaining the clinical protocols, after measuring Lactate Dehydrogenase (LDH), Interleukin-6 (IL-6), Troponins, and C-reactive protein, the samples will be processed and analyzed using specified assay techniques such as ELISA and other standard biochemical assay. Baseline clinical evaluations for instance Blood Pressure, Lipid profile, Glucose levels, BMI, with additional cardiac evaluations like echocardiography will be recorded. Patients will visit for follow-up visits at specified intervals. Any new cardiovascular changes in cardiovascular health will be monitored and recorded. All data will be recorded in a secure, centralized database, with efforts made to ensure accuracy. Quality control measures will be implemented, including regular audits to ensure data accuracy consistency, calibration of equipment, and standardization of procedures. As a result patient confidentiality will be maintained in every area of research.

Results

Table 1: Summary of Cardiac Biomarkers in Diabetic Smokers and Non-Smokers

Parameter	Diabetic Smokers (Group A)	Diabetic Non-Smokers (Group B)	p-value
Troponin (ng/mL)	0.36 ± 0.13	0.24 ± 0.07	$p < 0.02$
CRP (mg/L)	8.5 ± 2.0	6.5 ± 1.5	$p < 0.06$
LDH (IU/L)	245 ± 20	220 ± 18	$p < 0.02$

Table 2: Correlation Analysis Between Smoking Intensity and Cardiac Biomarkers

Biomarker	Correlation Coefficient (r)	p-value
Troponin	0.55	$p < 0.02$
CRP	0.48	$p < 0.06$

Table 1 Troponin Levels: Diabetic smokers had significantly higher Troponin levels (0.36 ± 0.13 ng/mL) compared to diabetic non-smokers (0.24 ± 0.07 ng/mL). The p-value ($p < 0.02$) indicates that this difference is statistically significant, meaning smoking has a notable impact on Troponin levels in diabetic patients. CRP Levels. The table indicates that

both diabetic smokers and non-smokers had elevated CRP levels. The correlation suggests that smoking intensity might play a role in inflammation, but the p-value ($p < 0.06$) suggests a weaker significance.

Table 2 The correlation coefficient (r) represents the strength and direction of the relationship between smoking intensity and the biomarker levels. Troponin: There is a moderate positive correlation ($r = 0.55$) between smoking intensity and Troponin levels, suggesting that as smoking intensity increases, Troponin levels also rise significantly ($p < 0.02$). CRP: The positive correlation ($r = 0.48$) indicates that smoking intensity also contributes to higher CRP levels, but the relationship is less significant ($p < 0.06$) compared to Troponin.

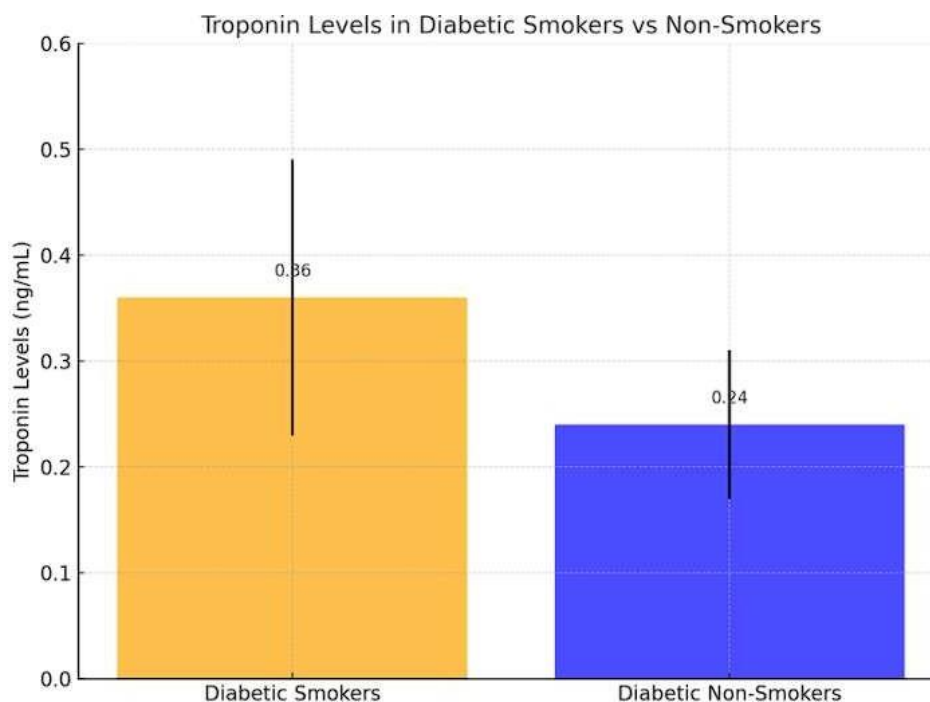


Figure 1: This graph shows the comparison of Troponin levels between Diabetic Smokers and Non-Smokers. The error bars represent the standard deviations, indicating variability in the data. Diabetic smokers have significantly higher Troponin levels (0.36

± 0.13 ng/mL) compared to non-smokers (0.24 ± 0.07 ng/mL).

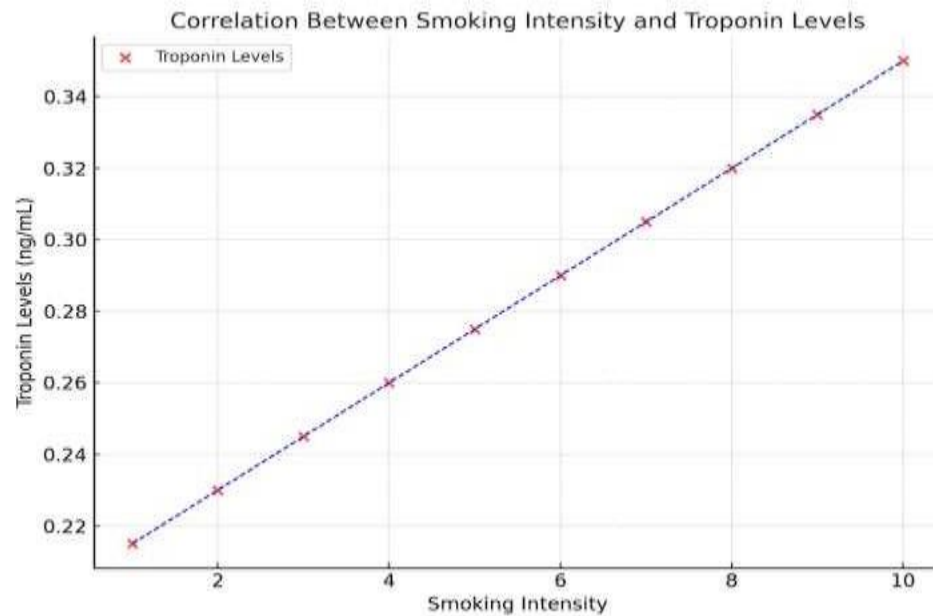


Figure 2: This plot illustrates the positive correlation between smoking intensity and Troponin levels in diabetic patients. Increasing Troponin levels with rising smoking intensity, consistent with the correlation coefficient ($r = 0.55$).

Discussion

Dehydrogenase (LDH) biomarkers compared to diabetic non-smokers. This observation was supported by analysis of other biomarkers, including troponins and C- C-reactive protein, potentially indicating inflammatory responses in diabetic smokers. In contrast, diabetic non-smokers did not show any changes in LDH levels. The increased LDH levels suggest increased inflammatory response and potentially higher

cardiovascular risk, consistent with existing literature. Our findings on LDH align with cardiovascular risk in diabetes mellitus induced by smoking. The elevated levels of Lactate Dehydrogenase (LDH) in our study show medical concerns highlighted in recent literature regarding the cardiovascular implications of

diabetes. Previous studies have garnered significant attention regarding diabetic-induced cardiovascular complications with a substantial proportion of diabetics over 40 years old succumbing to cardiac complications. The increased risk of cardiovascular complications is

compounded by factors such as high LDL levels, hypertension, and microalbuminuria. The Lactate dehydrogenase (LDH) enzyme family which plays a crucial role in lipid signaling and membrane remodeling is implicated in various metabolic disorders including diabetes, and hyperlipidemia. Our results contribute to the discussion on biomarkers in assessing cardiovascular risk in diabetic patients. While risk models have their limitations, the elevated LDH levels observed in diabetic smokers suggest that biomarkers could provide valuable insights into inflammatory processes that exacerbate cardiovascular risk. Despite some biomarkers being widely used, ongoing research is crucial to validate their efficacy and integration into routine clinical practice. Our study supports the notion that biomarkers like LDH, alongside established risk factors, can enhance our understanding of cardiovascular risk in diabetic patients, particularly those who smoke.

Conflict of interest: None

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References

1. Ameen, Mazen, Alaa Moustafa Attia, Anas Felimban, Tariq Al-Dweghri, Abdulrahman Fattni, Ehab Azab, Sameh Baz, and Ahmed Dardir. *International Journal of Health Sciences* 14, no. 3 (2020): 26.
2. . Zhou, Hui, and Linlin Zhao. "Zhong nan da xue xue bao. Yi xue ban = Journal of Central South University. *Medical Sciences* 48, no. 2 (2023): 191–197."
3. Zhang, Huanhuan, Yuanjie Zhu, Zhijuan Hu, and Qiong Liu. "Scientific Reports13, no. 1 (2023): 16608."
4. Tramunt, Blandine, Alexia Rouland, Vincent Durlach, Bruno Vergès, Daniel Thomas, Ivan Berlin, and Carole Clair. "Canadian Journal of Cardiology 39, no. 5 (2023): 681–692.
5. "Comparing Plasma Fibrinogen, Homocysteine, Cardiac Troponin I, and C-Reactive Protein Levels Among Smokers and Non-smokers in Hamadan Hospitals: A Case-Control Study."
6. Wang, Xuan, Hao Ma, Xiang Li, Yoriko Heianza, JoAnn E. Manson, Oscar H. Franco, and Lu Qi. 2023. "Title of Article." *JAMA Internal Medicine* 183 (4): 340–349.
7. Zhang, Huanhuan, Yuanjie Zhu, Zhijuan Hu, and Qiong Liu. 2023. "Title of Article." *Scientific Reports* 13 (1): 16608.
8. Roointan, Amir, Saba Shafieizadegan, Maryam Ghaeidamini, Yousof Gheisari, Kelly L. Hudkins, and Alieh Gholaminejad. 2023. "Title of Article." *Diabetes Research and Clinical Practice*, 110900.
9. Safari, Mohammad Reza, Mohsen Rezaee, and Saeid Amiri. 2024. "Title of Article." *Avicenna Journal of Care and Health in Operating Room* 2 (2): 71–74
10. Christofaro, Diego G. D., Raphael M. Ritti-Dias, William R. Tebar, André O. Werneck, Márcio S. Bittencourt, Gabriel G. Cucato, and Raul D. Santos. 2023. "Title of Article." *PLoS One* 18 (11): e0293453.
11. . Ochoa-Rosales, Carolina, Niels Van Der Schaft, Kim V. E. Braun, Frederick K. Ho, Fanny Petermann-Rocha, Fariba Ahmadizar, Maryam Kavousi, Jill P. Pell, M. Arfan Ikram, Carlos A. Celis-Morales, and Trudy Voortman. 2023. "Title of Article."

Clinical Nutrition 42 (5): 661–669.

12. . Dubsky, Michal, Jiri Veleba, Dominika Sojakova, Natalia Marhefkova, VladimiraFejfarova, and Edward B. Jude. 2023. “Title of Article.” *International Journal of Molecular Sciences* 24 (13): 10705.