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Original Research Article

Association of interleukin-1β levels in type 2 diabetes mellitus patients

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a complex, non-communicable illness mainly marked by two main features: insulin resistance and chronic inflammation. Interleukin-1β (IL-1β) has been implicated in metabolic processes including insulin secretion and b-cell apoptosis. Increased levels of pro-inflammatory markers such as IL-1β, tumor necrosis factor-α and adipokines has been reported in T2DM patients. Recent studies have demonstrated a positive correlation between Interleukins in T2DM patients. The objective of the study was to find association of IL-1β in T2DM patients.
Methods: We conducted a cross-sectional study among T2DM patients visiting the OPD of Pulmonary Medicine. T2DM patients aged between 40 to 65 years of either sex were recruited. Biochemical investigations were performed in blood sample.
Results: We recruited 340 T2DM patients (162 males and 178 females) for the present study. Biochemical analysis viz. Plasma glucose and HbA1c reported 78 subjects with uncontrolled diabetes and 262 subjects with controlled diabetes. IL-1β levels were found to be significantly increased ($p<0.001$, $t=21.76$) in uncontrolled diabetics (55.23±9.11) in comparison with controlled diabetics (25.39±11.04).
Conclusions: Results suggests a highly significant and positive ($p<0.001$) association of IL-1β with glucose levels in T2DM patients.

Keywords: Type 2 diabetes mellitus, Interleukin-1β, HbA1c, Inflammation

INTRODUCTION

The global population of diabetes patients is growing rapidly. Since the International Diabetes Federation (IDF) recorded 151 million cases worldwide in 2000, that number has nearly quadrupled, reaching 589 million adults in 2024. The increase in cases has been alarmingly fast: the global figure nearly doubled to 285 million by 2022. If this rate continues, an estimated 500 million people could be living with diabetes within the next decade. The long-term complications of diabetes severely diminish patients' quality of life, placing a substantial economic burden on both families and individuals.[1]

Type 2 diabetes mellitus (T2DM), which constitutes over 90% of all diabetes cases, arises from a complex interplay of various pathogenic factors.[2] Studies indicate that inflammation is a potential pathogenic mechanism underlying T2DM and various diseases associated with obesity.[3] Inflammation is an important biological immune defence mechanism.[4] In human, inflammation plays a dual role in various disease. Early inflammatory responses may promote tissue repair. However, severe inflammation can lead to tissue damage.[5] Chronic inflammation is recognized as a central mechanism in T2DM, impairing beta-cell function and contributing significantly to insulin resistance.[6] The inflammatory cytokines such as tumour necrosis factor-alpha (TNFα), and interleukins (ILs) can

promote the occurrence and development of T2DM and vascular atherosclerosis.[7]

A growing body of research confirms that persistent systemic or localized inflammation is a significant factor in the progression of many chronic non-infectious diseases, including diabetes and its serious complications such as diabetic kidney disease (DKD) and atherosclerosis.[8,9] Interleukin-1 beta (IL-1β) plays a central role in the pathogenesis of a wide range of pulmonary inflammatory disorders.[10] As an established mediator of beta-cell dysfunction and programmed cell death (apoptosis), the effects of IL-1β are potentiated by TNF-α and Interferon gamma (IFN-γ).[11]

Despite analysis using both random and fixed effects models, a meta-analytic review failed to show a statistically significant variation in IL-1β levels between T2DM patients and healthy subjects.[12] Both the severity of bronchial obstruction and the general progression of COPD have a direct impact on the body's inflammatory markers, including IL-1β, IL-4, IL-8, TNF-α, and IFN-γ.[13] This study aims to investigate the association of IL-1β in T2DM patients.

METHODS

This prospective, cross-sectional study was conducted at Era's Lucknow Medical College and Hospital, Lucknow. The study was carried out in the Department of Physiology in collaboration with the Department of Biochemistry and Medicine. Ethical approval for the study was granted by the Institutional Ethics Committee prior to commencement vide letter no NIMSUR/IEC/2023/506 dated 06.03.2023. Study was conducted during February 2023 to January 2025.

Subject recruitment

T2DM patients, attending the OPD of Medicine, Era's Lucknow Medical College, Lucknow, fulfilling the criteria were recruited. Informed consent was obtained from all study participants.

Inclusion criteria

Patients of T2DM aged between 40 to 65 years of either sex, T2DM patients for more than six months duration and patients willing to give informed written consent were included.

Exclusion criteria

Subjects with current smoking habit or a smoking history, pregnant women and subject having any other endocrine disorders e.g. hypo and hyperthyroidism, Cushing's syndrome, Grave's disease, and Addison's disease were excluded.

Study groups

T2DM patients were divided into controlled and uncontrolled as per American Diabetes Association criteria (2013).[14] Patients with HbA1C ≤7% were considered controlled and with HbA1C >7% were considered uncontrolled diabetic.

Cytokine analysis

3 ml blood was drawn from all the study participants for cytokine analysis. IL-1β was quantified using enzyme-linked immunosorbent assay (ELISA) kit. The assay procedure was performed according to the instructions provided in the technical bulletin supplied with the kit (R&D Systems, Minneapolis, MN, USA).

Statistical analysis

After the collection of data, statistical analysis was performed using statistical package for the social sciences (SPSS) version 18. Quantitative parametric data are expressed as mean±SD. Qualitative data are expressed as number and percent of total. Comparative analysis was done using one-way ANOVA. Correlations were done with Pearson's correlation coefficient. P value less than 0.05 was considered significant.

RESULTS

We conducted the study by recruiting 340 T2DM patients. Of the total T2DM patients, males were 162 (47.65%) and females were 178 (52.35%), indicating female predominance. Patients were categorised in uncontrolled and controlled diabetics depending on the HbA1C value and fasting blood glucose levels. A total of 78 patients had uncontrolled diabetes, compared to 262 patients with controlled diabetes.

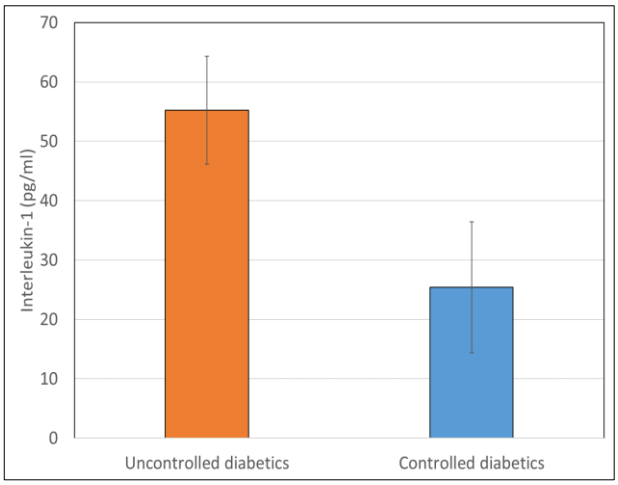

Figure 1: Comparison of IL-1β levels in T2DM patients.

Among 78 uncontrolled diabetics, 46.2% were male and 53.8% were female, while among 262 controlled diabetics, 48.1% were male and 51.9% were female. A higher proportion of females was observed in both controlled and uncontrolled T2DM groups compared to males i.e., uncontrolled diabetics (53.8%) as well controlled diabetics (51.9%), this increase was observed statistically non-significant (χ^2=0.132, p<0.716) (Table 1).

Fasting blood glucose levels were found highly significantly (p<0.001) increased in uncontrolled diabetics (241.10±49.79) as compared to controlled diabetics (93.17±8.40), similarly HbA1c values were also found highly significantly (p<0.001) increased in uncontrolled diabetics (9.21±1.14) as compared to controlled diabetics (5.72±0.60). IL-1β levels were found significantly increased (p<0.001, t=21.76) in uncontrolled diabetics (55.23±9.11) as compared to controlled diabetics (25.39±11.04) (Figure 1 and Table 2).

A multivariate model was constructed to assess the independent predictors of IL-1β levels. The analysis revealed a significant and positive association between IL-1β levels and glycemic control status (r^2=0.825). In multivariate regression model, we projected IL-1β level as an independent variable and glycemic control status was considered as independent variable. Glycemic control status was found to have a significant association with IL-1β levels (r^2=0.825) (Table 3).

Table 1: Characteristics of study participants.

S. no.	Characteristic/parameter	Uncontrolled diabetes (n=78)	Controlled diabetes (n=262)	Statistical significance
1	Age (years) Mean±SD (range)	56.24±6.87 (40-65)	51.59±7.15 (40-65)	t=5.280; p<0.001
2	Gender			
	Male	36 (46.2%)	126 (48.1%)	χ^2=0.132; p=0.716
	Female	42 (53.8%)	136 (51.9%)	

Table 2: Comparison of biochemical parameters between study groups.

S. no.	Parameter	Uncontrolled diabetes (n=78)	Controlled diabetes (n=262)	Statistical significance
1	HbA1c (%) (mean±SD)	9.21±1.14	5.72±0.60	t=35.75; p<0.001
2	Fasting glucose (mg/dl) (mean±SD)	241.10±49.79	93.17±8.40	t=46.09; p<0.001
3	IL-1β (pg/ml) (mean±SD)	55.23±9.11	25.39±11.04	t=21.760; p<0.001

Table 3: Association of Interleukin-1β levels with different study variables.

S. no.	Variable	β±SD	t	P value
1	Age (years)	0.043±0.065	0.661	0.509
2	Gender (1=male, 2=female)	-2.433±0.781	3.113	0.002
3	HbA_1C (%)	0.950±0.509	1.866	0.063
4	Fasting blood glucose (mg/dl)	0.079±0.016	5.063	<0.001
5	Group (1=uncontrolled; 2=controlled)	-11.11±2.85	3.896	<0.001
6	Constant	-113.10±27.54	4.107	<0.001

r^2=0.825

DISCUSSION

Although T2DM impacts over 350 million people globally and incurs a massive socioeconomic cost, its exact pathogenesis remains incompletely understood. The disorder's core physiological features involve insulin resistance (a reduced response to insulin) and β-cell failure.[15] Furthermore, The COVID-19 pandemic presents an elevated risk to individuals with diabetes because the viral infection directly impacts the respiratory system.[16]

In this study, we assessed IL-1β levels in patients with T2DM (controlled and uncontrolled diabetics). Biochemical investigations showed significantly high fasting blood glucose levels and HbA1c values in uncontrolled diabetics compared to controlled diabetics. These results indicate impaired glucose levels and HbA1c values in uncontrolled diabetics.

The role of inflammation in the development (pathogenesis) of T2DM has become increasingly clear in recent years. In addition to causing β-cell damage, IL-1β is also released by β-cells in response to glucose stimulation. Additionally, IL-1β promotes its own synthesis within β-cells and attracts macrophages, which serve as an additional source of IL-1β and several other inflammatory mediators.[17] Increased IL-1β production have been documented in stable COPD, and these levels

are further augmented during exacerbations of the disease. Elevated levels of IL-1β have been detected in the bronchoalveolar lavage fluid and tracheal biopsy samples of both asymptomatic and symptomatic individuals suffering from asthma.[18]

Haamid et al observed a significant link between elevated CRP levels and conditions such as insulin resistance, obesity, and dyslipidemia. They also noted that high TNF-α levels were strongly associated with female gender, poor glycemic control, and a strong familial link to diabetes.[19]

In another study conducted by a biochemistry research team at Iran revealed that higher levels of the cytokines IL-6 and IL-1β were significantly associated with worse metabolic health, specifically showing positive correlations with HbA1c, FBS, insulin, and insulin resistance. However, a significant negative correlation was found, linking lower levels of vitamin D to higher levels of IL-6 and IL-1β.[20]

Studies report that serum levels of TNF-α and IL-1β are significantly elevated in patients with diabetic retinopathy, with the increase becoming progressively greater as the disease advances.[21] Smokers with COPD have also been reported to exhibit greater susceptibility to cigarette smoke. This high susceptibility increases permeability and triggers the release of pro-inflammatory mediators, including IL-1β and sICAM-1.[22]

Although the present study has a limitation in its relatively small sample size, the results are consistent with a number of research data discussed earlier. The results of this study add to our understanding of the relationship involving IL-1β levels in individuals with T2DM. The findings of this study provide further evidence that IL-1β is a key cytokine in the progression of T2DM, shedding important light on its damaging effects in the body.

Limitations

The study was conducted with a small sample size; therefore, a large group study can provide deeper insight and prospective analysis to find mechanism of elevated IL-1β in diabetic population.

CONCLUSION

The findings of this study indicate that the simultaneous measurement of IL-1β in T2DM patients may represent a potential novel. Measuring serum IL-1β levels could offer valuable information as a marker for neutrophilic inflammation and help predict the occurrence of acute exacerbations in patients with T2DM.

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REFERENCES

1. Chinese Elderly Type 2 Diabetes Prevention and Treatment of Clinical Guidelines Writing Group; Geriatric Endocrinology and Metabolism Branch of Chinese Geriatric Society; Geriatric Endocrinology and Metabolism Branch of Chinese Geriatric Health Care Society; Geriatric Professional Committee of Beijing Medical Award Foundation; National Clinical Medical Research Center for Geriatric Diseases (PLA General Hospital). Clinical guidelines for prevention and treatment of type 2 diabetes mellitus in the elderly in China (2022 edition). Zhonghua Nei Ke Za Zhi. 2022;61(1):12-50.
2. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88-98.
3. Kohlgruber A, Lynch L. Adipose tissue inflammation in the pathogenesis of type 2 diabetes. Curr Diab Rep. 2015;15(11):92.
4. Cronkite DA, Strutt TM. The Regulation of Inflammation by Innate and Adaptive Lymphocytes. J Immunol Res. 2018;1-14.
5. Rathinam VAK, Chan FKM. Inflammasome, Inflammation, and Tissue Homeostasis. Trends Mol. Med. 2018;24:304-18.
6. Eguchi K, Manabe I, Oishi-Tanaka Y, Ohsugi M, Kono N, Ogata F, et al. Saturated fatty acid and TLR signaling link β cell dysfunction and islet inflammation. Cell Metabolism. 2012;15:518-33.
7. Palomo J, Dietrich D, Martin P, Palmer G, Gabay C. The interleukin (IL)-1 cytokine family-Balance between agonists and antagonists in inflammatory diseases. Cytokine. 2015;76(1):25-37.
8. Tang SCW, Yiu WH. Innate Immunity in Diabetic Kidney Disease. Nat Rev Nephrol. 2020;16:206-22.
9. Rohm TV, Meier DT, Olefsky JM, Donath MY. Inflammation in Obesity, Diabetes, and Related Disorders. Immunity. 2022;55:31-55.
10. Suwara MI, Green NJ, Borthwick LA, Mann J, Mayer-Barber KD, Barron L, et al. IL-1α released from damaged epithelial cells is sufficient and essential to trigger inflammatory responses in human lung fibroblasts. Mucosal Immunol. 2014;7:684-93.
11. Böni-Schnetzler M, Thorne J, Parnaud G, Marselli L, Ehses JA, Kerr-Conte J, et al. Increased interleukin (IL)-1beta messenger ribonucleic acid expression in beta -cells of individuals with type 2 diabetes and regulation of IL-1beta in human islets by glucose and autostimulation. J Clin Endocrinol Metab. 2008;93(10):4065-74.

12. Alfadul H, Sabico S and Al-Daghri NM. The role of interleukin-1β in type 2 diabetes mellitus: A systematic review and meta-analysis. Front Endocrinol. 2022;13:901616.
13. Kubysheva NI, Postnikova LB, Soodaeva SK, Novikov DV, Eliseeva TI, Novikov VV, et al. Comparative Study of the Levels of IL-1β, IL-4, IL-8, TNFα, and IFNγ in Stable Course and Exacerbation of Chronic Obstructive Pulmonary Disease of Varying Severity. Bull Exp Biol Med. 2022;173(6):745-8.
14. American Diabetes Association Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013;33:S62-9.
15. Tabák AG, Jokela M, Akbaraly TN, Brunner EJ, Kivimäki M, Witte DR. Trajectories of glycaemia, insulin sensitivity, and insulin secretion before diagnosis of type 2 diabetes: an analysis from the Whitehall II study. Lancet. 2009;373(9682):2215-21.
16. Zhang W, Li C, Xu Y, He B, Hu M, Cao G, et al. Hyperglycemia and correlated high levels of inflammation have a positive relationship with the severity of coronavirus disease 2019. Mediat Inflamm. 2021;18:8812304.
17. Rissanen A, Howard CP, Botha J, Thuren T. Effect of anti-IL-1b antibody (canakinumab) on insulin secretion rates in impaired glucose tolerance or type 2 diabetes: results of a randomized, placebo controlled trial. Diabetes Obesity Metab. 2012;14:1088-96.
18. Sousa AR, Lane SJ, Nakhosteen JA, Lee TH, Poston RN. Expression of interleukin-1b and interleukin-1 receptor antagonist (IL-1ra) on asthmatic bronchial epithelium. Am J Respir Crit Care Med. 1996;154:1061-6.
19. Haamid B, Sabhiya M, Mosin SK, Mohammad HB, Rabia H, Roohi A et al. Inter-relationship of Pro- and Anti- inflammatory Biomarkers with the development of Type 2 Diabetes Mellitus. Heliyon. 2022;8(11):e11329.
20. Vahid P, Sarmad M, Mohammad SY. Investigation of serum level relationship anti-glutamic acid decarboxylase antibody and inflammatory cytokines (IL-1β, IL-6) with vitamins D in type 2 diabetes. J Diabetes Metab Disord. 2022;13:21(1):181-7.
21. Jin Q, Ying H. Expression of TNF- α and IL-1β in Peripheral Blood of Patients with T2DM Retinopathy. Comput Math Methods Med. 2022;8:9073372.
22. Rusznak C, Mills PR, Devalia JL, Sapsford RJ, Davies RJ, Lozewicz S. Effect of cigarette smoke on the permeability and IL-1beta and sICAM-1 release from cultured human bronchial epithelial cells of never-smokers, smokers, and patients with chronic obstructive pulmonary disease. Am J Respir Cell Mol Biol. 2000;23(4):530-6.

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