Letter to the Editor

Serum TGF-β and IL-1β Levels in Patients with Type 1 Diabetes Mellitus in Babylon Province: Investigating Associations with Disease Risk

Dear Editor,

Diabetes mellitus (DM) is a persistent metabolic disorder that has attracted growing attention as a worldwide health issue in recent times. The pathophysiology of type 1 DM (T1DM) entails an intricate interplay of genetic, autoimmune, and environmental elements that culminate in the obliteration of insulin-producing beta cells in the pancreas. This process ultimately leads to a complete deficiency of insulin, crucial for the regulation of blood glucose levels.[1]

T1DM is affected by a combination of factors, including genetic predisposition, a family history of the condition, specific human leukocyte antigen genes, and the presence of other autoimmune disorders.[2]

Transforming growth factor-beta (TGF-β) is a family of multifunctional cytokines that play essential roles in regulating cell growth, differentiation, development, and immune responses in various tissues and organs. It is intricately linked to diabetes. It impacts various aspects of the disease, including pancreatic beta cell function and survival, the development of fibrosis within the pancreatic islets, inflammation, insulin resistance, extracellular matrix remodeling, and the progression of diabetes-related complications. TGF-β's role differs in T1DM and type 2 DM (T2DM), and its influence can be both detrimental, contributing to tissue fibrosis and inflammation, and potentially beneficial, offering opportunities for therapeutic interventions. Understanding the complex interplay of TGF-β in diabetes is essential for uncovering potential treatment strategies and improving the management of the disease and its complications.[3]

Interleukin-1 beta (IL-1β) is a pro-inflammatory cytokine crucial in the body's immune response and inflammatory processes. It contributes to fever, immune cells activation, and other inflammatory cytokines. While IL-1β is crucial for combating infections and initiating the body's defense mechanisms, excessive or chronic production of IL-1β can lead to chronic inflammation and has been implicated in the pathogenesis of various inflammatory and autoimmune diseases.[4]

IL-1β plays a multifaceted role in diabetes. In T1DM, it is implicated in the autoimmune destruction of insulin-producing beta cells within the pancreas. In T2DM, particularly in the context of obesity, IL-1β is linked to chronic inflammation and insulin resistance, contributing to elevated blood glucose levels.[5]

The study was a case–control study for patients with T1DM. The period extended from February 2023 until September 2023. This work was done at the University of Babylon and Marjan Medical City in Hilla, Iraq. Eighty patients diagnosed with T1DM were included in this study, along with 80 participants forming a seemingly healthy control group.

Inclusion criteria included the diagnosis of T1DM in all patients was established according to the criteria outlined by the American Diabetes Association. Exclusion Criteria included patients with all types of cancer, patients with renal disease, and patients with liver disease.

Determining the levels of serum TGF-β in the patient and control groups was done by a sandwich enzyme-linked immunosorbent assay (ELISA) for *in vitro* quantitative measurement by Elabscience ELISA Kits Company (Wuhan, Hubei, China).

Determining the levels of serum IL-1β in the patient and control groups was done by a sandwich ELISA for *in vitro* quantitative measurement by the Elabscience ELISA Kits Company. Determining the levels of serum tumor necrosis factor alpha (TNF-α) in the patient and control group was done by a sandwich ELISA for *in vitro* quantitative measurement by the Elabscience ELISA Kits Company.

Information's were examined by Statistical Package for the Social Sciences version (SPSS Inc., Chicago, IL, USA), and determined as a mean and standard deviation (SD), continued variables were analyzed through using (independent-sample *t* test) which has been utilized to detected the significant difference between the groups, whereas *P* value <0.05 was counted as significant and <0.001 counted as highly significant. Patients provided verbal consent before the sample was taken. The study protocol and the subject information were reviewed and approved by a local Ethics Committee at the College of Medicine, University of Babylon.

The distribution of mean ± SD of TGF-β was statistically significant decrease in T1DM groups from control group with a *P* value of <0.001 that the mean ± SD of TGF-β concentration in T1DM group was 13.16 ± 2.66 ng/mL, whereas for controls group was 21.42 ± 4.64 ng/mL as shown below in Table 1.

The distribution of mean ± SD of IL-1β showed a statistically significant increase in T1DM versus the control group with *P* value of <0.001 that the mean ± SD of IL-1β in T1DM group was 61.35 ± 12.43 ng/mL, whereas for the control group was 32.87 ± 8.92 ng/mL as shown in Table 2.

The distribution of mean ± SD of TNF-α showed a statistically significant increase in T1DM versus the control group with *P* value of <0.001 that the mean ± SD of TNF-α in T1DM group was 214.77 ± 23.65 pg/mL, whereas for controls group was 126.23 ± 14.87 pg/mL as shown in Table 3.

Additionally, a recent investigation by Cano-Cano *et al*.[6] suggests that the modulation of IL-1β levels during various stages of T1DM could serve as an indicator of disease progression and effective management over time.[6]

Table 1: TGF-β levels between patients and control groups

Parameter	Subjects	No.	Mean	Standard deviation (SD)	*P* value
TGF-β (ng/mL)	T1DM	80	13.16	±2.66	(>0.001)*
	Control	80	21.42	±4.64	(>0.001)**

1TGF-β: transforming growth factor-beta, T1DM: type 1 diabetes mellitus.

*Significant difference ($P < 0.001$).

**Highly significant ($P < 0.001$)

Table 2: IL-1β levels between patients and control groups

Parameter	Subjects	No.	Mean	SD	*P* value
IL-1β (ng/mL)	T1DM	80	61.35	±12.43	(<0.001)*
	Control	80	32.87	±8.92	(<0.001)**

IL-1β, interleukin-1 beta, T1DM: type 1 diabetes mellitus, SD: standard deviation.

*Significant difference ($P < 0.001$).

**Highly significant ($P < 0.001$)

Table 3: TNF-α levels between patients and control groups

Parameter	Subjects	No.	Mean	SD	*P* value
TNF-α (pg/mL)	T1DM	80	214.77	±23.65	(<0.001)*
	Control	80	126.23	±14.87	(<0.001)**

TNF-α, tumor necrosis factor alpha, T1DM: type 1 diabetes mellitus, SD: standard deviation.

*Significant difference ($P < 0.001$).

**Highly significant ($P < 0.001$)

Another study conducted by Dogan *et al*.[7] Revealed elevated levels of serum IL-1β and TNF-α, along with decreased IL-2 and IL-6 levels in newly diagnosed insulin-dependent diabetes mellitus (IDDM) patients compared to those with longer-standing cases. This pattern supports the activation of a systemic inflammatory process during the early phases of IDDM, potentially indicating ongoing β-cell destruction.[7]

Tsalamandris *et al*.[8] concluded that IL-1beta is among the three cytokines implicated in the inflammation of pancreatic beta cells in T1DM. The synergic action of interferon gamma and the innate inflammatory cytokines TNF-α and IL-1β plays a role in this process.[8]

It can be concluded from this study that patients with T1DM in Babylon Province tend to have lower TGF-β levels and higher IL-1β levels compared with the control group. These differences are statistically significant and may indicate immune system dysregulation and inflammation in individuals with T1DM, as both TGF-β and IL-1β play roles in immune regulation and inflammation.

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Conflicts of interest
There are no conflicts of interest.

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