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# Estimation level of IL-10 and TNF-α in Iraq T1DM patient.

**Sabreen Ali Mezil***

Department of medical Instrumentation engineering, Al-Hadi University Collage, Baghdad, Iraq.

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## Editorial Note

Tumor Necrosis Factor (TNF-α) also known as differentiation factor, is a key component in inflammatory and immune responses. TNF-α is a pleiotropic proinflammatory cytokine produced in response to infection, inflammation, and environmental stressors by activated macrophages, and lymphocytes. TNF-α has been shown to play a part in the autoimmune cascade that leads to beta cell death in type 1 diabetes, and repeated antibody-mediated TNF-α action inhibition has been shown to protect non-obese mice from beta cell death. We tried to demonstrate a correlation between pro-inflammatory and anti-inflammatory cytokines including IL-10 and TNF-α, which are both involved in the pathogenesis of T1D, in children and adolescents under the age of 15 with varying diabetes durations in this study.

T1D is an autoimmune disease that causes chronic inflammation of the Langerhans islets in a pancreatic, which are responsible for insulin secretion. 50 T1DM patients and 40 healthy as control included in this study, Enzyme-linked Immune-Sorbent assays were used to test IL-10 and TNF- levels in serum (ELISA). T1D patients had a significant rise in serum TNF- and IL-10 levels as compared to non-diabetic subjects. The AUC for IL-10 (0.870, p-value 0.0379) predicted T1DM with 72% sensitivity and 100% specificity and TNF-α (0.875, p-value 0.0348) with 76.0 % sensitivity and 85.0 % specificity, inflammatory cytokines may be used as possible markers for the progression of T1DM. The gradual and insidious death of β-cells is a symptom of the autoimmune disease like autoimmune diabetesT1DM.The prevalence of type 1 diabetes has grown by 2% to 5%.Immune cells (T helper 1 cells and macrophages), activation and infiltration of islet of Langerhans resulting in the death of pancreatic cells is thought to be mediated by cytokines released during response also auto-antibodies specific for beta cells may contribute to cell destruction leading to extreme hyperglycemia. As a result, three major factors come into play: genetic, environmental, and immunological. Self-targeting immune cascades can be induced in children by environmental stimulus factors and slurs. Insulitis and islet autoantibodies' degradation of beta cells was mediated by cytokines and chemokines, which are involved in immune cell recruitment, modulation, and intercellular signalling.

SAS was used to do statistical analysis of the results. To decide if there were substantial variations between means, an independent t test was used. Statistical significance is defined as a P value of less than 0.05. The validity of markers as disease indicators was determined using the receiver operation characteristic curve. Type 1 diabetes is mediated by immune cells (T helper 1 cells and macrophages), activation and infiltration of islet of Langerhans resulting in the death of pancreatic cells is thought to be mediated by cytokines released during response also auto-antibodies specific for beta cells may contribute to cell destruction leading to extreme hyperglycemia. Cardiovascular disease increases the risk of mortality in diabetic patients. The present study found that dyslipidemia was slightly higher in diabetic patients than in the non-diabetic control community, which is consistent with other worker.

Patients with T1DM had slightly higher levels of IL-10 and TNF- than non-diabetic , according to our results. These findings suggest that elevated levels of both pro-inflammatory and anti-inflammatory cytokines are linked to the development of T1D. This indicates that the amount of inflammatory markers in T1DM patients is age-dependently controlled, indicating that the inflammatory immune response system is activated.

Future research into the immunobiology of T cells in general, as well as the gene expression of their cytokines and the pathophysiology of T1D in relation to Tfh cells, could help in better T1D management.

***Correspondence to:**

Sabreen Ali Mezil

Department of medical Instrumentation engineering,

Al-Hadi University Collage

Baghdad

Iraq