

Immuno-inflammatory markers: Adenosine deaminase and IL-6 in pathogenesis of type 2 diabetes mellitus

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Diabetes mellitus (DM) is one of the most common non communicable disease that causes morbidity and mortality worldwide. Evidence implicates immunological and inflammation process as a potential pathway in the pathogenesis of type 2 diabetes. The objective of the present study is to assess the levels of adenosine deaminase (ADA) and interleukin-6 (IL-6) markers in pathogenesis of diabetes in South Indian population.

Adenosine is an endogenous anti-inflammatory metabolite that is increased in response to inflammation. The pro inflammatory cytokines IL-6 positively regulate CD-26 ecto-ADA complex thereby increasing the expression of adenosine deaminase activity, which will convert the adenosine to inosine.

Several epidemiological studies have recently demonstrated increased serum levels of inflammatory markers, such as (IL-6), being associated with increased risk of T2DM, suggesting that genetic factors involving cytokines playing an important role in the development of T2DM.

A total of 150 diabetes mellitus cases were recruited from Gandhi Hospital, Hyderabad and 150 age and gender matched healthy controls without any family histories of any disease were collected. Estimation of ADA was based on the method reported by Giusti and Galanti (1984). IL-6 was carried out by Sandwich ELISA method. The mean \pm SD levels of ADA and IL-6 in the diabetic patients were significantly high at $p < 0.01$ compared to healthy controls.

These findings may have important implications for the management of type 2 diabetes and its complications. Pharmacological agents with anti-inflammatory properties may also have a role in type 2 diabetes prevention and treatment.

Biography

M.Saraswati, with 15 yrs teaching experience, published papers in reputed journals, book in reputed academic publishing and an editorial board member of reputed. Recipient of best oral award for paper presentation, certificate of felicitation for speaker.

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