

ELEVATED SERUM IGA AT ONSET OF TYPE 1 DIABETES IN PEDIATRIC PATIENTS

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Keywords:

Background: IgA is the most produced antibody in the body and is abundantly present in the serum. There are two major forms of IgA, monomeric serum IgA and dimeric mucosal (secretory) IgA. Increasing evidence suggests that IgA is not only involved in gut homeostasis, but also plays an important role in the regulation of immune responses. Elevated serum IgA has been sporadically reported in autoimmune diseases including type 1 diabetes (T1D), although its role in T1D is not understood. Here, we analyzed serum IgA and clinical parameters in children with new onset T1D.

Materials/Methods: We analyzed 612 children 9.7 [4.2] (mean [SD]) years old, (59% Non-Hispanic White, 20% Hispanic, 16% Black, 5% Other) with new onset T1D diagnosed between 1/2008-2/2012. Serum IgA values above age-adjusted normal ranges were defined as elevated serum IgA. Demographic and clinical variables were analyzed. A significance level of 0.05 was used.

Results: At the onset of T1D, elevated serum IgA was present in 21% (128/612) of the children [$p<0.001$]. By multivariable analysis, compared with children with normal IgA, those with elevated serum IgA were more likely to be of Hispanic ethnicity, had a higher hemoglobin A1c (A1c) and insulin autoantibody (IAA) level at diagnosis [$p<0.05$]. They also had lower odds of having GAD antibody positive. [$p<0.05$]. After adjusting for other variables using the multivariable logistic regression model, elevated serum IgA was not significantly associated with age, glucose level or presence of DKA.

Conclusions: On analysis, 21% of children had elevated serum IgA at onset of T1D. After adjusting for other variables, Hispanic ethnicity, elevated A1C, GAD and insulin autoantibody levels were found to be independently associated with elevated IgA level at onset which suggests possibly more severe insulin deficiency and islet autoimmunity. Further studies are warranted to investigate the IgA response and whether they persist over time. Given that current tools to prevent T1D are limited, improved understanding of the natural history of the disease may lead to new targeted strategies to preserve or improve beta-cell function in individuals with clinical or pre-clinical T1D. The authors are currently working on a prospective case control study to see for an association between elevated serum IgA and gut dysbiosis at onset of T1D in children.

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