Prevalence of Islet Autoantibodies in Type 1 Diabetes

Sir,

The recent article by Sanyal *et al.* made for an interesting read.[1] In a well-conducted study, the authors documented a high prevalence of islet autoantibodies in fifty patients with Type 1 diabetes (T1D). The main pancreatic autoantibody, i.e., glutamic acid decarboxylase (GAD65) antibody and islet antigen-2 antibodies were detected in 78% and 30% patients, respectively. The prevalence is the highest ever reported in Indian patients with T1D. The reported prevalence in the previous Indian studies has ranged between 26% and 61%.[2,3] The prevalence of thyroid autoantibodies was, however, similar to that of other reports.[3] While the findings of the current study are interesting and an important addition to the scarce Indian data on T1D autoimmunity, an attempt to explain such a high prevalence of islet autoantibodies is missing in the paper.

The exact reasons for the differences in the prevalence of islet autoantibodies in different patient populations of T1D are poorly understood at present. Factors such as genetic heterogeneity of the patient populations, the timing of estimation from disease onset, differences in laboratory assays and threshold limits, patient recruitment procedures, and sample size have been cited as the potential causes for the observed differences in the prevalence of autoantibodies.[4] It has been long known that a clear association between specific human leukocyte antigen haplotypes and development of islet autoantibodies exists.[5] Similar to the distinct regional variations in the prevalence of celiac autoimmunity, the variations in the prevalence of islet autoimmunity in T1D may have some association with genetic heterogeneity of patient populations across India.

The prevalence of islet autoantibodies is known to decrease with the duration of disease. The maximum positivity approaching 95% is present before the disease onset in at-risk individuals.[4] The positivity rate after the onset of T1D decreases with the progression of the disease. In this context, the information on the duration of disease at the time of estimation of islet autoantibodies is important. Furthermore, an age-wise analysis of positivity rate may help us to know whether younger patients have contributed significantly to the higher positivity. The prevalence of islet autoantibodies is higher in younger patients at the time of the diagnosis of T1D.[4]

The laboratory assays used for measuring islet autoantibodies may also affect the positivity rates. The gold standard assays for islet autoantibodies measurement are radio binding, bridge-ELISA, and electrochemiluminescence immunoassays.[4] Although the radioimmunoassay used in the current study is a standard method of islet autoantibody measurement, ensuring its standardization is a problem in the developing countries unlike in the developed countries where programs such as the Islet Autoantibody Standardization Program supervised by the Immunology of Diabetes Society ensure high qualitative standards in islet autoantibody measurement.[4] The majority of Western studies use 99th percentile or receiver operating characteristic analysis for determining the threshold limits of GAD65 assays as compared to Indian studies which generally use mean +3 standard deviation of normal values.[2,3] When the latter method is applied to small numbers of patients as in the present study, it may limit the applicability of the findings.[2] The threshold limits used in the current study are lower than those used in the previous Indian studies[2,3] which may have contributed to a higher frequency of GAD65 autoantibodies. It is thus important to mention how the thresholds for GAD65 autoantibodies were determined.

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Conflicts of interest
There are no conflicts of interest.

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