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Islet Autoantibody Profiles Among Individuals with Type 1 Diabetes Mellitus and Their Biological Siblings: A Pilot Family-Based Study from South India

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Abstract

Type 1 diabetes mellitus is an autoimmune disorder characterized by immune-mediated destruction of pancreatic β-cells, resulting in progressive insulin deficiency. Islet autoantibodies, including glutamic acid decarboxylase antibody and insulinoma-associated antigen-2 antibody, are established biomarkers of β-cell autoimmunity. First-degree relatives of individuals with type 1 diabetes mellitus have an increased risk of developing islet autoimmunity; however, data describing islet autoantibody profiles among biological siblings in Indian populations remain limited. This pilot study evaluated islet autoantibody profiles among individuals with type 1 diabetes mellitus and their biological siblings. A retrospective analysis of data and stored serum samples from a family-based observational study was conducted. Serum samples were evaluated for glutamic acid decarboxylase antibody and insulinoma-associated antigen-2 antibody using enzyme-linked immunosorbent assays. Positivity patterns were analysed descriptively using Fisher's exact test and an unadjusted odds ratio with a 95% confidence interval. Among 38 participants, 20 index cases from 20 families and 18 biological siblings from 10 families were included. Three siblings (16.7%; 3/18) were positive for at least one autoantibody. One (5.6%; 1/18) had isolated glutamic acid decarboxylase antibody positivity and two (11.1%; 2/18) had isolated insulinoma-associated antigen-2 antibody positivity; none showed dual positivity. Among index cases, 10 (50.0%; 10/20) were positive, including six (30.0%; 6/20) with isolated glutamic acid decarboxylase antibody positivity and four (20.0%; 4/20) with combined positivity. The exploratory comparison of siblings and index cases yielded an unadjusted odds ratio

of 5.00 (95% confidence interval 1.10–22.82; Fisher's exact p = 0.043). Biological siblings demonstrated islet autoantibody positivity without a known clinical diagnosis of diabetes, suggesting a potential role for family-based autoimmune risk assessment. Larger prospective studies with additional autoantibodies, metabolic assessment, and longitudinal follow-up are required to determine the clinical significance of these findings and assess progression risk.

Keywords: Biological Siblings, B-Cell Autoimmunity, Glutamic Acid Decarboxylase Antibody, Islet Autoantibodies, Insulinoma-Associated Antigen-2 Antibody, Type 1 Diabetes Mellitus.

Introduction

Type 1 diabetes mellitus (T1DM) is a chronic autoimmune endocrine disorder characterised by progressive immune-mediated destruction of insulin-producing pancreatic β-cells, resulting in insulin deficiency[1]. The development of T1DM involves a complex interplay of genetic susceptibility, particularly human leukocyte antigen (HLA) class II alleles, environmental triggers and immune dysregulation[2–7]. The appearance of islet autoantibodies often precedes the clinical onset of T1DM by months to years and serves as an important marker of β-cell autoimmunity [8 - 12].

Presymptomatic T1DM is recognized as a continuum comprising distinct stages characterized by islet autoimmunity, with or without dysglycaemia, before the onset of symptomatic hyperglycaemia[13]. This staging concept has highlighted the importance of identifying individuals during the presymptomatic phase, particularly among first-degree relatives [14-17].

Glutamic acid decarboxylase antibody (GADA) and insulinoma-associated antigen-2 antibody (IA-2A) are widely used biomarkers of islet autoimmunity in both children and adults [8–10, 12, 18, 19]. The presence of multiple islet autoantibodies is associated with a substantially increased risk of progression to symptomatic T1DM [6, 9, 15, 20]. The incidence of T1DM has increased in many populations over the past several decades, suggesting that changes in environmental exposures may contribute to disease susceptibility through the emergence of risk factors or the loss of protective factors [21].

First-degree relatives of individuals with T1DM, particularly siblings, have an increased risk of developing islet autoimmunity compared with the general population [14-17,22]. Identification of autoantibody-positive relatives may facilitate early risk assessment, clinical follow-up, and counselling regarding the potential development of diabetes before the onset of symptomatic hyperglycaemia [10, 23].

Although substantial data on familial islet autoimmunity are available from populations outside India, studies evaluating islet autoantibody profiles among siblings of individuals with T1DM in India remain limited. Potential differences in genetic background, environmental exposures, and disease characteristics between populations underscore the need for population-specific evidence to better understand familial islet autoimmunity in the Indian setting [19, 24, 25].

Therefore, this pilot study aimed to evaluate islet autoantibody profiles among individuals with T1DM and their biological siblings in a South Indian cohort, with particular emphasis on islet autoantibody positivity among siblings without a known clinical diagnosis of diabetes at the time of evaluation.

Materials and Methods

- **Study Design and Study Population**

This study was a retrospective analysis of data and stored serum samples from a family-based observational pilot study conducted in 2009 among individuals with a clinical diagnosis of T1DM and their available biological

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siblings. The original study was conducted in the Department of Immunology, The Tamil Nadu Dr M.G.R. Medical University, Chennai, India.

- **Inclusion criteria:**

➢ Individuals with a clinical diagnosis of type 1 diabetes mellitus identified during the study period.

➢ Patients with available clinical information and serum samples for GADA and IA-2A testing.

➢ Biological siblings of the index cases who provided informed consent and had available serum samples for GADA and IA-2A testing. Biological siblings did not have a known clinical diagnosis of diabetes mellitus at the time of evaluation

- **Exclusion criteria:**

➢ Individuals with clinical features suggestive of type 2 diabetes mellitus, secondary diabetes, monogenic diabetes, or other atypical forms of diabetes were excluded.

➢ Individuals with incomplete clinical records or unavailable serum samples for autoantibody testing were excluded.

The index cases were individuals with a clinical diagnosis of type 1 diabetes mellitus (T1DM) who underwent immunological evaluation, including testing for GADA and IA-2A. Autoantibody testing was performed as part of the immunological evaluation for diabetes classification.

- **Study Period**

The original study was conducted in 2009, and clinical details and laboratory investigations were recorded at the time of participant evaluation.

- **Sample Collection and Autoantibody Testing**

Serum samples collected from the index cases and their biological siblings were analysed for islet autoantibodies. Glutamic acid decarboxylase antibody (GADA) and insulinoma-associated antigen-2 antibody (IA-2A) were detected using commercially available enzyme-linked immunosorbent assay (ELISA) kits (EUROIMMUN, Lübeck, Germany), according to the manufacturer's instructions. Results were interpreted as positive or negative based on the manufacturer's recommended cut-off values.

- **Data Collection and Statistical Analysis**

Demographic characteristics, including age and sex, along with GADA and IA-2A results, were recorded for all participants. Autoantibody profiles were categorized as isolated GADA positivity, isolated IA-2A positivity, combined GADA and IA-2A positivity, or negative for both autoantibodies. The frequency of autoantibody positivity was summarised using descriptive statistics. The difference in overall autoantibody positivity between index cases and siblings was explored using Fisher's exact test, and the odds ratio (OR) with 95% confidence interval (CI) was calculated as a measure of association.

Because siblings were clustered within families, the comparison between index cases and siblings was considered exploratory. Fisher's exact test and the unadjusted odds ratio were used to describe the individual-level association; however, these analyses did not explicitly account for within-family correlation. The findings were therefore interpreted cautiously, with emphasis on descriptive estimates rather than definitive statistical inference. A two-sided p-value <0.05 was considered statistically significant.

Results

Study Population

A total of 20 index cases from 20 families were identified during the study period. Biological siblings were available for evaluation in 10 families, contributing a total of 18 siblings. The remaining 10 families had no biological sibling available for evaluation and were therefore not included in the sibling analysis.

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Demographic characteristics and islet autoantibody profiles were analysed for the index cases and siblings.

Islet Autoantibody Profile Among Index Cases

Among the 20 index cases, GADA was detected in 10 (50%) participants. IA-2A positivity was observed in 4 (20%), all of whom were also positive for GADA. Among the GADA-positive index cases (n = 10), six (30%) had isolated GADA positivity, whereas four (20%) had combined GADA and IA-2A positivity. No index case demonstrated isolated IA-2A positivity. The remaining 10 (50%) index cases were negative for both autoantibodies (Table 1).

Table 1: Demographic and Islet Autoantibody Characteristics of Index Cases (n = 20)

Characteristic	Index cases (n = 20)
Age, mean (SD)years	21.55(8.45)
Sex, n (%)	
Male	11 (55%)
Female	9 (45%)
GADA positivity, n (%)	10 (50%)
IA-2A positivity, n (%)	4 (20%)
Combined GADA and IA-2A positivity, n (%)	4 (20%)
Isolated GADA positivity, n (%)	6 (30%)
Isolated IA-2A positivity, n (%)	0 (0%)
Negative for both antibodies, n (%)	10 (50%)

Islet Autoantibody Profile Among Siblings

Among the 18 siblings evaluated, three (16.7%) demonstrated positivity for at least one autoantibody despite having no known clinical diagnosis of diabetes at the time of evaluation. One sibling showed isolated GADA positivity, while two siblings showed isolated IA-2A positivity. None of the siblings showed combined GADA and IA-2A positivity. The remaining 15 siblings (83.3%) were negative for both autoantibodies (Table 2).

Table 2: Islet Autoantibody Profile Among Biological Siblings of Individuals with T1DM (n = 18)

Autoantibody profile	Frequency
Isolated GADA positivity	1
Isolated IA-2A positivity	2
Both GADA and IA-2A positive	0
Negative for both antibodies	15
Positive for at least one autoantibody	3

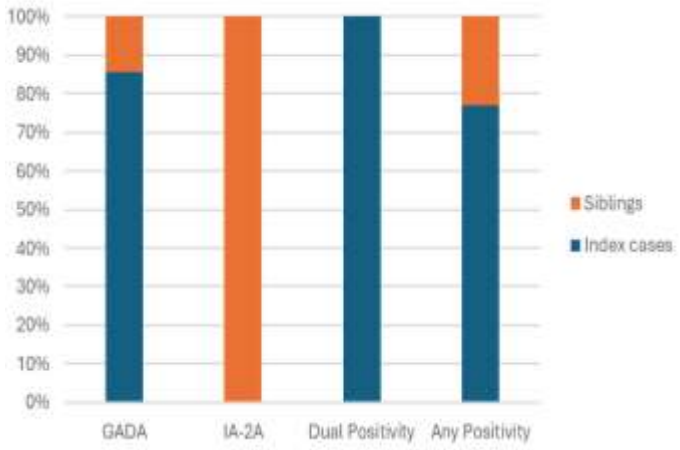


Figure 1: Distribution of Islet Autoantibody Positivity Among Index Cases and Biological Siblings

Comparison of Autoantibody Positivity Between Index Cases and Siblings

Overall, islet autoantibody positivity was detected in 10 of 20 (50.0%) index cases and 3 of 18 (16.7%) biological siblings. Autoantibody positivity was more frequent among index cases than among siblings. The exploratory analysis showed higher odds of autoantibody positivity among index cases than among biological siblings (OR 5.00; 95% CI 1.10–22.82; Fisher's exact p = 0.043). Because the sampling involved siblings clustered within families, this estimate does not account for within-family correlation and should be interpreted cautiously.

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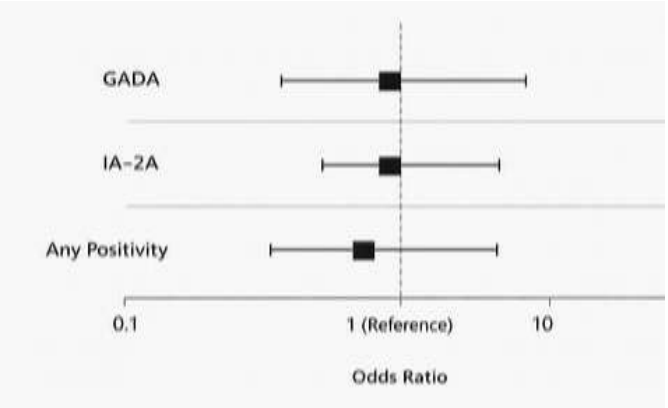


Figure 2: Forest plot of the unadjusted odds ratio for islet autoantibody positivity among index cases compared with biological siblings.

Family-Based Autoantibody Observations

Of the 20 families represented by the index cases, 10 had biological siblings available for evaluation, contributing a total of 18 siblings. The remaining 10 families (P3–P8, P12, P16, P18, and P19) had no biological siblings available for evaluation and were therefore not included in the family-wise sibling analysis presented in Table 3.

Among the 10 families with available siblings, both concordant and discordant autoantibody patterns were observed. One sibling showed GADA positivity concordant with GADA positivity in the corresponding index case, whereas two siblings showed isolated IA-2A positivity despite their respective index cases being negative for both tested autoantibodies.

Discordant autoantibody patterns were observed, with siblings demonstrating IA-2A positivity despite their respective index cases being negative. This underscores heterogeneity in autoimmune responses within families.

Table 3: Family-wise Distribution of Islet Autoantibody Profiles Among Index Cases and Their Siblings

Family ID	Sibling ID	Index case GADA	Index case IA-2A	Sibling GADA profile	Sibling IA-2A profile
P1	I	+	−	+	−
P2	I	+	+	−	−
	II	+	+	−	−
	III	+	+	−	−
P9	I	+	−	−	−
	II	+	−	−	−
P10	I	−	−	−	−
P11	I	−	−	−	−
P13	I	−	−	−	+
	II	−	−	−	−
P14	I	−	−	−	+
	II	−	−	−	−
P15	I	−	−	−	−
	II	−	−	−	−
P17	I	−	−	−	−
	II	−	−	−	−
P20	I	+	+	−	−
	II	+	+	−	−

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Total siblings tested =18

GADA, glutamic acid decarboxylase antibody;

IA-2A, insulinoma-associated antigen-2 antibody;

+, positive;

−, negative.

Note: The table includes only the 10 families with available biological siblings, contributing a total of 18 siblings

Discussion

Type 1 diabetes mellitus (T1DM) is an autoimmune disorder in which immune-mediated destruction of pancreatic β-cells results in progressive loss of insulin secretion [1, 2, 5, 6]. The presence of islet autoantibodies is an important marker of underlying autoimmune activity and may precede the clinical onset of diabetes by months to years [8-12]. This pilot family-based study evaluated the islet autoantibody profile among individuals with T1DM and their siblings in a South Indian cohort.

In this pilot family-based study, 10 of 20 individuals with T1DM (50%) and 3 of 18 biological siblings (16.7%) tested positive for at least one of the two islet autoantibodies. The unadjusted odds of autoantibody positivity were higher among index cases than among siblings (OR = 5.00, 95% CI: 1.10–22.82; Fisher's exact p = 0.043); however, this exploratory individual-level estimate did not account for within-family clustering and should therefore be interpreted cautiously.

Similar to previous observations, GADA is an important marker of autoimmune diabetes and is widely used in the evaluation of islet autoimmunity [8–10, 12, 18]. However, half of the index cases were negative for both tested autoantibodies. The absence of GADA and IA-2A in half of the index cases may reflect the limited autoantibody panel used in this study and highlights the potential contribution of additional markers such as zinc transporter 8 autoantibody (ZnT8A) and insulin autoantibody (IAA) [9, 12, 18, 19, 24].

An important observation of this study was the identification of islet autoantibody positivity among siblings of individuals with T1DM despite the absence of a known clinical diagnosis of diabetes at the time of evaluation. The antibody-positive siblings demonstrated isolated GADA or isolated IA-2A positivity, with no sibling showing combined positivity. These findings support further evaluation of family-based autoantibody assessment as a potential approach for identifying relatives with evidence of islet autoimmunity [17, 22, 26].

Previous studies have demonstrated that first-degree relatives of individuals with T1DM have an increased risk of developing islet autoimmunity compared with the general population. The presence of multiple islet autoantibodies is associated with a substantially higher likelihood of progression towards clinical diabetes [14–17, 27]. Although none of the antibody-positive siblings in the present study showed combined positivity for GADA and IA-2A, this finding highlights the potential relevance of clinical and metabolic surveillance of antibody-positive first-degree relatives in the context of established familial risk [14–17].

A noteworthy observation in the present study was the identification of isolated IA-2A positivity among siblings whose corresponding index cases were negative for both GADA and IA-2A. The family-level analysis further demonstrated both concordant and discordant autoantibody patterns between index cases and their siblings. One sibling showed GADA positivity concordant with GADA positivity in the corresponding index case, whereas two siblings showed isolated IA-2A positivity despite their respective index cases being negative for both tested autoantibodies. Although based on a limited number of families, these findings suggest

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heterogeneity in autoimmune responses among genetically related individuals and indicate that islet autoantibody profiles may differ between affected individuals and their siblings [12, 14, 16, 17, 26].

These findings support further evaluation of family-based autoantibody assessment in larger studies, particularly in settings where autoimmune risk assessment is not routinely performed. Early identification of at-risk relatives may provide opportunities for regular monitoring, education regarding symptoms, and appropriate clinical follow-up if evidence of metabolic abnormalities develops [14, 17, 23, 28]

This study has several limitations. The sample size was small, and only two islet autoantibodies (GADA and IA-2A) were evaluated. Additional markers such as ZnT8A and IAA were not assessed. Furthermore, longitudinal follow-up of antibody-positive siblings was not available; therefore, progression towards dysglycaemia or diabetes could not be evaluated. Future prospective studies involving larger family cohorts, comprehensive autoantibody panels, and long-term follow-up are required [12, 17, 27].

Despite these limitations, this pilot study provides preliminary evidence of islet autoimmunity among siblings of individuals with T1DM in a South Indian cohort. The identification of antibody-positive relatives, including those with isolated GADA or IA-2A positivity, highlights the potential value of family-based autoimmune risk assessment. Larger prospective studies incorporating comprehensive autoantibody profiling, metabolic evaluation, genetic risk assessment and longitudinal follow-up are required to determine the predictive significance of antibody positivity among first-degree relatives. Isolated autoantibody positivity among siblings may indicate the presence of islet-directed immune activity; however, longitudinal follow-up is required to determine whether such positivity represents persistent autoimmunity or predicts progression toward clinical diabetes. While progression risk is lower than with multiple antibody positivity, such findings warrant metabolic surveillance and family counselling.

Strengths of The Study

- This study provides preliminary family-based data on islet autoantibody profiles from a South Indian cohort.
- Both affected individuals with T1DM and their available siblings were evaluated, providing insight into familial autoimmune patterns.
- The study provides a family-level comparison of autoantibody profiles, allowing assessment of concordant and discordant autoimmune patterns within families.
- Detection of autoantibody positivity among siblings without a known clinical diagnosis of diabetes at the time of evaluation highlights the potential value of risk assessment in first-degree relatives.

Limitations of The Study

- Only two islet autoantibodies (GADA and IA-2A) were evaluated; additional markers such as ZnT8 antibody and insulin autoantibody were not available**.**
- Metabolic parameters such as oral glucose tolerance testing, HbA1c and C-peptide levels were not assessed in the siblings.
- Absence of longitudinal follow-up precludes assessment of progression from autoantibody positivity to dysglycaemia or overt diabetes**.**
- The relatively small sample size limited statistical comparisons and evaluation of associations between autoantibody status and clinical characteristics.
- The comparison between index cases and siblings was exploratory and did not explicitly account for within-family correlation; therefore, the estimated

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odds ratio, confidence interval, and p-value should be interpreted cautiously, as clustering may have affected the precision of the statistical estimates.

- The study was based on available biological siblings, which may have introduced selection bias and limited the generalizability of the findings

Conclusion

This study is among the first to provide family-based autoantibody data from a South Indian cohort, addressing a critical gap in regional evidence on islet autoimmunity among individuals with type 1 diabetes mellitus and their biological siblings. Although most siblings were negative for GADA and IA-2A, the identification of autoantibody positivity among siblings without a known clinical diagnosis of diabetes at the time of evaluation demonstrates the presence of islet autoantibodies in a subset of siblings.

Family-based autoantibody assessment may provide an opportunity for early identification of individuals with evidence of islet autoimmunity and support appropriate metabolic surveillance among first-degree relatives of individuals with T1DM. Further prospective studies involving larger cohorts, comprehensive autoantibody panels, metabolic assessment, and longitudinal follow-up are required to determine the risk of progression from islet autoimmunity to clinical diabetes [10, 12, 14, 15, 17, 27.]

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