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Frequency of Autoimmunity Markers in Type 2 Diabetes

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ABSTRACT

Background: Type 2 Diabetes (T2D) is associated with various autoimmune markers that may impact disease management and outcomes. Understanding the prevalence of these markers in T2D patients can provide insights into their metabolic profiles and potential complications. **Objective:** To assess the frequency of autoimmunity markers in patients with Type 2 diabetes. **Study Design:** Cross-sectional study. **Duration and Place of Study:** Conducted from December 2024 to April 2025 at the Department of General Medicine, PAF Hospital, Islamabad. **Methodology:** A total of 369 patients aged 30 to 70 years with a confirmed diagnosis of T2D were enrolled. Blood samples were analyzed for autoantibodies, including Anti-Glutamic Acid Decarboxylase (GAD), Antinuclear Antibodies (ANA), Antiparietal Cell Antibodies (APCA), and Anti-Smooth Muscle Antibodies (SMA). Statistical analyses were performed to assess associations between demographic factors and the presence of autoimmunity markers. **Results:** The participants had a mean age of 55.7 ± 6.18 years, with 61.5% being male. Among the cohort, 18.2% tested positive for GAD, 26.6% for ANA, 7.3% for APCA, and 4.6% for SMA. Significant associations were found between GAD positivity and older age, male gender, and higher BMI. **Conclusion:** The study reveals a notable prevalence of autoimmune markers in T2D patients, particularly GAD and APCA. These findings underscore the importance of screening for these autoantibodies in T2D patients to enhance risk stratification and management strategies.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is an insulin resistant and insulin deficient chronic syndrome characterized by hyperglycemia.[1] Type 1 Diabetes is an autoimmune disease that is mostly caused by the destruction of pancreatic beta cells, whereas Type 2 Diabetes is a disease having different pathogenesis.[2] T2DM is a pathophysiology of an expression of multifactorial the interactions of the lifestyle, environmental and genetic model, that disables insulin action and glucose metabolism.[3] This can lead up to chronic unduly high or low levels of blood glucose resulting in devastating end points such as cardiovascular disease, neuropathy, nephropathy, and retinopathy.[3] T2DM is usually considered as a non-autoimmune disease, whereas recent evidence now indicates that autoimmunity may play a role in the pathogenesis of T2DM in at least some patient populations.[4] In recent years, researchers are interested in searching for an overlap of the autoimmunity mechanisms with the traditional T2DM pathophysiology, so autoimmunity markers of Type 2 Diabetes have influenced researchers to pay more attention.[5]

Though T2DM is not typically an autoimmune disease, the literature has documented the presence of autoantibodies in a few patients, suggesting an intrinsic autoimmune nature.[6] Examples of such markers include antibodies to pancreatic antigens, nuclear structures, and other cellular components. The presence of such autoantibodies in T2DM patients negates the Type 1/Type 2 Diabetes dichotomy and introduces the possibility of a hybrid diabetes form known as Latent Autoimmune Diabetes in Adults (LADA).[7]

Anti-Glutamic Acid Decarboxylase Antibodies (GAD-Abs) are the most studied diabetes autoantibodies and are classically associated with Type 1 Diabetes.[8] However, GAD-Abs are also found in a proportion of T2DM subjects, specifically with LADA.[9] GAD-Abs target the enzyme glutamic acid decarboxylase, a step in the neurotransmitter synthesis and insulin release.[9] They might represent ongoing beta cell destruction and progressive insulin deficiency in patients with T2DM. And some T2DM patients have been found to have Antinuclear Antibodies (ANAs) typically found with systemic autoimmune disorders such as lupus.[10] Another complication related to the T2DM picture could be elevated ANAs that could reflect systemic inflammation or immune deregulation.[11] Other autoantibodies linked to T2DM include Antiparietal Cell Antibodies (APCAs) and Anti-Smooth Muscle Antibodies (ASMAs), although they tend to be rarer.

APCAs are directed against parietal cells of the stomach and can reduce acid secretion and cause gastrointestinal symptoms or malabsorption of nutrients in the patient.[12] In T2DM APCAs can indicate association with autoimmune gastritis or pernicious anemia that can worsen metabolic derangements.[13] Typically, ASMA's target are smooth muscle cells, and it is typically associated with autoimmune hepatitis or other liver disease.[14]

A study conducted by Piatkiewicz P. and colleagues found that the prevalence of various antibodies in individuals with type 2 diabetes were as follows: Anti-Glutamic Acid Decarboxylase Antibodies were present in 16% of patients, Antinuclear Antibodies in 22%, Antiparietal Cell Antibodies in 10%, and Anti-Smooth Muscle Antibodies in 4%.[15]

Investigating the frequency of autoimmunity markers in type 2 diabetes is essential to further elucidate the autoimmune mechanisms that may contribute to the development and progression of the disease. As far as type 2 diabetes is a quintessential metabolic disease, emerging data indicate the eventual role of autoimmune processes in its pathophysiology. The presence of specific autoantibodies can aid in early detection, improve diagnosis, and perhaps open windows for specific targeted therapy, augmenting a more generalized management of the disease.

METHODOLOGY

This cross-sectional study was conducted from December 2024 to April 2025 in the Department of General Medicine, PAF Hospital Islamabad. The study included a total of 369 Type 2 Diabetes patients, for a period of more than one year. The sample size was calculated by using the WHO sample size calculator, 95% confidence level, 2% margin of error, and expected frequency 4% of Anti-Smooth Muscle Antibodies in Type 2 Diabetes patients.[15] The inclusion criteria was patients must be between 30 and 70 years old and have a definite diagnosis of Type 2 Diabetes. Type 2 diabetes was accepted as the presence of any one of the following: fasting blood sugar ≥ 126 mg/dL after overnight fasting, random blood sugar ≥ 200 mg/dL, or a documented history of the use of antihyperglycemic drugs. Exclusion criteria were patients with a history of autoimmune diseases such as autoimmune thyroiditis, rheumatoid arthritis, inflammatory bowel disease, autoimmune hepatitis, or primary biliary cirrhosis. Patients with a history of pancreatic exocrine insufficiency, pancreatitis, impaired renal function (eGFR < 30 mL/min/1.73m^2), or those who were pregnant or lactating were excluded.

Following ethical approval and informed consent, baseline demographic data in the form of age, sex, body mass index (BMI), and diabetes duration were collected from each patient. Blood was then drawn for special investigations to determine the levels of a number of autoantibodies. Anti-Glutamic Acid Decarboxylase (anti-GAD65) antibodies were quantitated by a radioligand binding assay. Anti-GAD65 antibodies were considered positive if serum levels were >10 IU/mL. Antinuclear Antibodies (ANA) titers were quantitated by indirect immunofluorescence (IIF), and positivity was a serum ANA level of at least a dilution titer of 1:100. Antiparietal Cell Antibodies were tested using an ELISA test, and positivity was a titer >15 U/mL. Anti-Smooth Muscle Antibodies were quantitated by immunofluorescence using primate smooth muscle substrates, and positivity was a titer >1:80.

Data was analyzed by SPSS version 26. Descriptive statistics were used to report frequencies and percentages for categorical variables, i.e., gender and presence of various autoantibodies. For continuous variables, i.e., age, diabetes duration, and BMI, mean ± standard deviation or median (interquartile range) was reported, as applicable to the data distribution. The normality of the data was checked using the Shapiro-Wilk test. Stratification was used to control for effect modifiers, i.e., age, gender, BMI, and diabetes duration, and their effect on autoimmunity markers. Following stratification, the Chi-square test or Fisher's exact test was applied, with the significance level at $p \leq 0.05$.

RESULTS

The cohort consisted of 369 participants, with a mean age of 55.7 ± 6.18 years, a BMI of 30.54 ± 2.22 kg/m^2, and a mean duration of diabetes of 6.09 ± 1.51 years. Among the participants, 61.5% were male, and 38.5% were female, indicating a higher male representation (as shown in Table-I).

Table I

Patient Demographics (n=369)

Demographics		Mean ± SD
Age (years)		55.704±6.18
BMI (Kg/m^2)		30.543±2.22
Duration of Diabetes (years)		6.094±1.51
Gender	Male n (%)	227 (61.5%)
	Female n (%)	142 (38.5%)

Regarding the prevalence of autoimmunity markers, the study revealed that 18.2% of participants tested positive for Anti-Glutamic Acid Decarboxylase Antibodies (GAD), 26.6% had Antinuclear Antibodies (ANA), 7.3% had Antiparietal Cell Antibodies (APCA), and 4.6% had Anti-Smooth Muscle Antibodies (SMA). These results suggest a considerable variation in the frequency of autoimmunity markers within this cohort (as shown in Table-II).

Table II

Autoimmunity markers in Type 2 Diabetes

Autoimmunity markers	Frequency	% age
Anti-Glutamic Acid Decarboxylase Antibodies	67	18.2%
Antinuclear Antibodies	98	26.6%
Antiparietal Cell Antibodies	27	7.3%
Anti-Smooth Muscle Antibodies	17	4.6%

The stratified analyses examined the association of demographic factors (age, gender, BMI, and diabetes duration) with the presence of these autoimmunity markers. For GAD, significant associations were observed with age, gender, and BMI. A higher prevalence of GAD was noted in participants older than 50 years (21.8%) compared to those aged 50 years or younger (11.1%) (p-value: 0.011). Males had a higher prevalence of GAD (21.6%) compared to females (12.7%) (p-value: 0.031), and individuals with a BMI greater than 27 kg/m^2 had a

higher prevalence of GAD (22.4%) compared to those with a BMI ≤27 (9.2%) (p-value: 0.002). However, no significant association was found between GAD and diabetes duration (p-value: 0.318) (as shown in Table-III).

For ANA, the analysis did not reveal any significant demographic associations. Prevalence of ANA was similar across age groups (≤50 years: 28.6%, >50 years: 25.5%, p-value: 0.528), genders (males: 26%, females: 27.5%, p-value: 0.755), BMI categories (≤27 kg/m^2: 21.8%, >27 kg/m^2: 28.8%, p-value: 0.158), or diabetes duration (≤5 years: 24.9%, >5 years: 28%, p-value: 0.495) (as shown in Table-III).

Antiparietal Cell Antibodies (APCA) showed significant associations with age, BMI, and diabetes duration. The prevalence of APCA was significantly higher in individuals aged >50 years (11.1%) compared to those ≤50 years (0%, p-value <0.001). Similarly, the prevalence was higher in participants with a BMI >27 (10.8%) compared to those with a BMI ≤27 (0%, p-value <0.001). A strong association was also found with diabetes duration, with a marked increase in prevalence in those with diabetes duration >5 years (13%) compared to those with diabetes duration ≤5 years (0.6%, p-value <0.001). Gender did not significantly impact APCA prevalence (p-value: 0.326) (as shown in Table-III).

Lastly, Anti-Smooth Muscle Antibodies (SMA) did not show any significant associations with demographic factors. The prevalence of SMA was relatively low across all age groups (≤50 years: 4%, >50 years: 4.9%, p-value: 0.797), genders (males: 3.5%, females: 6.3%, p-value: 0.210), BMI categories (≤27 kg/m^2: 4.2%, >27 kg/m^2: 4.8%, p-value: 1.000), and diabetes durations (≤5 years: 3.6%, >5 years: 5.5%, p-value: 0.373) (as shown in Table-III and Graph-I).

Table III

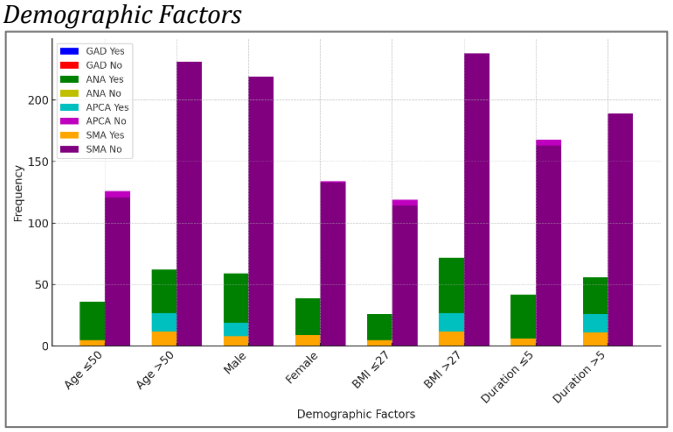
Association of Autoimmunity markers with Demographic Factors

Demographic Factors		Anti-Glutamic Acid Decarboxylase Antibodies		p-value
		Yes n(%)	No n(%)	
Age (years)	≤50	14 (11.1%)	112 (88.9%)	0.011
	>50	53 (21.8%)	190 (78.2%)	
Gender	Male	49 (21.6%)	178 (78.4%)	0.031
	Female	18 (12.7%)	124 (87.3%)	
BMI (Kg/m^2)	≤27	11 (9.2%)	108 (90.8%)	0.002
	>27	56 (22.4%)	194 (77.6%)	
Duration of Diabetes (years)	≤5	27 (16%)	142 (84%)	0.318
	>5	40 (20%)	160 (80%)	
Demographic Factors		**Antinuclear Antibodies**		**p-value**
		Yes n(%)	**No n(%)**	
Age (years)	≤50	36 (28.6%)	90 (71.4%)	0.528
	>50	62 (25.5%)	181 (74.5%)	
Gender	Male	59 (26%)	168 (74%)	0.755
	Female	39 (27.5%)	103 (72.5%)	
BMI (Kg/m^2)	≤27	26 (21.8%)	93 (78.2%)	0.158
	>27	72 (28.8%)	178 (71.2%)	
Duration of Diabetes (years)	≤5	42 (24.9%)	127 (75.1%)	0.495
	>5	56 (28%)	144 (72%)	
Demographic Factors		**Antiparietal Cell Antibodies**		**p-value**
		Yes n(%)	**No n(%)**	
Age (years)	≤50	0 (0%)	126 (100%)	<0.001*
	>50	27 (11.1%)	216 (88.9%)	
Gender	Male	19 (8.4%)	208 (91.6%)	0.326
	Female	8 (5.6%)	134 (94.4%)	
BMI (Kg/m^2)	≤27	0 (0%)	119 (100%)	<0.001*
	>27	27 (10.8%)	223 (89.2%)	
Duration of Diabetes (years)	≤5	1 (0.6%)	168 (99.4%)	<0.001*
	>5	26 (13%)	174 (87%)	
Demographic Factors		**Anti-Smooth Muscle Antibodies**		**p-value**
		Yes n(%)	**No n(%)**	
Age (years)	≤50	5 (4%)	121 (96%)	0.797*
	>50	12 (4.9%)	231 (95.1%)	
Gender	Male	8 (3.5%)	219 (96.5%)	0.210
	Female	9 (6.3%)	133 (93.7%)	
BMI (Kg/m^2)	≤27	5 (4.2%)	114 (95.8%)	1.000*
	>27	12 (4.8%)	238 (95.2%)	
Duration of Diabetes (years)	≤5	6 (3.6%)	163 (96.4%)	0.373
	>5	11 (5.5%)	189 (94.5%)	

Fisher Exact Test*

Graph I

Prevalence of Autoimmunity Markers in Type 2 Diabetes by Demographic Factors



DISCUSSION

The results describe a notable manifestation of autoimmunity markers in T2D patients with varied associations with demographic characteristics. Specifically, Anti-Glutamic Acid Decarboxylase (GAD) antibodies were significantly more prevalent in older patients, males, and those with higher BMI, suggesting a possible link between metabolic and autoimmune pathways. Antinuclear Antibodies (ANA), on the other hand, were not significantly linked with any of the

demographic factors, suggesting that ANA occurrence might not be substantially influenced by these characteristics in T2D patients.

The association of Antiparietal Cell Antibodies (APCA) with age, BMI, and diabetes duration is especially intriguing. The higher prevalence in older patients and those with longer diabetes duration could reflect the cumulative impact of immune dysregulation over time, a process already characterized in long-standing conditions like diabetes. The association of APCA with higher BMI is also consistent with recent evidence implicating obesity, a major feature of T2D, in the heightened autoimmune response. None of these associations were seen for Anti-Smooth Muscle Antibodies (SMA) with demographic variables, which could indicate SMA being less connected to these variables in the context of T2D.

The demographic profile revealed a higher male representation, with 61.5% males and 38.5% females, in concordance with findings from previous studies that commonly report a predominance of autoimmune diseases in males.[15,16] For autoimmunity markers, our findings revealed that 18.2% of subjects were positive for Anti-Glutamic Acid Decarboxylase Antibodies (GAD), 26.6% for Antinuclear Antibodies (ANA), 7.3% for Antiparietal Cell Antibodies (APCA), and 4.6% for Anti-Smooth Muscle Antibodies (SMA). The range of frequencies is suggestive of a significant prevalence of autoimmune markers, in concordance with the higher prevalence rates from the studies by Piatkiewicz et al. [15] where 16% of patients were identified with anti-GAD antibodies, and Primo et al. [17] which identified a prevalence of 8.9% for diabetes markers in autoimmune thyroid disease patients. In contrast, the studies by Litwińczuk-Hajduk et al. [16] and Moosaie et al. [18] reported lower frequencies, with GAD positivity at 30% and 11%, respectively, indicating variability in populations and methodologies.

Our stratified analysis showed associations between demographic factors and GAD presence. A greater GAD prevalence was found in participants older than 50 years (21.8%) compared to participants 50 years and younger (11.1%) (statistical significance: p-value: 0.011) and in males (21.6%) compared to females (12.7%) (statistical significance: p-value: 0.031). These findings are consistent with previous reports that age and gender are significant factors influencing GAD positivity rates.[15,16] However, contrasting with the correlations established in our study, Litwińczuk-Hajduk et al. [16] found no statistically significant correlation between diabetes duration and GAD, as in our study where no statistically significant correlation was found (p-value: 0.318).

In ANA, our study did not find statistically significant demographic correlations, with prevalence rates comparable across age groups and between genders. This is consistent with the findings described by Piatkiewicz et al. [15] where ANA was not correlated with diabetes complications, suggesting that while ANA is frequent, it may not be a useful marker of disease severity or disease progression in this context. ANA prevalence in our study group (26.6%) was higher than the 22% described in the type 2 diabetes cohort by Litwińczuk-Hajduk et al. [16] which can potentially reflect population differences.

For APCA, our results showed robust correlations with age (higher prevalence in those >50 years), BMI, and diabetes duration. The prevalence of APCA was statistically significantly higher in those >50 years (11.1%) compared to those ≤50 years (0%, p-value <0.001). Similarly, the prevalence was higher in those with BMI >27 (10.8%) compared to those with BMI ≤27 (0%, p-value <0.001). A highly significant association was also found with diabetes duration, with a statistically significant increase in prevalence in those with diabetes duration >5 years (13%) compared to those with diabetes duration ≤5 years (0.6%, p-value <0.001). These findings are in agreement with those of Moosaie et al. [18] where it was found that some autoantibodies could differentiate between diabetes types and their metabolic profiles, again highlighting the progressive nature of autoimmune responses.

Lastly, SMA did not have robust demographic associations in our cohort, similar to other studies in which SMA positivity was low in diverse demographics.[16,17] The prevalence of SMA in our study was 4.6%, which was very similar to the 4% in the Moosaie et al. study.[18] This suggests that while SMA may be present, it is less representative of the autoimmune process compared to GAD or ANA.

By identifying these markers, clinicians are able to stratify risk more effectively in patients and customize interventions for improved outcomes. Variability in prevalence rates between studies indicates that more studies are required to elucidate the underlying determinants of these differences, including genetic, environmental, and demographic factors.

There are certain limitations to this study, however. Being a single-center study, the findings may not be generalizable to larger populations. The cross-sectional design also constrains the ability to establish cause-and-effect relationships between autoantibody presence and disease progression. A multi-center study with a larger, more diverse population would render the findings more robust and provide more insights into the relationship between autoimmunity and diabetes.

CONCLUSION

In conclusion, our study highlights the frequency of various autoimmune markers in patients with Type 2 Diabetes. The findings indicate Anti-Glutamic Acid Decarboxylase Antibodies to be more prevalent in older patients, males, and patients with higher BMI, while Antiparietal Cell Antibodies have more prevalence in older patients, patients with higher BMI, and longer diabetes duration. Antinuclear and Anti-Smooth Muscle Antibodies, however, did not correlate significantly with demographic variables.

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