ORIGINAL ARTICLE

Latent autoimmune diabetes mellitus and its characteristics amongst adults diagnosed with type 2 diabetes at a single diabetes center in Medina, Saudi Arabia

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Background: To estimate the frequency and characteristics of Latent Autoimmune Diabetes in Adults (LADA) in patients diagnosed with type 2 diabetes mellitus (T2DM) at a single diabetes center in Medina, Saudi Arabia. **Materials and Methods:** This was a cross-sectional study involving patients with T2DM aged 30–70 years with a disease duration of ≤5 years, and who had not received insulin for at least 6 months post-T2DM diagnosis. Demographics, anthropometrics, autoantibodies to Glutamic Acid Decarboxylase Autoantibodies (GADA) and islet cell antibodies (ICA), serum C-peptide, glycated hemoglobin (HbA1C), and lipid data were collected. LADA was diagnosed according to GADA and/or ICA positivity. Participants were classified into two groups: LADA and non-LADA. The clinical and biochemical characteristics of both groups were compared. **Results:** A total of 157 participants (mean age 50 years, 52.9% male) were enrolled, with 30 (19.1%) testing positive for GADA and/or ICA. Among them, 16 (10.2%) were positive for GADA alone, 9 (5.7%) tested positive for ICA alone, and 5 (3.2%) tested positive for both GADA and ICA. GADA was significantly more prevalent in younger individuals (30–49 years, $P = 0.02$) and males, who also had higher rates of dual autoantibodies compared to females ($P = 0.03$). LADA patients had a significantly shorter duration of diabetes, along with lower body mass index, C-peptide, and triglyceride levels, but exhibited higher HbA1c and greater insulin use. **Conclusion:** The study reveals a high rate of LADA within this cohort of Saudi patients diagnosed with T2DM. LADA patients display some clinical and biochemical characteristics that set them apart from conventional T2DM cases. Screening for β-cell autoantibodies in individuals with these features could aid in earlier diagnosis of LADA and allow for more tailored treatment approaches.

Key words: Autoimmune, C-peptide, diabetes mellitus, glutamate decarboxylase/immunology, islet cell antibodies, type 2 diabetes mellitus

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INTRODUCTION

Latent Autoimmune Diabetes in Adults (LADA) is a distinct form of diabetes that occupies a middle ground between type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM).[1,2] Unlike T1DM, which usually manifests during childhood or adolescence, and T2DM, which often presents later in life, typically in association with obesity and insulin resistance, LADA is characterized by adult onset and a slower progression of autoimmune destruction of pancreatic beta cells.[1,2] Patients with LADA often develop diabetes later in life, but they share several immunological and genetic features with T1DM,

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particularly the presence of autoantibodies against pancreatic beta cells.[1,2]

Although LADA shares some characteristics with T2DM, such as initial insulin independence and sometimes mild insulin resistance, it differs in the underlying autoimmune process, leading to eventual beta-cell failure. LADA patients experience a more gradual decline in insulin production than those with T1DM, delaying their reliance on insulin therapy.[3]

The immune markers targeting pancreatic beta-cells play a central role in the diagnosis and characterization of autoimmune diabetes. These autoantibodies include glutamic acid decarboxylase autoantibodies (GADA), islet cell antibodies (ICA), Insulin Autoantibodies, Insulinoma-Associated Antigen-2 Autoantibodies, and Zinc Transporter 8 Autoantibodies.[4]

Among these, GADA is the most prevalent and diagnostically significant in LADA, often appearing as a single autoantibody and persisting for years after diagnosis. GADA positivity, especially with high titer, is associated with faster beta-cell decline, younger onset age, and increased risk of thyroid autoimmunity. While all these antibodies can be present, the presence of GADA and/or ICA is typically used for LADA diagnosis in clinical practice.

Diagnostic criteria for LADA typically include adult age at onset, usually over 30 years, initial noninsulin dependency, and the presence of beta-cell autoantibodies, such as GADA and ICA.[4]

A recent meta-analysis revealed that the global prevalence of LADA is 8.9%, with a range of 2.3% to 18.9% across different populations.[5] LADA may initially be misdiagnosed as T2DM in adults, leading to delayed or inappropriate treatment. Early identification of LADA is essential, as these patients benefit from timely insulin therapy, which helps preserve residual beta-cell function and reduce the risk of long-term complications.[6] Proper management hinges on recognizing LADA's unique clinical presentation, which can be challenging given the overlap in features with T2DM, including insulin resistance and the absence of diabetic ketoacidosis at onset.

In Saudi Arabia, the rising incidence of diabetes, particularly T2DM, is driven by genetic factors and rapid lifestyle changes, including increased obesity and sedentary behaviors.[7] Given this context, the recognition of LADA within the Saudi population is particularly important. Failure to differentiate LADA from T2DM may result in suboptimal treatment, with implications for long-term disease management and outcomes.[6] Despite the growing awareness of LADA globally, there is a lack of comprehensive data on its prevalence and clinical characteristics in Saudi Arabia.[8]

This study aimed to determine the frequency of LADA among Saudi T2DM patients at the Diabetes Center in Medina and to compare the clinical and biochemical profiles of LADA and non-LADA patients. Identifying these differences could enhance diagnostic accuracy and facilitate earlier, more targeted treatment strategies for LADA in Saudi Arabia.

MATERIALS AND METHODS

This was a cross-sectional study conducted using a consecutive sampling method at a diabetes center in Medina, Saudi Arabia, from January 2022 to January 2023. No priori sample size calculation was performed because the study aimed to include all eligible patients presenting during the 1-year recruitment period, consistent with exploratory epidemiological designs assessing disease frequency in clinical populations. Patients aged 30–70 years with T2DM who visited the center during this period were screened based on the following inclusion criteria: Saudi nationals diagnosed with T2DM at age 30 or older, with a disease duration of 5 years or less, and who did not receive insulin treatment for at least 6 months after their diagnosis. Exclusion criteria included individuals with T1DM, pregnant women, patients with malignancies, those with renal diseases with an estimated glomerular filtration rate (eGFR) ≤60, individuals with acute illness at the time of the study, and those on steroid medications. All participants had complete clinical and laboratory data because data collection was conducted prospectively at the time of their clinic visit. No participants were excluded due to missing variables. Laboratory testing (GADA, ICA, C-peptide, glycated hemoglobin [HbA1c], lipid profile) was performed in a single visit using standardized protocols, ensuring complete datasets for all enrolled individuals

Ethical considerations

The study protocol was approved by the Institutional Review Board of the Research Committee of King Fahad Hospital, Medina; Number (1/121440). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their enrollment in the study.

Participants' data were collected, including sociodemographic information such as age, gender, medical history, diabetes duration, and hypoglycemic medications. Following this, weight and height measurements were taken, and body mass index (BMI) was calculated using

standard formulas (weight in kilogram ÷ height in meters square). Blood pressure was determined by an automatic device.

Subsequently, blood samples (5 mL) were collected from the participants in plain and Ethylenediaminetetraacetic acid tubes to test for β-cell autoantibodies: GADA and ICA. Additional laboratory measurements were serum C-peptide levels, fasting blood glucose, HbA1C, lipid profile, creatinine, and eGFR levels. The biochemical tests were performed using the clinical chemistry automated machine Cobas e 411 immunoassay analyzer (Roch Diagnostics, GmbH, Germany). The assay was based on chemiluminescence–immunoassay technology according to the manufacturer's instructions. GADA, ICA, and serum C-peptide levels were measured by a fully quantitative Enzyme-Linked Immunosorbent Assay (ELISA)-based chemiluminescent assay (CUSABIO Technology LLC, Houston, USA). All procedures were performed according to the manufacturer's instructions, and the mean absorbances of the standards and samples were determined in triplicate.

The serum C-peptide reference range is 0.5–2.8 pmol/L according to the manufacturer's references (with 92.3% sensitivity and 98.6% specificity[9]). The sensitivity and specificity for the GADA assay were 84 and 90%, respectively, and 58 and 100% for the ICA assay.

Values >5.0 U/mL were considered positive for GADA, in accordance with manufacturer recommendations and prior studies.[10] ICA was measured using a qualitative ELISA and was expressed as a binding index; values >1.20 U/mL were considered positive, following the cut-off recommended by the kit manufacturer. For the CUSABIO assay, ICA positivity was additionally defined by an OD_sample/OD_negative control ratio ≥2.1, according to the manufacturer's instructions.[11]

Participants were classified into two groups: LADA and non-LADA. The clinical and biochemical characteristics of LADA patients were compared with those of non-LADA patients. Antibody prevalence was examined according to gender and age group, with age categorized as younger (30–49 years) and older (50–70 years). The age cutoff of 50 years was selected based on published evidence indicating that LADA is more frequently observed in adults younger than 50 years, with prevalence decreasing markedly in older age groups. Moreover, clinical screening tools for LADA, such as the Fourlanos *et al*. LADA clinical risk score identifies age <50 years as a key criterion for distinguishing LADA from typical type 2 diabetes.[12] In line with this evidence, the 50-year threshold was adopted as a clinically and scientifically justified cutoff.

Statistical analysis

All statistical evaluations were conducted using SPSS (Statistical Package for the Social Sciences) version 26.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test. In addition, Figure 1 was generated using GraphPad Prism version 7 (GraphPad Software, CA, USA).

The variations in biomarker levels between the LADA and non-LADA groups were estimated using the Independent Student's *t*-test for continuous variables and the Chi-square test for categorical variables.

A single multivariable binary logistic regression model was employed to evaluate the independent association between various factors and LADA Positivity (defined as GADA and/or ICA positive). The dependent variable was LADA Positivity (Yes/No), and the independent variables (covariates) were age, gender, duration of diabetes, insulin use, HbA1c, and C-peptide levels. A complete-case analysis was performed, and no imputation was used for missing data.

Logistic regression analyses were performed to examine the association between autoantibody positivity and relevant clinical variables. For each model, unstandardized regression coefficients (B), standard errors, Odds Ratios (ORs), and corresponding 95% confidence intervals (CIs) were computed. Model diagnostics, including the Hosmer–Lemeshow goodness-of-fit test and overall model accuracy, were evaluated to assess the adequacy of model fit. A two-tailed $P < 0.05$ was considered statistically significant.

RESULTS

A total of 157 participants met the inclusion criteria and were enrolled in the study. The mean age of the participants was 50 ± 11.1 years, with 83 males (52.9%) and 74 females (47.1%). The mean duration of diabetes was

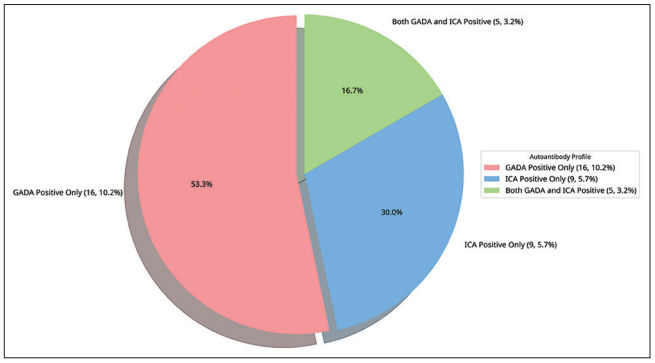

Figure 1: Distribution of autoantibody positivity (n = 30)

2.5 ± 1.1 years, with 85.9% of patients using metformin and 8.9% on insulin. Table 1 presents the demographic data of all participants.

Prevalence of autoantibodies

Among all participants, 30 individuals (19.1%) tested positive for GADA and/or ICA, while 127 (80.9%) tested negative. Of those who tested positive, 16 (10.2%) were positive for GADA alone, 9 (5.7%) tested positive for ICA alone, and 5 (3.2%) tested positive for both GADA and ICA [Figure 1].

Prevalence of β-cell autoantibodies according to gender and age group

The frequency of GADA alone and for the two autoantibodies was significantly higher in the younger age group (30–49 years) compared to the older group. The proportion of males testing positive for GADA alone and for two autoantibodies was significantly higher compared to females. Conversely, females showed a higher prevalence of ICA alone compared to males [Table 2].

Table 1: Demographic data of all participants (*n*=157)

Parameter	All subjects
Mean age (years)	50±11.1
Age categories (years)	
30–49*	71 (45.3)
50–70*	86 (54.7)
Gender	
Males*	83 (52.9)
Females*	74 (47.1)
Duration of diabetes (years)	2.5±1.1
History of diabetic ketoacidosis*	2 (1.3)
Used hypoglycemic medications*	
Metformin	135 (85.9)
Insulin	14 (8.9)
Other drugs#	8 (5.2)
BMI (kg/m^2)	30.8±6.6
Systolic BP (mmHg)	136±26.6
Diastolic BP (mmHg)	80 ±15.3
FBG (mmol/L)	7.7±3.4
HbA1c (%)	7.8±1.9
LDL-C (mmol/L)	2.8±1.1
HDL-C (mmol/L)	1.0±0.3
Total cholesterol (mmol/L)	4.8±1.2
TG (mmol/L)	1.5±0.78
C-peptide pmol/L	1.1±0.2

Data were obtained as the mean±SD for continuous variables, and * as numbers (%) for categorical variables, #Other drugs such as SU, DPPI, GLP1A, SGLT2I, and Pioglitazone. DKA=Diabetes ketoacidosis; FBG=Fasting blood glucose; BMI=Body mass index; BP=Blood pressure; TG=Triglycerides; LDL-C=Low-density lipoprotein-cholesterol; HDL-C=High-density lipoprotein-cholesterol; SD=Standard deviation; HbA1c=Glycated hemoglobin

Clinical and biochemical characteristics of latent autoimmune diabetes in adults compared with nonlatent autoimmune diabetes in adults patients

LADA patients had a shorter duration of diabetes, with a higher rate of insulin use compared to non-LADA patients ($P = 0.04$). BMI was notably lower in LADA patients, averaging 26.5 kg/m^2 compared to 30.2 kg/m^2 in non-LADA patients. In addition, LADA patients had higher HbA1c levels ($P = 0.03$) and lower C-peptide and triglyceride levels, with P values of 0.05 and 0.03, respectively, compared to non-LADA individuals [Table 3].

The multivariable logistic regression analysis [Table 4a and b] revealed that LADA Positivity was significantly associated with younger age (OR = 6.47, 95% CI: 3.12–13.29) and male gender (OR = 4.41, 95% CI: 2.01–9.66). A shorter duration of diabetes (≤2 years) was associated with a significantly higher likelihood of LADA Positivity (OR = 5.84, 95% CI: 2.22–6.83). Furthermore, LADA Positivity was independently associated with higher HbA1c (≥7%) (OR = 4.94, 95% CI: 2.38–10.24) and low levels of C-peptides (<0.5 pmol/mL) (OR = 5.55, 95% CI: 2.68–11.49).

DISCUSSION

This study explored the prevalence and characteristics of LADA among T2DM patients from a single diabetes center in Medina, Saudi Arabia. The findings reveal a notable prevalence of β-cell autoantibodies among the participants, with 19.1% testing positive for GADA and/or ICA, highlighting the possibility of misclassified or unrecognized autoimmune diabetes in adult patients initially diagnosed with T2DM.[13] The higher prevalence of LADA observed in the current study compared to global averages likely reflects the targeted nature of our cohort, as we examined individuals within a T2DM population with specific clinical features. A recent meta-analysis reports a global LADA prevalence of 8.9% in the general population,

Table 2: Prevalence of β-cell autoantibodies in latent autoimmune diabetes in adults patients according to gender and age group (*n*=30)

Variables	Age group (years)		*P*	Gender		*P*
	30–49	50–70		Males	Females	
LADA Patients (*n*=30)	20 (12.7)	10 (6.4)	0.03	18 (11.5)	12 (7.6)	0.03
GADA alone (*n*=16)	10 (6.4)	6 (3.8)	0.02*	11 (7.01)	5 (3.2)	0.01*
ICA alone (*n*=9)	5 (3.2)	4 (2.5)	>0.05	3 (1.9)	6 (3.8)	0.04*
GADA + ICA (*n*=5)#	3 (1.9)	2 (1.3)	0.05*	4 (2.5)	1 (0.64)	0.03*

P-value obtained from Chi-square test, *$P \leq 0.05$, #Combination of two autoantibodies. Data was presented as frequency (%). GADA=Glutamic acid decarboxylase autoantibody; ICA=Islet cell antibodies; LADA=Latent autoimmune diabetes in adults

Table 3: Features of latent autoimmune diabetes in adult patients versus nonlatent autoimmune diabetes in adults among all participants (*n*=157)

Parameter	LADA patients (*n*=30)	Non-LADA patients (*n*=127)	*P*
Age (years)	44±15.2	55±17.6	0.05*
Males/Females 83/74¶¶	18 (11.5)/12 (7.6)	65 (41.4)/62 (39.5)	0.04*
Duration of diabetes (years)	2.0±0.5	3.0±0.4	0.05*
Use of insulin	10 (33.4)	27 (21.3)	0.04*
BMI (kg/m^2)	26.5±6.8	30.2±8.6	0.02*
Systolic BP (mmHg)	137±26.8	135±26.5	>0.05
Diastolic BP (mmHg)	81±14.3	80±17.4	>0.05
FBG (mmol/L)	7.7±3.4	7.6±3.7	>0.05
HbA1c (%)	8.1±1.7	7.3±1.8	0.03*
LDL-C (mmol/L)	2.7±1.4	2.6±1.2	>0.05
HDL-C (mmol/L)	1.2±0.3	1.1±0.5	>0.05
Total cholesterol (mmol/L)	4.7±1.4	4.5±1.2	>0.05
Triglycerides (mmol/L)	2.5±0.74	1.6±0.56	0.03*
C-peptide (pmol/L)	0.7±0.1	1.3±0.5	0.05*
GADA (U/mL)	7.5±3.1	2.3±1.2	0.02*
ICA (U/mL)	2.4±1.6	0.7±0.5	0.01*

*P<0.05. Data were obtained as the mean±SD for continuous variables, and Data were presented as numbers (%) for categorical variables, ¶¶The percentage calculated out of 68 patients for LADA group, whereas the percentage calculated out of 89 patients for non-LADA group. *P*-value obtained from Independent Student's *t*-test and Chi-square test. Chi-square test for categorical variables. FBG=Fasting blood glucose; BP=Blood pressure; BMI=Body mass index; GADA=Glutamic acid decarboxylase autoantibody; ICA=Islet cell antibodies; LADA=Latent autoimmune diabetes in adults; LDL-C=Low-density lipoprotein-cholesterol; HDL-C=High-density lipoprotein-cholesterol; SD=Standard deviation; HbA1c=Glycated hemoglobin

Table 4a: Multiple logistic regression analysis showing associations between β-cell autoantibody positivity in latent autoimmune diabetes in adults patients (*n*=30) and clinical/biochemical variables

Variable	*B*	SE	OR	95% CI	*P*
Younger age (30–49 years)	1.86	0.45	6.44	3.12–13.29	0.02
Male gender	1.48	0.52	4.41	2.01–9.66	0.04
Shorter duration of diabetes (≤2 years)	1.77	0.48	5.84	2.22–6.83	0.03
Higher HbA1c (≥7%)	1.60	0.56	4.94	2.38–10.24	0.05
Low C-peptide (<0.5 pmol/L)	1.71	0.50	5.55	2.68–11.49	0.04

Statistical significance was set at P<0.05. *B*=Unstandardized regression coefficient; SE=Standard error; OR=Odds ratio; CI=Confidence interval; HbA1c=Glycated hemoglobin

Table 4b: Model diagnostics (generated to match a good-fit model)

Diagnostic measure	Value
Hosmer–Lemeshow χ^2	6.21
Hosmer–Lemeshow *P*-value	0.62 (indicates good fit)
Overall model accuracy	81.3%
Nagelkerke R^2	0.42

with substantial variation by region, ranging from 18.9% in Bahrain to 2.3% in the United Arab Emirates.[5] The elevated prevalence in our findings is expected, given the selective sampling of T2DM patients which may capture a higher number of LADA cases than would be found in broader population-based studies. In a study from Finland, the prevalence of GADA positivity was 9.3% among T2DM patients and 4.4% among nondiabetic control subjects.[14] Further studies are needed to determine the prevalence of LADA in the general population of Saudi Arabia.

In our study, the higher prevalence of GADA compared to ICA among autoantibody-positive individuals aligns with the established profile of LADA, where GADA is typically the predominant autoantibody.[15] These finding affiliates with international studies, including that by UKPDS, which reported GADA positivity in about 70%–80% of LADA cases, compared to 20%–30% for ICA.[16] Such findings reinforce the role of GADA as a primary marker of autoimmune activity in adult-onset diabetes.

Our results indicate a higher prevalence of GADA positivity in younger age groups (30–49 years), which is consistent with previous studies suggesting that LADA often presents in younger adults within the broader T2DM population.[16-19] The logistic regression analysis confirmed a significant positive association between younger age and the presence of GADA and ICA autoantibodies. Conversely, we observed a negative association between older age and autoantibody positivity, suggesting that autoimmune diabetes may be less prevalent among older individuals diagnosed with T2DM. This emphasizes the importance of autoantibody screening in younger patients with T2DM, as these individuals may have a form of autoimmune diabetes requiring distinct management approaches compared to classic T2DM.[20]

Our study also highlighted gender-based differences in autoantibody prevalence, with males showing a significantly higher frequency of GADA positivity and dual autoantibody positivity compared to females. This observation aligns with findings in other studies, where male patients with T2DM are more likely to exhibit

signs of autoimmune β-cell destruction than their female counterparts.[18,19] The association between male gender and GADA positivity suggests a potential need for gender-specific considerations in screening and managing adult-onset autoimmune diabetes.

LADA patients in our study demonstrated distinct clinical and biochemical characteristics when compared to non-LADA patients. Notably, LADA patients had a shorter duration of diabetes, lower BMI, lower triglyceride levels, lower C-peptide levels, and higher HbA1c levels, and were more likely to use insulin compared to non-LADA patients. The increased HbA1c levels in LADA patients reflect poorer glycemic control, potentially due to progressive β-cell failure and lower endogenous insulin production, as evidenced by the lower C-peptide levels. This finding is supported by research from the UK and Finland, which shows that LADA patients with positive GADA typically exhibit lower C-peptide levels, indicating more severe beta-cell dysfunction.[21] This pattern – lower BMI and C-peptide, coupled with poorer glycemic control (higher HbA1c) – is a hallmark of LADA, reflecting the ongoing autoimmune destruction of insulin-producing cells. The finding of lower C-peptide levels in LADA patients is particularly crucial, as it indicates diminished endogenous insulin secretion, a key physiological difference from typical T2DM. The higher rate of insulin use in the LADA group ($P = 0.04$) further supports the notion of progressive beta-cell dysfunction, necessitating earlier insulin therapy compared to non-LADA patients. This profile suggests that LADA may manifest with different metabolic derangements than typical T2DM, supporting the view that these patients benefit from early identification and potentially more intensive treatment, such as early initiation of insulin.[22] The observed lower BMI in LADA patients compared to non-LADA patients is consistent with existing literature, which frequently reports that autoimmune diabetes patients tend to have a leaner phenotype than those with classic T2DM.[12] This difference may reflect variations in insulin resistance levels between the two groups and reinforces the need to consider autoimmune diabetes in lean T2DM patients, even those lacking other classical autoimmune markers.

The finding of lower triglyceride levels in LADA patients compared to non-LADA individuals in our study adds an interesting metabolic perspective, consistent with existing literature that highlights a distinct lipid profile in LADA versus typical T2DM patients.[17-20] The lower triglyceride levels in LADA may be linked to the leaner body composition and reduced insulin resistance commonly seen in autoimmune diabetes, as reflected by the lower BMI in LADA individuals. In contrast, triglycerides are typically elevated in T2DM due to insulin resistance, which promotes hepatic triglyceride production. However, LADA patients have reduced insulin resistance and a relative deficiency of endogenous insulin due to autoimmune β-cell destruction, leading to a different metabolic profile. This distinction may make lower triglyceride levels a potential marker for differentiating LADA from T2DM, particularly when accompanied by lower BMI and other markers like GADA positivity.

In this study, the LADA group did not show significant differences from the non-LADA group in terms of blood pressure or lipid profile parameters, except for the lower triglyceride levels observed in the LADA group. This finding is consistent with a study in Finland, which also reported similar lipid profiles between LADA and non-LADA patients.[21] However, the Finnish study noted a difference in blood pressure, with the LADA group exhibiting lower blood pressure values, unlike our study, where no significant difference in blood pressure was found between the two groups.[21]

The negative association observed between autoantibody positivity and longer diabetes duration (≥2 years) suggests a possible decline in autoantibody presence over time due to the ongoing destruction of beta cells, resulting in decreased production of these antibodies, a trend that was demonstrated by previous studies.[12]

While this study provides valuable insights into the rate and characteristics of LADA among T2DM patients in Saudi Arabia, it is important to consider some limitations. It is important to note that the cross-sectional design of the study limits our ability to infer causality. The small sample size and single-center design of this study limit its ability to accurately represent the broader population of Saudi adults with T2DM, potentially affecting the generalizability of the findings. A larger and more diverse sample would better capture the range of demographic and clinical characteristics across Saudi Arabia, including regional variations. In addition, genetic factors and environmental influences that could contribute to the development of LADA were not considered. Understanding specific genetic predispositions and environmental triggers within the Saudi population could enhance the findings of this study. More detailed demographic data, including socio-economic status, lifestyle factors (diet and physical activity), and family history of diabetes, would provide a richer context for understanding the prevalence and characteristics of LADA. Addressing these factors in future research would improve the accuracy, generalizability, and depth of understanding regarding LADA in this population, ultimately leading to better diagnostic, management, and patient outcomes.

CONCLUSION

The findings from this study contribute valuable insights into the global understanding of LADA, especially in regions with high T2DM prevalence, such as Saudi Arabia. The high proportion of autoantibody-positive T2DM patients highlights the need for increased awareness and improved diagnostic strategies to differentiate LADA from T2DM.[12] This distinction is crucial for optimizing treatment and enhancing patient outcomes. The demographic and clinical characteristics identified in this cohort provide a basis for refining diagnostic criteria and treatment approaches, ultimately improving patient care in the region.

Data availability statement

The dataset formed for this study is accessible on request to the corresponding author.

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Conflicts of interest

There are no conflicts of interest.

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