Original Article

Biochemical and Metabolic Profiles in Gad-Positive Versus Gad-Negative Type 2 Diabetes Mellitus Patients in Benin City, Nigeria

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Article History
Submitted: 10/05/2026; Accepted: 19/05/2026: Published: 28/05/2026

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ABSTRACT

The autoimmune subtype of diabetes in adults, characterized by the presence of glutamic acid decarboxylase-65 (GAD-65) autoantibodies, may present with a distinct biochemical and metabolic phenotype compared to classical type 2 diabetes mellitus (T2DM). Data from sub-Saharan Africa on the metabolic correlates of GAD positivity remain limited. This study compared biochemical and metabolic profiles, including glycemic control, lipid parameters, C-peptide levels, and microalbuminuria, between GAD-positive and GAD-negative patients with apparent T2DM attending the University of Benin Teaching Hospital (UBTH), Benin City, Nigeria. A descriptive cross-sectional study was conducted among 240 consecutively recruited T2DM patients. Fasting blood samples were analyzed for GAD-65 autoantibodies, C-peptide, HbA1c, fasting blood glucose, and a full lipid profile. GAD positivity was defined as a titer of 0.62 ng/ml or above. Student t-test, Mann-Whitney U test, and chi-square tests were used for comparisons; a p value of 0.05 or less was considered statistically significant. Of 240 patients, 67 (27.9%) were GAD-positive. GAD-positive patients had significantly lower median C-peptide (0.40 IQR 0.80 ng/ml vs. 0.60 IQR 1.00 ng/ml, p=0.050) and a higher prevalence of insulinopenia (35.8% vs. 23.7%). Mean serum triglycerides were significantly lower in GAD-positive patients (143.00 vs. 188.95 mg/dl, p=0.025). No significant differences were found in HbA1c, fasting blood glucose, other lipid fractions, or microalbuminuria. GAD-positive T2DM patients demonstrated greater beta-cell dysfunction and lower triglyceride levels, consistent with an autoimmune diabetic phenotype. Routine GAD antibody and C-peptide screening is recommended for T2DM patients in Nigeria.

Keywords: Biochemical profiles, C-peptide, GAD-65 autoantibodies, LADA, Lipid profile, Nigeria, Type 2 diabetes mellitus

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a heterogeneous metabolic disorder characterized by progressive insulin resistance and relative insulin deficiency.[1,2] However, a subset of patients presenting with the clinical features of T2DM harbor circulating autoantibodies against pancreatic beta-cell antigens, most notably glutamic acid decarboxylase-65 (GAD-65).[3] This condition, termed Latent Autoimmune Diabetes in Adults (LADA), represents the most prevalent form of adult-onset autoimmune diabetes and may account for a substantial proportion of patients initially classified as T2DM, particularly in resource-limited settings where differentiation is seldom pursued.[4–6]

The biochemical and metabolic phenotype of LADA

Article Access
Website: www.wjmbs.site
10.5281/zenodo.20816069

How to cite this article
Ekhator NP, Ehondor OT. Biochemical and Metabolic Profiles in Gad-Positive Versus Gad-Negative Type 2 Diabetes Mellitus Patients in Benin City, Nigeria. West J Med & Biomed Sci. 2026;7(2):297-305. DOI:10.5281/zenodo.20816069

and GAD-positive diabetes is of considerable clinical importance, as GAD-positive patients have been reported to exhibit greater degrees of beta-cell loss, manifested as lower C-peptide concentrations and a tendency toward insulinopenia, compared to their GAD-negative counterparts.[7] They also tend to have poorer long-term glycemic control and faster progression to insulin dependence, although this trajectory may be modified by GAD antibody titer.[8] The lipid profile of GAD-positive patients has been less consistently characterized, with some studies suggesting differences in triglyceride and high density lipoprotein- cholesterol (HDL-cholesterol) levels reflecting pathophysiological distinctions from classical insulin-resistant T2DM.[9]

In sub-Saharan Africa, including Nigeria, the burden of T2DM is rapidly increasing, yet systematic screening for autoimmune diabetes remains largely absent from routine clinical practice.[10,11] Misclassification of LADA as T2DM may have significant implications for treatment outcomes, particularly with respect to delays in insulin initiation.[12] Data on the biochemical correlates of GAD positivity among Nigerian T2DM patients are few, and comparative metabolic profiling of GAD-positive and GAD-negative patients has not been well described in this setting.

This study aimed to describe and compare biochemical and metabolic profiles, including glycemic indices, C-peptide levels, lipid parameters, and microalbuminuria, between GAD-positive and GAD-negative T2DM patients attending a Nigerian tertiary hospital

MATERIALS AND METHODS

Study Area

This study was conducted at the Diabetes Clinic of the University of Benin Teaching Hospital (UBTH), a 900-bed federal tertiary institution in Benin City, Edo State, South-South Nigeria. The clinic operates twice weekly, with an average attendance of approximately 130 patients per clinic day, and serves as a tertiary referral centre for Edo, Delta, Ondo, Ekiti, Kogi, and Bayelsa States.

Study Design, Sampling Technique, and Data Collection

A descriptive cross-sectional study was conducted among T2DM patients attending the Diabetes Clinic at UBTH. Consecutive sampling was employed over six months from April to September 2015. A researcher-administered questionnaire collected sociodemographic and clinical data. Fasting blood samples were collected from all participants following an overnight fast of at least 12 hours for assays of Anti-GAD 65 autoantibodies, C- Peptide, glycated haemoglobin (HbA1C), fasting plasma glucose, fasting serum lipid profile. Also, a morning mid-stream urine was collected for urinalysis using Micral test strips (Roche diagnostics) to check for microalbuminuria (which is a semi quantitative test for microalbuminuria).

Definition of study criteria

Latent Autoimmune Diabetes in Adults was diagnosed in an individual previously diagnosed with T2DM at ≥ 30 years of age, with positivity for anti-GAD autoantibodies and who required insulin for glycaemic control within 6 months of diagnosis.

Normal fasting blood glucose <110mg/dl (6.1mmol/l).

Type 2 DM: persons with hyperglycaemia diagnosed by a fasting plasma glucose ≥ 126mg/dl (7.0mmol/l) or 2 hours post glucose load ≥ 200mg/dl (11.1mmol/l) on a single occasion in the presence of classic symptoms of diabetes mellitus or confirmed on a repeat occasion in the absence of symptoms.

Hypertension: individuals with raised arterial pressure ≥ 140/90mmHg or patients on antihypertensive medication were deemed to have hypertension.

Glycated haemoglobin (HbA1c) was considered optimal if HbA1c < 7%.

GAD levels ≥ 0.62ng/ml were deemed to be positive.

C –peptide levels lower than 0.3ng/ml were deemed to be indicative of insulinopaenia.

Lipid abnormalities were defined as biochemical evidence of elevated values of:

Total Cholesterol ≥ 200mg/dl

HDL-cholesterol for men ≤ 40mg/dl and women ≤ 50mg/dl

Low density lipoprotein cholesterol (LDL-c)

≥100mg/dl

Triglycerides ≥ 150mg/dl, either singly or in combination.

Study Population, Selection Criteria, and Sample Size

A total of 240 consenting adults with previously diagnosed T2DM, aged 30 years and above at diagnosis, were recruited. Participants met the World Health Organization (WHO) 1999 criteria for T2DM diagnosis and provided informed consent. Patients with type 1 diabetes, other specific forms of diabetes, pregnant women, patients on steroids or managed for malignancies, and non-consenting individuals were excluded. The minimum sample size was 185 (Fisher's formula, 14% LADA prevalence); expanded to 240 with a 10% attrition allowance for improved validity.

Data Analysis

Data were analyzed using IBM SPSS version 25. Continuous variables were expressed as means with standard deviations, or medians with interquartile ranges for skewed data. Categorical variables were expressed as frequencies and percentages. Student t-test and Mann-Whitney U test compared continuous parameters between GAD groups; chi-square tests compared proportions. Pearson correlation assessed relationships between continuous biochemical parameters and GAD levels. A p-value of 0.05 or less was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the Ethics and Research Committee of the University of Benin Teaching Hospital. Written informed consent was obtained from all participants prior to enrolment.

RESULTS

Biochemical Characteristics of the Study Population

A total of 240 patients with T2DM were studied. Among the 240 respondents, 67 (27.9%) were GAD positive, while 173 (72.1%) were GAD negative. The mean (SD) GAD titer was 0.56 (0.22) ng/dl, mean (SD) C-peptide was 1.01 (0.32) ng/ml, and mean (SD) HbA1c was 9.76 (2.25)%. Mean (SD) fasting blood glucose was 132.90 (58.96) mg/dl. Mean (SD) values for total cholesterol, triglycerides, high density lipoprotein-cholesterol (HDL-cholesterol), and LDL-cholesterol were 163.35 (54.24) mg/dl, 125.92 (77.62) mg/dl, 35.86 (15.44) mg/dl, and 110.07 (6.05) mg/dl, respectively. Elevated cholesterol was recorded in 45 (18.7%) respondents; elevated triglycerides in 69 (28.7%); reduced HDL-cholesterol in 54 (22.5%); and LDL-cholesterol at or above 100 mg/dl in nearly half, 119 (49.6%). Insulinopenia was recorded in 65 (27.1%) respondents and hyperglycemia in 110 (45.8%). A large proportion, 208 (86.7%), had elevated HbA1c at or above 7%. (Table 1, Figure 1)

Clinical History Parameters and GAD Status

The mean (SD) age at diabetes diagnosis was 49.5 (13.8) years overall; GAD-positive patients had a slightly higher mean age at diagnosis of 51.7 (12.1) years compared to 48.6 (14.4) years in GAD-negative patients, though not statistically significant (p=0.121). Oral glucose lowering agents (OGLAs) were the most common treatment in both groups, used by 80.6% (n=54) of GAD-positive and 72.3% (n=125) of GAD-negative patients. Insulin alone was used by 6 (9.0%) of GAD-positive and 12 (6.9%) of GAD-negative respondents. A combination of insulin and OGLAs was used by 6 (9.0%) of GAD-positive compared to 32 (18.5%) of GAD-negative patients. No statistically significant association was found between medication regimen and GAD status (p=0.320). The mean (SD) interval from diagnosis to insulin commencement was 1.4 (0.9) years in GAD-positive and 1.7 (1.8) years in GAD-negative patients (p=0.095). The median (IQR) duration of diabetes was 7.0 (7.0) years and 7.0 (11.5) years in GAD-positive and GAD-negative patients respectively (p=0.490). (Table 2)

Comparison of Mean Biochemical Parameters Between GAD-Positive and GAD-Negative Patients

The mean (SD) GAD titer was 0.82 (0.24) ng/dl in GAD-positive and 0.46 (0.09) ng/dl in GAD-negative patients (p<0.001). Median (IQR) C-peptide was significantly lower in GAD-positive patients at 0.40 (0.80) ng/ml compared to 0.60 (1.00) ng/ml in GAD-negative patients (p=0.050), reflecting greater beta-cell dysfunction. Mean

HbA1c was comparable: 9.68 (2.51)% in GAD-positive and 9.79 (2.53)% in GAD-negative patients (p=0.766). Fasting blood glucose was similarly equivalent (p=0.945). Triglyceride levels were significantly lower in GAD-positive patients at 143.00 (92.52) mg/dl compared to 188.95 (70.08) mg/dl in GAD-negative patients (p=0.025). Total cholesterol, HDL-cholesterol, and LDL-cholesterol did not differ significantly (all p>0.05). (Table 3, Figure 2, Figure 3, Figure 4)

Relationship Between Categorical Biochemical Parameters and GAD Status

Abnormal cholesterol was found in 19.4% of GAD-positive and 18.5% of GAD-negative patients (p=0.872). Elevated triglycerides were seen in 34.3% of GAD-positive versus 26.6% of GAD-negative respondents, though categorical analysis did not reach significance (p=0.235). Normal HDL-cholesterol was present in 79.1% of GAD-positive and 76.9% of GAD-negative patients (p=0.711). LDL-cholesterol at or above 100 mg/dl was seen in 52.2% of GAD-positive and 48.6% of GAD-negative patients (p=0.609). Insulinopenia was more prevalent in GAD-positive respondents (35.8% vs. 23.7%), approaching but not reaching significance (p=0.058). Hyperglycemia was present in 49.3% of GAD-positive and 44.5% of GAD-negative respondents (p=0.508). Elevated HbA1c was present in 85.1% of GAD-positive and 87.3% of GAD-negative patients (p=0.652). Microalbuminuria was present in exactly half the population, with 46.3% of GAD-positive and 51.4% of GAD-negative respondents affected (p=0.472). (Table 4, Figure 5)

Correlation of Biochemical Parameters with GAD Levels

C-peptide showed a weak negative, non-significant correlation with GAD (r=-0.12, p=0.058). HbA1c and fasting blood glucose similarly demonstrated weak negative non-significant correlations (r=-0.03, p=0.625 and r=-0.02, p=0.750 respectively). Total cholesterol and triglycerides showed weak positive non-significant correlations (r=0.09 each), while HDL-cholesterol and LDL-cholesterol showed weak negative non-significant correlations (r=-0.03 and r=-0.04 respectively). (Table 5)

Table 1: Biochemical Characteristics of Respondents

Variables	Frequency (n=240)	Percentage (%)
Cholesterol		
<200 mg/dl	195	81.3
≥200 mg/dl	45	18.7
Triglyceride		
<150 mg/dl	171	71.3
≥150 mg/dl	69	28.7
HDL - Cholesterol		
Normal	186	77.5
Decreased	54	22.5
LDL - Cholesterol		
<100 mg/dl	121	50.4
≥100 mg/dl	119	49.6
C - Peptide		
Normal	175	72.9
Insulinopaenia	65	27.1
Fasting Blood Glucose		
Normal	130	54.2
Hyperglycaemia	110	45.8
HbA1c		
<7%	32	13.3
≥7%	208	86.7

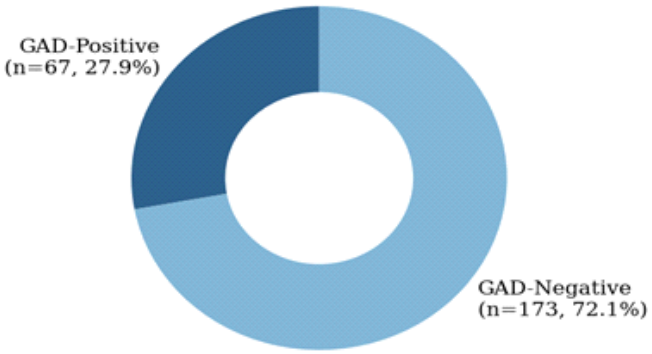

Figure 1: Prevalence of GAD-65 Positivity Among T2DM Patients (N=240)

Table 2: Clinical History of Respondents by GAD Status

Variable	GAD <0.62 (n=173)	GAD ≥0.62 (n=67)	Total (N=240)	Statistic	p
Mean Age at Diagnosis (years)	48.6 (14.4)	51.7 (12.1)	49.5 (13.8)	t=−1.56	0.121
Current Medication				X^2=3.51	0.320
OGLA	125 (72.3)	54 (80.6)	179 (74.6)		
Insulin	12 (6.9)	6 (9.0)	18 (7.5)		
Diet	2 (1.2)	1 (1.5)	3 (1.3)		
Insulin/OGLA	32 (18.5)	6 (9.0)	38 (15.8)		
Mean Interval to Insulin (years)	1.7 (1.8)	1.4 (0.9)	1.6 (1.1)	t=1.68	0.095
Duration of DM (years, median IQR)	7.0 (11.5)	7.0 (7.0)	7.0 (10.0)	MWU	0.490

OGLA: Oral Glucose-Lowering Agent; MWU: Mann-Whitney U test

Table 3: Comparison of Mean Biochemical Parameters Between GAD Positive and GAD Negative Patients

Parameter	GAD <0.62 n=173 Mean (SD)	GAD ≥0.62 n=67 Mean (SD)	Total n=240 Mean (SD)	t	p
GAD (ng/dl)	0.46 (0.09)	0.82 (0.24)	0.56 (0.22)	17.14	<0.001*
C-Peptide (ng/ml)**	0.60 (1.00)	0.40 (0.80)	0.50 (0.90)	—	0.050*
HbA1c (%)	9.79 (2.53)	9.68 (2.51)	9.76 (2.25)	0.30	0.766
Fasting Blood Glucose (mg/dl)	133.06 (60.80)	132.48 (54.36)	132.90 (58.96)	0.07	0.945
Total Cholesterol (mg/dl)	161.69 (50.59)	167.63 (62.90)	163.35 (54.24)	0.76	0.448
Triglycerides (mg/dl)	188.95 (70.08)	143.00 (92.52)	125.92 (77.62)	2.26	0.025*
HDL-Cholesterol (mg/dl)**	30.3 (22.2)	31.5 (14.6)	31.0 (21.6)	—	0.993
LDL-Cholesterol (mg/dl)	113.13 (8.02)	102.17 (5.28)	110.07 (6.05)	0.81	0.417

***Median (IQR); Mann -Whitney U test applied. * p ≤ 0.05*

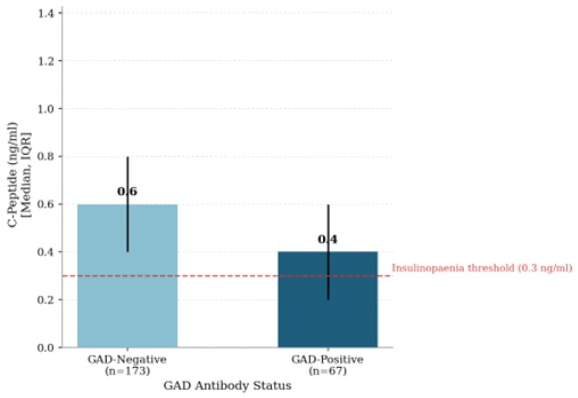

Figure 2: C-Peptide Levels by GAD Status (Median, IQR; p = 0.050)

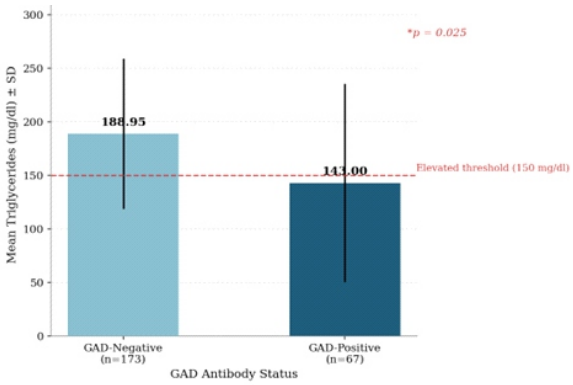

Figure 3: Serum Triglyceride Levels by GAD Status (Mean ± SD; p = 0.025*)

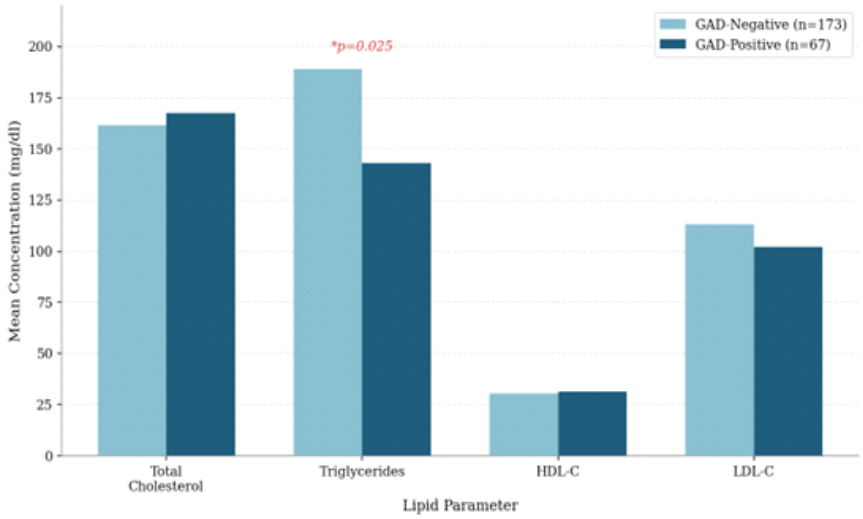

Figure 4: Full Lipid Profile Comparison by GAD Status (* Triglycerides p = 0.025)

Table 4: Relationship Between Categorical Biochemical Parameters and GAD Status

Biochemical Parameter	GAD <0.62 (n=173)	GAD ≥0.62 (n=67)	Total (N=240)	X^2	p
Cholesterol				0.03	0.872
<200 mg/dl	141 (81.5)	54 (80.6)	195 (81.3)		
≥200 mg/dl	32 (18.5)	13 (19.4)	45 (18.7)		
Triglyceride				1.41	0.235
<150 mg/dl	127 (73.4)	44 (65.7)	171 (71.3)		
≥150 mg/dl	46 (26.6)	23 (34.3)	69 (28.7)		
HDL-Cholesterol				0.14	0.711
Normal	133 (76.9)	53 (79.1)	186 (77.5)		
Decreased	40 (23.1)	14 (20.9)	54 (22.5)		
LDL-Cholesterol				0.26	0.609
<100 mg/dl	89 (51.4)	32 (47.8)	121 (50.4)		
≥100 mg/dl	84 (48.6)	35 (52.2)	119 (49.6)		
C-Peptide				3.59	0.058
Normal	132 (76.3)	43 (64.2)	175 (72.9)		
Insulinopenia	41 (23.7)	24 (35.8)	65 (27.1)		
Fasting Blood Glucose				0.44	0.508
Normal	96 (55.5)	34 (50.7)	130 (54.2)		
Hyperglycemia	77 (44.5)	33 (49.3)	110 (45.8)		
HbA1c				0.20	0.652
<7%	22 (12.7)	10 (14.9)	32 (13.3)		
≥7%	151 (87.3)	57 (85.1)	208 (86.7)		
Microalbuminuria				0.52	0.472
Present	89 (51.4)	31 (46.3)	120 (50.0)		
Absent	84 (48.6)	36 (53.7)	120 (50.0)		

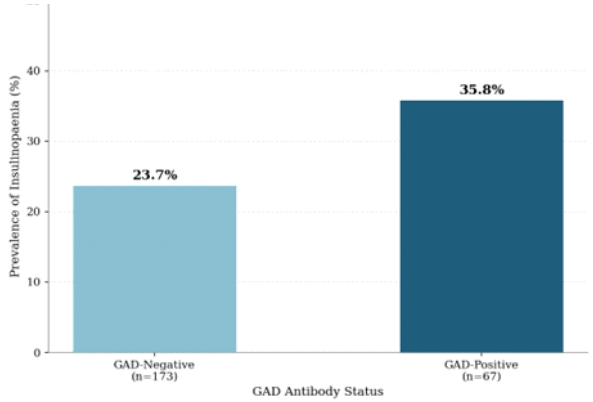

Figure 5: Prevalence of Insulinopenia by GAD Status (p = 0.058)

Table 5: Correlation of Biochemical Parameters with GAD Levels (n=240)

Biochemical Parameter	r-value	p-value
C-Peptide	-0.12	0.058
HbA1c	-0.03	0.625
Fasting Blood Glucose	-0.02	0.750
Total Cholesterol	0.09	0.154
Triglycerides	0.09	0.134
HDL-Cholesterol	-0.03	0.703
LDL-Cholesterol	-0.04	0.550

DISCUSSION

This study compared the biochemical and metabolic profiles of GAD-positive and GAD-negative T2DM patients at a Nigerian tertiary hospital. The central findings were that GAD-positive patients had significantly lower C-peptide levels and significantly lower serum triglyceride concentrations compared to their GAD-negative counterparts, while no significant differences were found in glycemic indices, other lipid parameters, or microalbuminuria. These findings are consistent with an autoimmune diabetic phenotype characterized by progressive beta-cell loss rather than the classical insulin resistance and dyslipidaemia seen in T2DM.

The finding of lower C-peptide levels in GAD-positive patients, with a median of 0.40 (IQR 0.80) ng/ml compared to 0.60 (IQR 1.00) ng/ml in GAD-negative patients (p=0.050), reflects the established pathophysiology of autoimmune diabetes, in which ongoing T-cell-mediated destruction of pancreatic beta cells leads to progressive reduction in endogenous insulin secretory capacity.[13] Fan et al.[8], in a comparative study of GAD antibody prevalence and C-peptide among over 5,000 Chinese patients, demonstrated that GAD-positive patients had markedly reduced C-peptide responses to glucagon stimulation, confirming reduced beta-cell reserve as a hallmark of this subtype. A strength of the cohort was its prospective design and standardized stimulated C-peptide testing; however, the predominantly Asian population limits direct comparability with the Nigerian setting. The borderline significance of the C-peptide association in the current study may reflect the mixed GAD titer range, as low-titer GAD positivity is associated with slower beta-cell decline. Routine C-peptide measurement alongside GAD antibody testing in Nigerian diabetic patients would help stratify patients by residual beta-cell function and guide insulin initiation decisions.

The observation of insulinopenia in 35.8% of GAD-positive patients compared to 23.7% of GAD-negative patients, though not reaching significance on categorical analysis, further supports the trend toward greater beta-cell insufficiency in the autoimmune subgroup. Hwangbo et al.[14] in South Korea, and Muazu et al.[15] in Northern Nigeria, similarly reported that GAD-positive patients had lower fasting C-peptide and a higher prevalence of insulinopenia at diagnosis compared to GAD-negative controls.

The significantly lower mean triglyceride levels in GAD-positive patients is a metabolically important finding, as classical T2DM is strongly associated with hypertriglyceridemia as a component of the metabolic syndrome, driven by peripheral insulin resistance and consequent increased hepatic VLDL production.[16,17] The lower triglyceride levels in GAD-positive patients suggest a fundamentally different metabolic issue, one characterized more by insulin deficiency than insulin resistance, thereby weakening the hypertriglyceridemia phenotype. Ipadeola et al.[18] in a cross-sectional study carried out in Oyo, Nigeria, reported reduced BMI and waist circumference in GAD-positive compared to GAD-negative diabetic patients, while Adeleye et al.[19] in Lagos, Nigeria reported significantly lower lipid levels in GAD-positive cases when compared with GAD-negative cases, consistent with an autoimmune rather than an insulin-resistant metabolic phenotype. These results suggest that cardiovascular risk stratification in Nigerian T2DM patients may need to be contextualized by GAD status.

The absence of a significant difference in HbA1c between GAD-positive (9.68%) and GAD-negative (9.79%) patients may be because groups in this study had very poor glycemic control, with over 86% of respondents having HbA1c above 7%, highlighting a systemic challenge in diabetes management in the Nigerian healthcare context, irrespective of GAD status.

Microalbuminuria was present in half the study population and did not differ significantly between GAD-positive and GAD-negative groups, similar to

Mottl et al.[20] in a United States-based longitudinal study of over 2,000 diabetic children, which found that autoimmunity status was not associated with albuminuria. The high overall prevalence of microalbuminuria highlights the importance of renal protection strategies across all T2DM subtypes in Nigeria.

Taken together, the biochemical profile of GAD-positive patients in this study, characterized by reduced C-peptide, lower triglycerides, and comparable glycemic indices to GAD-negative patients, is consistent with an autoimmune diabetic phenotype metabolically distinct from classical insulin-resistant T2DM. GAD antibody testing and C-peptide measurement should be considered in Nigerian T2DM patients who are not obese, have unexpectedly low triglyceride levels, or demonstrate progressive glycemic failure despite adequate oral therapy.

CONCLUSION

GAD-positive T2DM patients at a Nigerian tertiary hospital demonstrated significantly lower C-peptide levels and serum triglyceride concentrations compared to GAD-negative counterparts, consistent with an autoimmune diabetic phenotype. No significant differences were however observed in glycemic control, other lipid parameters, or microalbuminuria.

Recommendation

These findings support the inclusion of GAD antibody and C-peptide screening in the routine evaluation of T2DM patients in Nigeria to facilitate the timely identification of autoimmune diabetes and optimize clinical management.

Limitations

Use of a single GAD threshold without titre stratification may have obscured titer-dependent biochemical variation. Single-centre consecutive sampling may limit generalizability to the broader Nigerian T2DM population.

Declarations

Conflict of Interest: The authors declare no conflict of interest.

Funding: No external funding was received for this study.

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