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ORIGINAL RESEARCH

Clinical Characteristics and Related Risk Factors of Thyroid Dysfunction in LADA Patients: A Single-Center Cross-Sectional Study in Xuzhou, Jiangsu, China

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Objective: The aim of our study was to further explore the clinical characteristics and related risk factors of thyroid dysfunction in latent autoimmune diabetes in adults (LADA) patients in the Xuzhou region of Jiangsu, China, to facilitate early intervention and treatment.

Methods: It is conducted a single-center, cross-sectional study involving 95 hospitalized LADA patients from the Affiliated Hospital of Xuzhou Medical University between January 2024 and April 2025. The patients were divided into two groups based on the presence of thyroid dysfunction: 39 LADA patients without thyroid dysfunction and 56 LADA patients with thyroid dysfunction. Data collection included clinical indicators and thyroid function assessments. Correlation analysis and univariate binary logistic regression analysis were performed on the study data.

Results: Significant differences were observed between the two groups in urinary albumin-to-creatinine ratio (UACR), fasting C-peptide (FCP), postprandial 2-hour C-peptide (P2hCP), free triiodothyronine (FT3), free thyroxine (FT4), thyroid peroxidase antibody (TPOAb), thyroglobulin antibody (TGAb), thyrotropin receptor antibody (TRAb), and insulin autoantibody (IAA). Correlation analysis revealed that FCP was positively correlated with P2hCP and FT3 but negatively correlated with IAA. FT3 was positively correlated with P2hCP and FT4. TPOAb was positively correlated with TGAb and TRAb. Univariate binary logistic regression analysis indicated that low levels of FCP, P2hCP, and FT3, as well as high levels of TPOAb and TGAb, were associated with thyroid dysfunction in LADA patients.

Conclusion: Lower FCP, P2hCP, and FT3 levels, along with higher TPOAb and TGAb levels, are associated with thyroid dysfunction in LADA patients. Therefore, it is recommended that LADA patients undergo screening for thyroid antibodies and FT3 at disease onset and every 1–2 years thereafter to minimize the risk of undiagnosed thyroid dysfunction. Additionally, reassessment of pancreatic function every 3–6 months is advised to optimize the timing of insulin therapy.

Keywords: clinical characteristics, risk factors, thyroid dysfunction, latent autoimmune diabetes in adults, LADA

Introduction

Diabetes mellitus is primarily classified into type 1 diabetes (T1D) and type 2 diabetes (T2D). However, some patients exhibit clinical features intermediate between T1D and T2D, a condition termed latent autoimmune diabetes in adults (LADA), also known as type 1.5 diabetes. LADA is a slowly progressive autoimmune diabetes characterized by the presence of autoantibodies, with glutamic acid decarboxylase 65 antibodies (GAD65-Ab) being the most significant

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immunological marker. GAD65-Ab may coexist with other pancreatic autoantibodies, such as insulin autoantibodies (IAA), which serve as early indicators of pancreatic β-cell dysfunction and play a crucial role in the pathogenesis of LADA.[1]

Patients with one autoimmune disorder are at an increased risk of developing another. LADA has been associated with other autoimmune diseases, including Hashimoto's thyroiditis, Sjögren's syndrome, and dermatomyositis, which share common autoimmune components and genetic susceptibility loci, such as human leukocyte antigen DR (HLA-DR), cytotoxic T-lymphocyte-associated protein 4(CTLA-4), CD25, protein tyrosine phosphatase non-receptor 22(PTPN22), and forkhead box P3 (FOXP3) genes.[2–6] Recent studies suggest that specific variants in these genes, such as those in PTPN22, may determine the onset of distinct diseases with different phenotypic and clinical characteristics.[7–9] These findings, both clinically and mechanistically, support the association between autoimmune thyroid diseases and LADA. However, the clinical characteristics of thyroid dysfunction in LADA patients from the Xuzhou region of Jiangsu, China, remain unclear. Therefore, the objective of this study is to investigate the clinical features and risk factors associated with thyroid dysfunction in LADA patients in Xuzhou, Jiangsu, to facilitate early intervention and treatment.

Materials and Methods

We conducted a single-center, cross-sectional study involving 95 hospitalized LADA patients from the Affiliated Hospital of Xuzhou Medical University between January 2024 and April 2025. The patients were divided into two groups based on the presence or absence of thyroid dysfunction: 39 LADA patients without thyroid dysfunction and 56 LADA patients with thyroid dysfunction.

Inclusion Criteria

1. Diagnosis of LADA (based on WHO criteria and the latest guidelines).
2. No recent use of medications affecting thyroid function.

Exclusion Criteria

1. Known history of thyroid disease.
2. Pregnancy or severe hepatic/renal dysfunction.
3. Other types of diabetes.

Data collection included clinical parameters and thyroid function assessment. The following variables were recorded: age, disease duration (Duration), systolic blood pressure (SBP), diastolic blood pressure (DBP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum creatinine (Scr), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), uric acid (UA), fasting blood glucose (FBG), glycated hemoglobin (HbA1c), GAD65-Ab (GADA), urinary albumin-to-creatinine ratio (UACR), triglyceride-glucose index (TyG), TyG-body mass index (TyG-BMI), estimated glomerular filtration rate (eGFR), fasting C-peptide (FCP), postprandial 2-hour C-peptide (P2hCP), free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH), thyroid peroxidase antibodies (TPOAb), thyroglobulin antibodies (TGAb), thyrotropin receptor antibodies (TRAb), IAA, 25-hydroxyvitamin D [25(OH)D], proinsulin (PI), and calcitonin (CT). Blood samples were stored at −80°C. Thyroid function was evaluated by measuring FT3, FT4, TSH, TPOAb, TGAb, and TRAb levels using chemiluminescence assays.

Statistical analysis was performed using SPSS 24.0. Normally distributed data were expressed as mean ± standard deviation and compared using independent samples t-tests. Non-normally distributed data were expressed as median (interquartile range) and analyzed using the Mann–Whitney *U*-test. To explore correlations between variables, Pearson's correlation analysis was performed for features with $P < 0.05$, and a heatmap of correlation coefficients was generated. Univariate logistic regression analysis was also conducted. A P-value < 0.05 was considered statistically significant.

All participants provided written informed consent, and the study was approved by the Ethics Committee of the Affiliated Hospital of Xuzhou Medical University. The study complies with the Declaration of Helsinki.

Results

Baseline characteristics (Table 1) revealed significant differences between the two groups in UACR, FCP, P2hCP, FT3, FT4, TPOAb, TGAb, TRAb, and IAA (all $P < 0.05$). Compared to LADA patients without thyroid dysfunction, those with thyroid dysfunction exhibited higher UACR, lower FCP and P2hCP levels, reduced FT3 and FT4, significantly elevated TPOAb and TGAb, and increased TRAb and IAA levels.

Subsequently, to explore inter-feature correlations, we performed Pearson correlation tests on variables with $P < 0.05$, calculating correlation coefficients between variables and generating Pearson correlation heatmaps. We investigated associations among UACR, FCP, P2hCP, FT3, FT4, TPOAb, TGAb, TRAb, and IAA in LADA patients. The correlation analysis revealed that FCP showed positive correlations with P2hCP and FT3, while demonstrating a negative correlation

Table 1 Comparison of Baseline Characteristics Between LADA Patients without Thyroid Dysfunction and LADA Patients with Thyroid Dysfunction

Clinical Characteristics	LADA Patients without Thyroid Dysfunction (n=39)	LADA Patients with Thyroid Dysfunction (n=56)	P
Age (years)	44.03±17.42	46.32±19.55	0.280
Duration (years)	2 (1, 6)	3 (1, 10)	0.720
SBP (mmHg)	125.54±17.82	123.88±19.18	0.427
DBP (mmHg)	81.90±13.42	79.29±14.21	0.531
ALT (U/L)	18.00 (11.50, 22.50)	14.50 (10.00, 20.50)	0.118
AST (U/L)	18.00 (15.00, 24.00)	17.00(13.00, 22.25)	0.184
Scr (umol/L)	48.00 (45.00, 59.50)	47.50 (40.75, 55.00)	0.215
TC (mmol/L)	5.04±1.45	5.07±1.40	0.928
TG (mmol/L)	1.11 (0.82, 1.63)	1.05 (0.73, 1.47)	0.539
HDL-C (mmol/L)	1.03 (0.91, 1.37)	1.15 (0.96, 1.46)	0.167
LDL-C (mmol/L)	2.87±0.98	2.79±0.91	0.555
UA (umol/L)	286.79±110.74	276.45±154.91	0.869
FBG (mmol/L)	10.39±5.84	10.94±5.07	0.643
HbA1c (%)	9.46±2.92	10.43±2.42	0.138
GADA (IU/mL)	60.60 (45.25, 269.50)	132.50 (45.52, 280.00)	0.208
UACR(mg/mmol)	0.57 (0.41, 1.36)	1.12 (0.59, 2.08)	0.026
TyG index	5.00(3.00, 9.00)	5.00 (3.00, 9.25)	0.810
TyG-BMI	120.00 (67.50, 207.50)	114.50 (77.25, 223.75)	0.981
eGFR(mL/min)	120 (120, 120)	120(120, 120)	0.604
FCP (ng/mL)	0.66 (0.24, 1.33)	0.3 (0.06, 0.79)	0.028
P2hCP (ng/mL)	1.04 (0.41, 3.16)	0.46 (0.17, 1.08)	0.009
FT3(pmol/L)	4.38±0.82	3.54±1.37	0.006
FT4(pmol/L)	17.34±2.41	17.20±4.66	0.006
TSH(mIU/L)	1.65 (1.24, 2.64)	1.90 (1.21, 3.31)	0.525
TPOAb(IU/ML)	2.83 (1.63, 12.89)	249.96 (3.15, 811.5)	0.000
TGAb(IU/ML)	26.30 (6.50, 65.60)	82.75 (12.85, 172.16)	0.001
TRAb(IU/L)	0.25 (0.25, 0.25)	0.25 (0.25, 0.43)	0.005
IAA (IU/mL)	6.54 (5.52, 8.33)	9.44 (6.41, 22.05)	0.003
25(OH)D(ng/mL)	16.60 (12.95, 18.50)	15.05 (12.97, 19.92)	0.773
PI(pg/mL)	74.63 (39.0, 131.94)	52.03 (18.75, 116.25)	0.152
CT(pg/mL)	2.05 (2.00, 2.33)	2.00 (2.00, 2.48)	0.578

Abbreviations: Duration, sisease duration; SBP, systolic blood pressure; DBP, diastolic blood pressure; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Scr, serum creatinine; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; UA, uric acid; FBG, fasting blood glucose; HbA1c, glycated hemoglobin; GADA, GAD65-Ab; UACR, urinary albumin-to-creatinine ratio; TyG, triglyceride-glucose index; TyG-BMI, TyG-body mass index; eGFR, estimated glomerular filtration rate; FCP, fasting C-peptide; P2hCP, postprandial 2-hour C-peptide; FT3, free triiodothyronine; FT4, free thyroxine; TSH, thyroid-stimulating hormone; TPOAb, thyroid peroxidase antibodies; TGAb, thyroglobulin antibodies; TRAb, thyrotropin receptor antibodies; IAA, insulin autoantibodies; 25 (OH)D, 25-hydroxyvitamin D ; PI, proinsulin; CT, calcitonin.

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with IAA. FT3 was positively correlated with P2hCP and FT4. Additionally, TPOAb exhibited positive correlations with both TGAb and TRAb (see Table 2 and Figure 1).

Finally, univariate binary logistic regression analysis (Table 3) revealed that lower FCP, P2hCP, and FT3 levels, as well as higher TPOAb and TGAb levels, were associated with the occurrence of thyroid dysfunction in LADA patients.

Discussion

Our findings demonstrate that LADA patients with thyroid dysfunction exhibit significantly higher UACR, lower FCP and P2hCP levels, decreased FT3 and FT4, along with markedly elevated TPOAb and TGAb, and increased TRAb and IAA levels. These results suggest that compared to LADA patients without thyroid dysfunction, those with concomitant thyroid abnormalities are more prone to diabetic nephropathy, exhibit more severe islet dysfunction, and have higher susceptibility to autoimmune thyroid diseases—particularly Hashimoto's thyroiditis—while also indicating greater disease severity and progression risk in LADA.

The observed association between thyroid dysfunction and LADA corroborates previous cross-sectional observations.[10–14] A 2025 Diabetologia study further elucidated this relationship mechanistically, revealing that MHC-driven immune responses—encompassing both innate and adaptive pathways—represent the primary shared biological pathway connecting LADA with Crohn's disease, ulcerative colitis, hypothyroidism, hyperthyroidism, and vitiligo. These pathways involve diverse immune cells (B cells, T cells, and natural killer cells) and molecules (cytokines, immunoglobulins, and interferons).[15]

Diabetes mellitus (DM) substantially increases chronic complication risks. Diabetic kidney disease (DKD), one of the most prevalent microvascular complications affecting up to 25% of DM patients, constitutes a leading cause of chronic kidney disease and end-stage renal disease,[16,17] imposing significant economic burdens and severely compromising quality of life. Thyroid hormones critically influence glucose metabolism, where both overt and subclinical thyroid dysfunction adversely affect disease control in DM patients, particularly in LADA. DKD diagnosis primarily relies on elevated urinary albumin-to-creatinine ratio (UACR) and reduced estimated glomerular filtration rate (eGFR). The 2025 Diabetologia study reported higher retinopathy incidence in LADA versus T2DM, corroborating prior findings.[15,18] Clinically, elevated UACR shows positive association with subclinical hypothyroidism (OR 3.51, 95% CI: 1.10–10.0),[19,20] aligning with our results. Mechanistically, thyroid hormones and DKD interact bidirectionally:

Table 2 Correlations Among UACR, FCP, P2hCP, FT3, FT4, TPOAb, TGAb, TRAb, and IAA in LADA Patients

		UACR	FCP	P2hCP	FT3	FT4	TPOAb	TGAb	TRAb	IAA
UACR	r	1	−0.022	−0.121	−0.117	0.090	−0.105	−0.078	−0.032	−0.073
	P		0.830	0.244	0.258	0.387	0.312	0.450	0.759	0.482
FCP	r	−0.022	1	0.654	0.236	0.094	−0.120	−0.071	0.138	−0.236
	P	0.830		0.000	0.021	0.367	0.245	0.492	0.183	0.021
P2hCP	r	−0.121	0.654	1	0.229	0.089	−0.164	−0.082	0.065	−0.192
	P	0.244	0.000		0.026	0.391	0.113	0.431	0.529	0.062
FT3	r	−0.117	0.236	0.229	1	0.535	0.025	−0.130	0.120	0.044
	P	0.258	0.021	0.026		0.000	0.810	0.208	0.247	0.670
FT4	r	0.090	0.094	0.089	0.535	1	0.013	−0.053	0.121	−0.141
	P	0.387	0.367	0.391	0.000		0.901	0.611	0.245	0.172
TPOAb	r	−0.105	−0.120	−0.164	0.025	0.013	1	0.558	0.311	0.095
	P	0.312	0.245	0.113	0.810	0.901		0.000	0.002	0.359
TGAb	r	−0.078	−0.071	−0.082	−0.130	−0.053	0.558	1	−0.032	0.033
	P	0.450	0.492	0.431	0.208	0.611	0.000		0.755	0.754
TRAb	r	−0.032	0.138	0.065	0.120	0.121	0.311	−0.032	1	0.028
	P	0.759	0.183	0.529	0.247	0.245	0.002	0.755		0.784
IAA	r	−0.073	−0.236	−0.192	0.044	−0.141	0.095	0.033	0.028	1
	P	0.482	0.021	0.062	0.670	0.172	0.359	0.754	0.784	

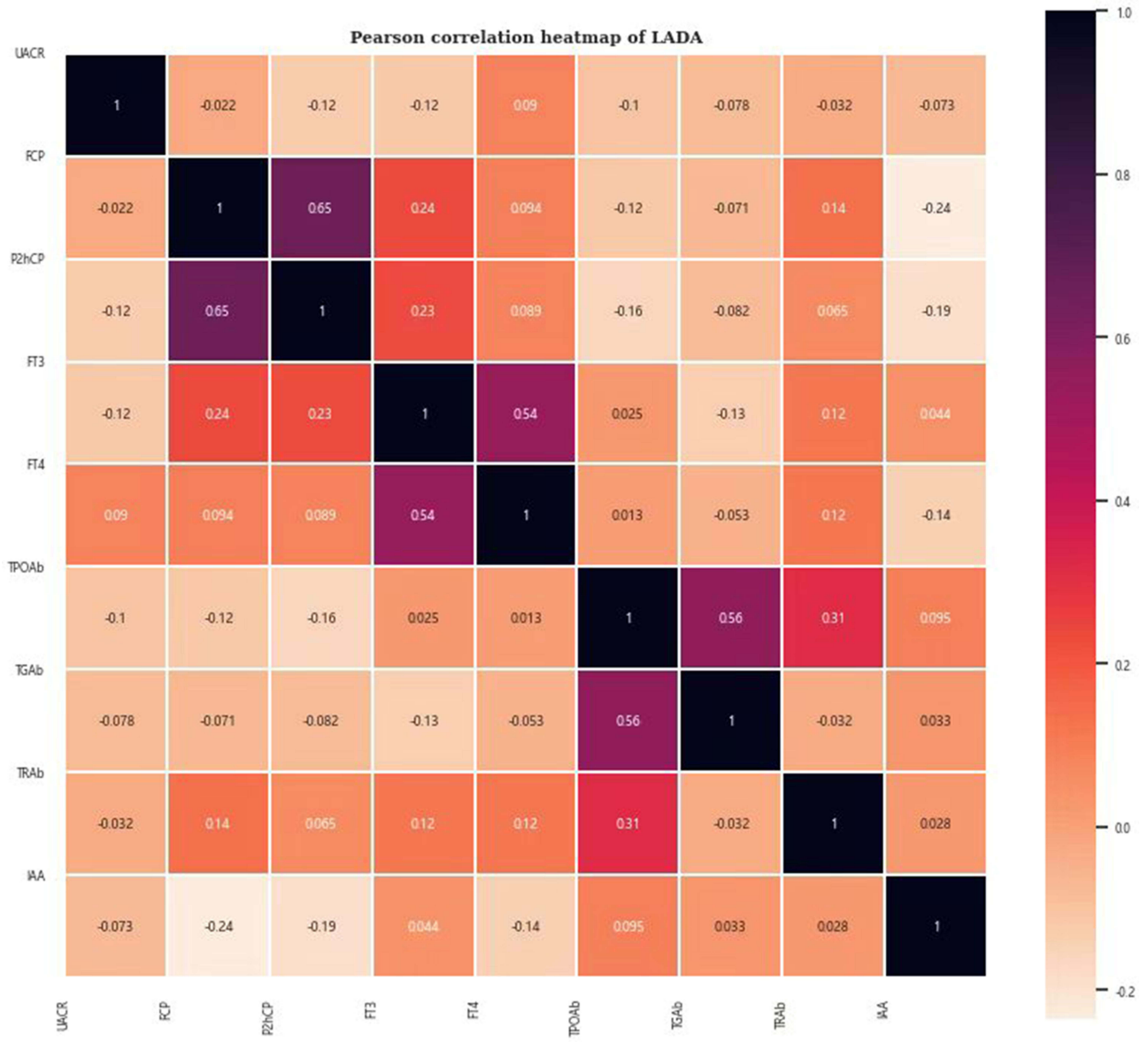

Figure 1 Heatmap of Pearson Correlation Coefficients Among UACR, FCP, P2hCP, FT3, FT4, TPOAb, TGAb, TRAb, and IAA in LADA Patients.

hyperglycemia-induced inflammation suppresses 5'-deiodinase activity, impairing peripheral T4-to-T3 conversion[21] and reducing FT3, thereby disrupting the hypothalamic-pituitary-thyroid axis.[22] Concurrently, glomerular structural damage in DKD promotes protein loss, further depressing FT3 and exacerbating hypothyroidism.[23,24] Thus, worsening DKD

Table 3 Univariate Binary Logistic Regression Analysis of Thyroid Dysfunction in LADA Patients

	ß	Std. Error	Wald	df	P-value	Exp(ß)
FCP	−0.710	0.278	6.529	1	0.011	0.491
P2hCP	−0.252	0.113	4.985	1	0.026	0.777
FT3	−0.619	0.205	9.090	1	0.003	0.538
TPOAb	0.006	0.002	10.914	1	0.001	1.006
TGAb	0.007	0.003	5.110	1	0.024	1.007

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severity progressively lowers FT3, intensifying thyroid dysfunction, which in turn accelerates DKD progression—creating a vicious cycle.

Correlation analyses revealed positive associations between FT3 and FCP/P2hCP/FT4. Clinically, severe LADA often manifests low T3 syndrome which is a thyroid hormone metabolism disorder caused by systemic non-thyroidal illnesses, where pronounced glucotoxicity exacerbates islet dysfunction—consistent with the proposed mechanism of hyperglycemia-induced 5'-deiodinase suppression impairing T3 conversion.[21,22] We also observed positive correlations among TPOAb, TGAb, and TRAb, explaining the frequent coexistence of autoimmune disorders (eg, Hashimoto's thyroiditis and Graves' disease) through shared immunopathogenic mechanisms.[15] Notably, the inverse FCP-IAA correlation aligns with 2024 Endocrine findings: elevated plasmablasts and Breg (B10) phenotypes in LADA positively correlate with islet cell antibodies and IAA but negatively with FCP, suggesting PB-mediated β-cell destruction[25]—mechanistically supporting our observations.

Univariate binary logistic regression confirmed that low FCP, P2hCP, and FT3, alongside high TPOAb and TGAb, predict thyroid dysfunction in LADA. Prior studies validate thyroid antibodies as key predictors.[26] Thus, we recommend: (1) annual/biennial thyroid antibody and FT3 screening from LADA onset to minimize undiagnosed thyroid dysfunction;[27] and (2) triannual/semiannual islet function assessments to optimize insulin initiation timing and tailor thyroid therapy, enabling early intervention to mitigate progression risks.

Limitations: First, single-center design and limited sample size may introduce bias, necessitating multicenter cohorts with refined subgroup analyses. Second, GADA assay specificity (98%) permits potential misclassification of T2DM as LADA, potentially attenuating observed thyroid dysfunction associations. Finally, the study selected a hospitalized population, which may introduce some selection bias and lacks a healthy control group. Therefore, further improvements are needed in future research.

Conclusion

In conclusion, our study identifies low FCP/P2hCP/FT3 and high TPOAb/TGAb as predictors of thyroid dysfunction in LADA, warranting regular thyroid and islet function monitoring for timely intervention.

Ethical Statement

Ethics approval and consent to participate Written informed consent for all data was obtained from patients during their hospitalization, and the Ethics Committee of the Affiliated Hospital of Xuzhou Medical University approved the study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no conflicts of interest in this work.

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