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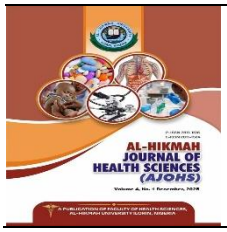

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Original Article

Alterations in Serum Prolactin and Progesterone Profiles among Women with Type 2 Diabetes Mellitus in Ilorin, Nigeria: Implications for Reproductive Endocrine Dysfunction

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ABSTRACT

Background: Emerging evidence suggests that reproductive hormones such as prolactin and progesterone may influence glucose metabolism and insulin sensitivity. However, data from sub-Saharan Africa, particularly Nigeria, remains limited. Thus, this study assesses the impact of Type 2 Diabetes Mellitus (T2DM) on serum prolactin and progesterone levels among female patients in Ilorin, Nigeria.

Methods: A cross-sectional case-control study was conducted among 90 participants recruited from General Hospital, Ilorin. Fasting plasma glucose (FPG) was estimated using the glucose oxidase-peroxidase method, while serum prolactin and progesterone levels were measured using enzyme-linked immunosorbent assay (ELISA). Data were analyzed using SPSS version 25. Statistical significance was set at $p < 0.05$.

Results: The mean FPG was significantly higher in T2DM patients (10.36 ± 4.51 mmol/L) compared to controls (4.63 ± 0.50 mmol/L; $p < 0.001$). Serum prolactin levels were significantly lower in T2DM patients (12.46 ± 7.57 ng/mL) compared to controls (18.03 ± 8.12 ng/mL; $p < 0.05$). Similarly, progesterone levels were significantly lower in T2DM patients (1.59 ± 0.33 ng/mL) compared to controls (1.91 ± 0.34 ng/mL; $p < 0.05$). However, FPG showed a non-significant negative correlation with prolactin and progesterone levels.

Conclusion: Female patients with T2DM had significantly lower prolactin and progesterone levels than healthy controls. These findings suggest a potential association between reproductive hormone dysregulation and T2DM pathophysiology in women.

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1. Introduction

Type 2 diabetes mellitus (T2DM) remains a major global public health challenge characterized by chronic hyperglycemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction, with prevalence and projections substantially higher than previously estimated. International Diabetes Federation (IDF) data show the global burden has grown significantly, with Saeedi *et al*. (2019) reporting 463 million adults with diabetes in 2019, projected to reach 700 million by 2045. More recent estimates indicate this trajectory is accelerating, with Sun *et al*. (2021) documenting 536.6 million people in 2021, rising to 783.2 million by 2045. Motala *et al*. (2022) describe that Sub-Saharan Africa (SSA) faces challenges from a rapid epidemiological transition driven by urbanization, with a notable report from Chilunga *et al.* (2023) that diabetes prevalence in SSA increased by 83% between 1990 and 2021, with projections of a 57% further increase by 2050. Goedecke *et al.* (2022) emphasize that SSA has the highest projected increase globally (129% by 2045), with distinct pathophysiological mechanisms in Black African populations.

Beyond classical metabolic disturbances, T2DM has been increasingly linked to endocrine dysfunction, including alterations in reproductive hormones. Prolactin is an emerging endocrine regulator of type 2 diabetes risk, with physiological levels conferring metabolic protection, whereas dysregulation increases disease susceptibility. Recent evidence demonstrates this relationship across multiple metabolic tissues. Wu *et al*. (2024) established that prolactin has evolved from a reproductive hormone to a key metabolic regulator, with deviations in prolactin levels, both elevated and reduced, associated with adverse glucose and lipid metabolism. Macotela *et al*. (2020) demonstrated that low circulating prolactin levels represent a risk factor for T2D, whereas higher physiological levels are metabolically beneficial. Zhang *et al*. (2022) found that decreasing prolactin quartiles were associated with increased future T2D risk in women with a history of gestational diabetes. Mechanistically, Park *et al*. (2011) showed that prolactin dosage determines whether it improves or impairs β-cell function and insulin sensitivity. Zaidalkilani *et al*. (2024) characterized prolactin's dual effects, noting that normal physiological levels are essential for insulin sensitivity and adipose tissue regulation, whereas dysregulation contributes to the pathogenesis of metabolic syndrome. These findings suggest prolactin warrants investigation as a therapeutic target in T2DM prevention and management.

Progesterone influences glucose metabolism through multiple mechanisms affecting insulin secretion, β-cell function, and insulin sensitivity, though evidence reveals complex and sometimes contradictory effects across different physiological contexts. Multiple studies demonstrate progesterone's multifaceted role in glucose homeostasis. Progesterone modulates insulin secretion and β-cell function (Mauvais-Jarvis, 2016; Handgraaf & Philippe, 2019), with progesterone receptor knockout mice showing improved glucose tolerance and increased β-cell proliferation. However, evidence of harmful effects also exists: progesterone induces β-cell apoptosis through oxidative stress mechanisms (Nunes *et al*., 2014) and antagonizes insulin effects on peripheral tissues (Sutter-Dub & Dazey, 1981). Additionally, progesterone impairs peripheral insulin sensitivity through post-receptor mechanisms in adipocytes (Sutter-Dub, 1986). These divergent findings, ranging from β-cell protective effects to apoptosis induction, underscore the inconsistency noted across populations and suggest progesterone's effects may be context-dependent, particularly during pregnancy when insulin demand increases (Brănişteanu & Mathieu, 2003; De Sa *et al.*, 2011).

Despite growing international evidence, limited data exist regarding prolactin and progesterone alterations among African women with T2DM. This study, therefore, aimed to evaluate serum prolactin and progesterone levels in female T2DM patients in Ilorin, Nigeria, and explore their relationship with glycemic status.

2. Materials and Methods

2.1 Study Design and Setting

This cross-sectional case-control study was conducted at Kwara State General Hospital, Ilorin, Nigeria.

2.2 Study Population

A total of 90 female participants, comprising 70 diagnosed T2DM patients and 20 apparently healthy age-matched controls, were recruited from General Hospital Ilorin, Kwara State, Nigeria.

Inclusion Criteria

i. Reproductive-aged women diagnosed with T2DM.
ii. Apparently healthy, non-diabetic, age-matched reproductive women as controls.

Exclusion Criteria

i. Pregnant women
ii. Patients with type 1 diabetes
iii. Women on hormonal contraceptives
iv. Individuals outside reproductive age were excluded.

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2.3 Ethical Approval

Ethical approval was obtained from the Kwara State Ministry of Health and the Management of General Hospital, Ilorin (reference numbers MOH/KS/EU/777/572 and GHI/ADM/134/VOL.II/391, respectively), in accordance with the principles of the World Medical Association's Declaration of Helsinki, 1964, revised in 2013 (Millum *et al*., 2013). Both written and verbal informed consent were obtained from all participants after a comprehensive explanation of the study's objectives, potential risks and benefits, and assurances regarding the confidentiality of their data.

2.4 Sample Collection and Laboratory Analysis

Following 10–12 hours of overnight fasting, 5 mL of venous blood was collected, with 2 mL dispensed into fluoride oxalate for fasting plasma glucose (FPG) and 3 mL into a gel-activator vacuum tube for serum prolactin and progesterone. Blood samples were spun at 3000 rpm for 5 minutes, and the plasma and serum were separated into plain tubes for quantitative analysis of FBG, serum prolactin and progesterone levels.

2.5 Laboratory Analysis

Quantitative Estimation of Glucose Using the Glucose Oxidase-Peroxidase Method (Trinder, 2002)

Principle

Glucose oxidase (GOD) catalyzes the oxidation of β-D-glucose present in the plasma to D-glucono-1,5-lactone with the formation of hydrogen peroxide; the lactone is then slowly hydrolyzed to gluconic acid. The hydrogen peroxide produced is then broken down to oxygen and water by a peroxidase enzyme. Oxygen reacts with oxygen acceptors such as orthotoluidine, which is converted to a colored compound, the amount of which can be measured colorimetrically.

Quantitative Estimation of Serum Progesterone and Prolactin Using the Enzyme-Linked Immunosorbent Assay (ELISA) Method

Principle

The wells' surfaces are pre-coated with purified progesterone and prolactin antigens. When patient serum is added to the wells, the astrovirus-specific human immunoglobulin G (IgG) antibodies present bind to the antigens, while unbound materials are subsequently washed away. Following this, an enzyme conjugate is added, which binds to the antibody-antigen complex. Any excess enzyme conjugate is also washed away before the thiomethyl benzoate (TMB) chromogenic substrate is added. The enzyme conjugate's catalytic reaction is then stopped, and the color intensity is proportional to the concentration of IgG-specific antibodies in the sample. Which was then measured using a microplate reader, and the results were compared to the calibrators and controls plotted on the standard curves.

2.6 Data Analysis

Data were analyzed using SPSS version 25. Results were expressed as mean ± standard deviation (SD). Independent sample t-tests compared means between groups. Pearson correlation assessed relationships between variables. Statistical significance was set at $p < 0.05$.

3. Results

Table 3.1: Socio-Demographic Characteristics of Study Participants

Variables	Type 2 DM patients N = 70	Controls N = 20	P-Value
Age (years)			
18-30	6(8.6)	12(60.0)	
31-40	33(47.1)	6 (30.0)	
41-50	31 (44.3)	2(10.0)	
Mean ± SD	39.17±5.10	29.80±7.89	0.001*
BMI (kg/m²)			
Normal weight (18-24.9)	54 (77.1)	20(100.0)	
Overweight (25.0-29.9)	15 (21.4)	0(0.0)	
Obese (30.0-34.9)	1(1.5)	0(0.0)	
Mean ± SD	23.78±2.57	21.27±1.76	0.04*

BMI: Body Mass Index; kg/m²: Kilogram per square meter; Mean ± SD: Mean ± Standard Deviation; Values in parentheses are in percentages; *: significance at $p < 0.05$

The socio-demographic profile of the study population (Table 3.1) revealed significant baseline disparities between patients with Type 2 Diabetes Mellitus (T2DM) and the healthy control group. The T2DM cohort was significantly older than the controls (39.17

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± 5.10 vs. 29.80 ± 7.89 years; p = 0.001), with the majority of diabetic participants (91.4%) aged 31-50 years, whereas 60% of the control group were aged 18–30 years. Furthermore, the T2DM group exhibited a significantly higher mean body mass index (BMI) compared to the control group (23.78 ± 2.57 vs. 21.27 ± 1.76 kg/m²; p = 0.04). While the mean values for both groups remained within the normal range, the clinical distribution of BMI showed that 22.9% of the T2DM patients were overweight or obese, whereas the control participants were exclusively (100%) of normal weight.

Table 3.2: Comparison of FBG, Prolactin, and Progesterone Between Female Type 2 Dm Patients and Controls

Parameters	Type 2 DM patients Mean ± SD	Controls Mean ± SD	P-value
Fasting Blood Sugar (mmol/L)	10.36±4.51	4.63±0.50	0.001*
Prolactin (ng/mL)	12.46±7.57	18.03±8.1	0.005*
Progesterone (ng/mL)	1.59±0.33	1.91±0.34	0.001*

*ng/ml: nanogram per milliliter; *: significant at p < 0.05*

The biochemical profile comparison between female patients with Type 2 Diabetes Mellitus (T2DM) and healthy controls revealed significant endocrine and metabolic disruptions, as summarized in Table 3.2. Patients with T2DM exhibited significantly elevated fasting blood glucose levels (10.36±4.51 mmol/L) compared to the control group (4.63±0.50 mmol/L; p = 0.001), indicating poor glycemic control. Furthermore, the hormonal analysis indicated a significant reduction in both prolactin (12.46±7.57 ng/mL vs. 18.03±8.12 ng/mL; p = 0.005) and progesterone levels (1.59±0.33 ng/mL vs. 1.91±0.34 ng/mL; p = 0.001) in the diabetic cohort.

Figure 3.1 below demonstrates that the majority of female patients with type 2 diabetes mellitus had prolactin concentrations within the normal reference range (4.6–25 ng/mL), accounting for 84% of the study population, while a smaller proportion (16%) presented with low prolactin levels (<4.6 ng/mL).

Figure 3.1: Pattern of Prolactin Levels in Female Type 2 DM

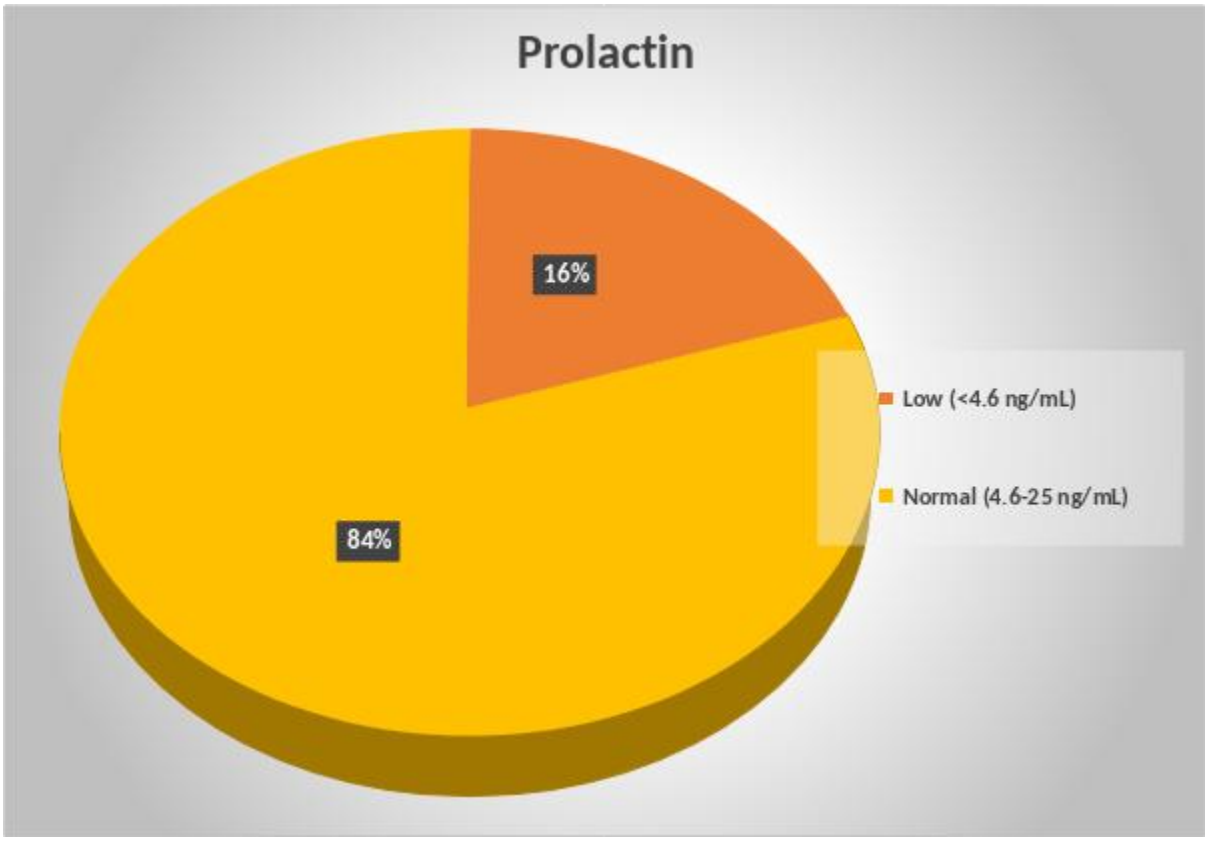

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Figure 3.2 below demonstrates that all female participants with type 2 diabetes mellitus had progesterone levels within the normal reference range (0.2–2.1 ng/mL), representing 100% of the study population.

Figure 3.2: Pattern of Progesterone Levels in Female Type 2 DM

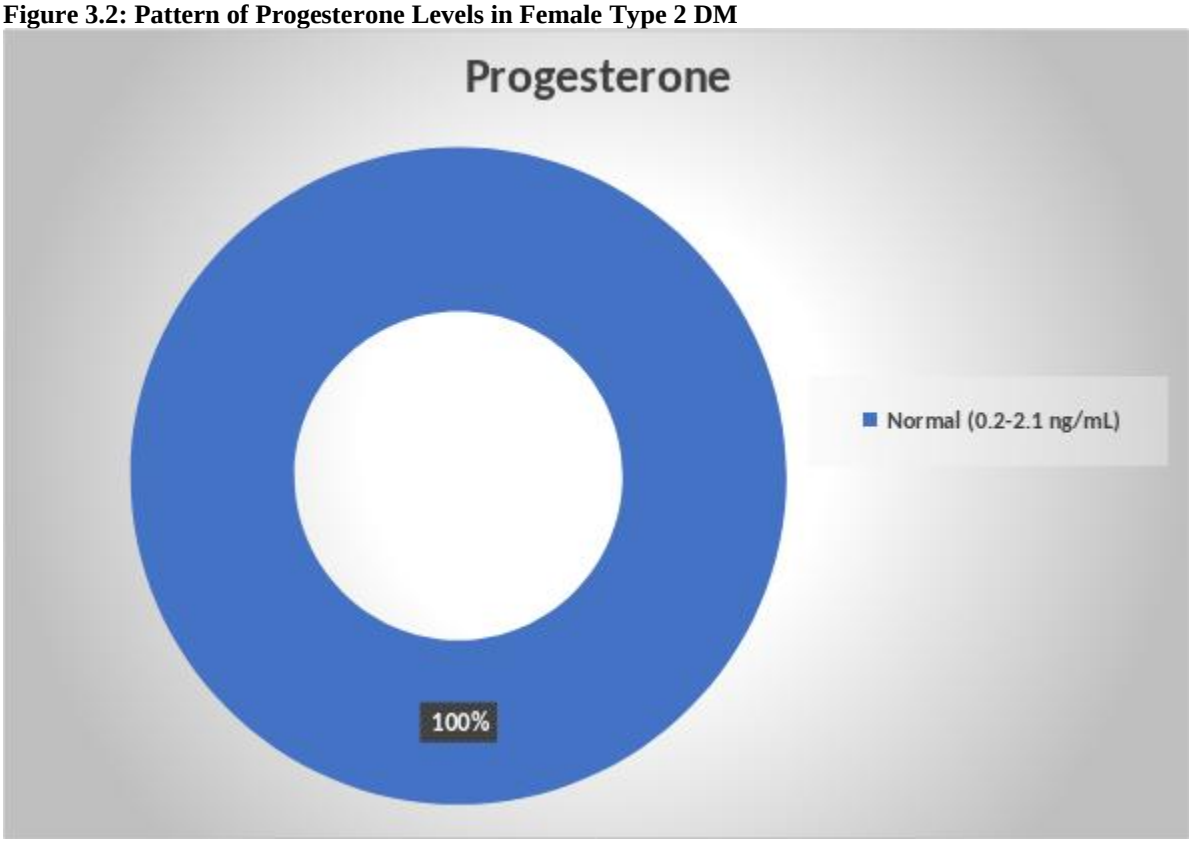

Table 3.3: Relationship between Fasting Blood Glucose, Serum Prolactin and Progesterone

Parameters	FBG (r)	Prolactin (r)	Progesterone (r)	P-value	Remarks
FBG	-	-0.019	-0.163	0.877	NS
Prolactin	-0.019	-	-0.032	0.795	NS
Progesterone	-0.163	-0.032	-	0.179	NS

NS: Non-significance; r: Pearson Correlation Coefficient

As shown in Table 3.3, a Pearson correlation analysis was conducted to evaluate the relationships between fasting blood glucose (FBS), serum prolactin, and progesterone levels. The results indicate that FBS maintained a non-significant negative correlation with both prolactin ($r = -0.019$, $p = 0.877$) and progesterone ($r = -0.163$, $p = 0.179$). Similarly, the association between prolactin and progesterone was weak and non-significant ($r = -0.032$, $p = 0.795$). Collectively, these data demonstrate that there are no statistically significant linear dependencies among the glycemic and hormonal parameters evaluated in this study population.

4. Discussion

This study demonstrated significantly lower prolactin and progesterone levels among female T2DM patients compared to healthy controls. Lower prolactin levels in female T2DM patients represent a robust finding supported by multiple population-based studies, though the progesterone association requires further investigation. The prolactin-diabetes association is well-established across diverse populations. Four independent studies, accounting for over 8,000 participants, consistently demonstrated significantly lower prolactin in T2DM patients compared to controls. A meta-analysis of 19,873 participants confirmed that higher prolactin within physiological ranges was associated with reduced prevalent T2DM

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risk (OR 0.46-0.52 in women) (Faria de Castro *et al*., 2020). Prospective cohort studies in US women (n=8,615) similarly showed inverse associations with incident diabetes (Li *et al*., 2018). These findings suggest that lower physiological prolactin levels may be associated with increased diabetes risk.

Prolactin has been shown to promote β-cell proliferation and insulin secretion through prolactin receptor-mediated mechanisms. Reduced prolactin levels may therefore contribute to impaired β-cell adaptation and worsening glycemic control. Genetic studies demonstrate that prolactin receptor (PRLR)-deficient mice exhibit 26-42% reductions in β-cell mass and blunted glucose-stimulated insulin secretion (Freemark *et al*., 2002), with a 30% reduction in β-cell mass evident in newborns (Auffret *et al*., 2013). Mechanistically, PRLR signaling promotes β-cell proliferation through mTOR pathway activation while protecting against apoptosis (Ramos-Martínez *et al*., 2022). Clinically, a large cross-sectional study of 2,377 participants found that high circulating prolactin is associated with significantly lower diabetes prevalence (adjusted odds ratio 0.38-0.47 across sexes), whereas low prolactin levels represent an independent risk factor for metabolic disease (Wang *et al*., 2013; Macotela *et al*., 2020). These findings establish reduced prolactin as a contributor to impaired β-cell adaptation and glycemic dysregulation.

Similarly, decreased progesterone levels observed in this study contrast with some reports linking elevated progesterone to insulin resistance. These discrepancies may reflect population-specific differences, menopausal status variation, or methodological heterogeneity. There is a limited number of studies relating progesterone to T2DM. However, data on gestational diabetes show lower progesterone associated with increased GDM risk (Li *et al.*, 2020), with the findings not directly transferable to T2DM. The mechanistic basis for progesterone's role in T2DM pathogenesis remains unexplored in available literature, underscoring the need for more dedicated investigation for confirmation.

In addition, no significant correlation was observed between prolactin/progesterone levels and FBG. This finding aligns with Macotela *et al*.'s (2020) framework, which reported that prolactin regulates metabolic homeostasis through multiple tissue-specific mechanisms (pancreas, liver, adipose tissue, hypothalamus) rather than simple adiposity-dependent pathways. Also, this is in concordance with Jayashankar *et al*. (2022) and Li *et al*. (2018), who found inverse associations between prolactin and glucose parameters, supporting metabolic complexity. Conversely, it is in contrast with Sattar *et al*. (2016), who reported a significant positive correlation between fasting serum glucose and prolactin, and Liu *et al.* (2021), who demonstrated that FBG was significantly associated with prolactin in multivariate analysis. Suggesting that hormonal alterations may operate through insulin sensitivity, lipid metabolism, or inflammatory pathways independent of body composition measures.

5. Conclusion

This study showed that female patients with T2DM demonstrated significantly lower serum prolactin and progesterone levels compared to non-diabetic controls. These findings suggest that reproductive hormone dysregulation may be associated with T2DM in women. Routine endocrine evaluation may be beneficial in comprehensive diabetes management.

6. Limitations of this Study

The cross-sectional design limits causal inference. Additionally, the relatively small control group may affect statistical power. Longitudinal studies are recommended to clarify temporal relationships and mechanistic pathways.

7. Acknowledgements

7.1 Conflict of Interest

The authors declare no conflict of interest.

7.2 Funding

None

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