

Research Article

Hormonal Profiles in Women with Uterine Leiomyoma: A Case-control Study in a Nigerian Cohort

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Abstract

Uterine leiomyomas (fibroids) are hormone-responsive benign tumors prevalent among women of African descent. While ovarian steroids are known contributors, the roles of gonadotropins and prolactin remain less defined in African populations. This case-control study evaluated serum levels of FSH, LH, prolactin, oestradiol, and progesterone in Nigerian women with ultrasound-confirmed leiomyomas versus matched controls. Hormonal assays were conducted using ELISA, and data were analysed using SPSS v26.0 with significance set at $p < 0.05$. Women with fibroids had significantly higher prolactin ($P < 0.001$) and oestradiol ($P < 0.01$), and lower progesterone ($P < 0.01$), while FSH and LH showed non-significant increases. Among obese leiomyoma patients, the mean levels of oestradiol, FSH, LH, prolactin, and progesterone were not significantly different across the group ($p > 0.05$). Nulliparous women with leiomyoma had significant lower level of LH compared to multiiparous women with leiomyoma ($P < 0.05$). Ethnically, Igbo women exhibited significantly elevated prolactin ($P < 0.05$), with no other hormonal differences. These findings suggest a distinct endocrine profile in Nigerian women with uterine fibroids, characterized by elevated estradiol and prolactin, and decreased progesterone. Trends observed with BMI, parity, and ethnicity imply that hormonal modulation in fibroid pathophysiology may be influenced by both metabolic and sociobiological factors. The study emphasizes the importance of considering demographic and ethnic differences in hormonal evaluation and supports the need for personalized approaches to fibroid diagnosis and treatment. Prolactin, in particular, emerges as a potential ethnic-specific biomarker with diagnostic and therapeutic relevance.

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Keywords

Uterine Fibroids, Hormonal Profiles, Gonadotropins, Prolactin, Ethnic Variation

1. Introduction

Uterine leiomyomas (UL), commonly referred to as fibroids, are monoclonal smooth muscle tumors arising from the myometrium, affecting up to 70% of women by menopause and causing significant reproductive and systemic morbidity [1]. In sub-Saharan Africa, including Nigeria, fibroid prevalence mirrors that of other populations with Black African heritage but occurs at an earlier age and with greater severity. Known risk factors include age, family history, obesity, nulliparity, and ethnicity [2]. While the central role of steroid hormones-especially estradiol and progesterone-in fibroid development has been well established [3], there remains a gap in our understanding of how upstream regulatory hormones such as follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin interplay with end-organ sensitivity to drive tumorigenesis.

Existing evidence implicates ovarian steroids in fibroid pathogenesis through their direct effects on cell proliferation, extracellular matrix (ECM) deposition, and paracrine signaling. Estradiol promotes upregulation of growth factors such as IGF-1, TGF- β , and EGF, while progesterone supports fibroid growth via induction of anti-apoptotic molecules like Bcl-2 and fibrogenic factors including TGF- β 3 [3]. However, the upstream regulators-FSH and LH-govern estrogen and progesterone synthesis and secretion, and their dysregulation may contribute to fibroid initiation or progression, independent of absolute estrogen levels. Similarly, prolactin, a peptide hormone with known effects on cell proliferation and immune modulation, has been reported to be elevated in women with fibroids, suggesting possible contributions to pathophysiology.

Local Nigerian data also point to associations between metabolic and nutritional factors and fibroid risk. For instance, a Lagos study found low serum calcium inversely associated with fibroid presence and size, although magnesium showed no association [1]. These findings underscore that the fibroid microenvironment in Nigerian women may encompass disrupted mineral metabolism in addition to hormonal dysregulation. PubMed-indexed case-control data from Nigeria confirm classical risk factors including nulliparity, family history, and obesity, but few studies have investigated reproductive hormone patterns in these patients [1].

A recent fertility-hormone profile analysis conducted in Owerri, Nigeria, revealed significantly elevated FSH, LH, estradiol, and prolactin in women with intramural fibroids compared to matched controls ($p = 0.005$) [4]. Although this study did not evaluate progesterone levels, its findings point

to significant dysregulation of the reproductive hormone axis. These findings are aligned with global observations: women with fibroids often exhibit aberrant gonadotropin and estrogen dynamics-though the underlying mechanisms remain unclear-raising questions about whether FSH and LH act as triggers or respondents to tumorous growth.

The endocrine circuitry underlying fibroid pathogenesis likely involves a complex feedback loop: dysregulated gonadotropin levels influence ovarian steroid output, which in turn modulates fibroid cell proliferation and ECM production. Conversely, fibroid presence and its endocrine activity could exert downstream effects on the hypothalamic-pituitary-gonadal axis. These interactions may differ across ethnicities, BMI categories, and parity states, making it essential to study hormonal profiles within specific clinical settings, such as Nigerian women with fibroids.

Despite the established importance of estrogen and progesterone in fibroid biology, few studies in Nigeria have systematically assessed reproductive hormone levels in women with fibroids. The Owerri study provided preliminary evidence of elevated estradiol and prolactin, but lacked evaluation of progesterones. Furthermore, no Nigerian studies have simultaneously measured the full spectrum of reproductive hormonal markers in a controlled setting, nor have they correlated hormonal profiles with clinical parameters such as fibroid size, number, BMI, and parity [5].

The objective of this study is to compare serum levels of FSH, LH, estradiol, progesterone, and prolactin between women with and without uterine leiomyomas in a case-control design. We hypothesize that women with fibroids will demonstrate distinct hormonal dysregulation-reflected in both gonadotropin and steroid hormone levels-compared to controls, and that these differences will correlate with tumor burden and clinical features.

By focusing on a Nigerian cohort, this study addresses a critical research gap. Ethnic-specific hormonal profiles may have implications for risk stratification, early diagnosis, and tailored medical management. Given the high fibroid-related morbidity and limited access to advanced surgical or interventional therapies in Nigeria, an improved understanding of hormonal dysregulation could open avenues for medical therapies that modulate hormonal axes, such as selective progesterone receptor modulators or gonadotropin-releasing hormone antagonists. We also aim to provide baseline data for larger prospective or interventional studies targeting endocrine pathways.

In summary, this study seeks to advance our understanding of fibroid endocrinology by integrating comprehensive hormonal profiling with clinical assessment in Nigerian women. The analysis elucidated whether reproductive hormone variations are consistent with international findings or reflect population-specific endocrine dynamics. Ultimately, the goal is to inform evidence-based prevention and treatment strategies that are appropriate for the Nigerian and broader sub-Saharan African context.

2. Materials and Methods

2.1. Study Design and Location

This was an analytical case-control study, age-matched subjects with different parity and body mass index status s conducted at selected diagnostic and clinical laboratories in Warri, Delta State, Nigeria. Warri is a major commercial city in southern Nigeria with a multi-ethnic population and a high rate of spontaneous clinic attendance for gynecologic ultrasonography.

2.1.1. Study Population and Ethical Approval

Four hundred (400) women were recruited into this study. Three hundred (300) women with ultrasound-guided confirmed cases of uterine leiomyomas were recruited as study subjects, while 100 women who were confirmed via ultrasound without uterine leiomyoma were recruited as control subjects. These women with fibroid were drawn from women attending Gynaecology clinic in Government Hospital Ekpan and Central Hospital, Warri, both in Delta State, Nigeria. In this study, the controls were drawn from healthy general public without leiomyoma in Ekpan and Warri. Ethical approval was obtained from the Ethics Committee of Government Hospital, Ekpan and Central Hospital Warri, Delta State with reference number and EMZ/GHE/ETN/VOL./1/5, 29/6/2016 and CHW/ECCVOL 1/ 129, 25/5/2017 respectively.

2.1.2. Sample Size Calculation

The technique used for the sample size determination was the formula by [6]. This is as shown below:

$$n = \frac{z^2 \times P(1-P)}{d^2}$$

Where; n= required sample size.

Z=Value of normal distribution at confidence level at 95% (standard value of 1.96)

P=Estimated prevalence of patient with uterine leiomyomas 18% [7].

d=Margin of error at 5% (standard value of 0.05).

Substituting into the above formula; we have

$$= \frac{1.96^2 \times 0.18(1-0.18)}{0.05^2} = \frac{0.5670202}{0.0025} = 226.8 = 227.0$$

The minimum size calculated was 227, but for the purpose of this study, the size was increased to 300 women due to

attritions and 100 controls subjects were enrolled from among the same communities.

Inclusion criteria were women of reproductive age (20-50 years), not pregnant, not on hormonal therapy, and without chronic diseases such as autoimmune disorders or cancer. Exclusion criteria included recent infections (within four weeks), immunosuppressive drug use, or prior surgical treatment for fibroids.

2.1.3. Sample Collection and Laboratory Analysis

Approximately 10 mL of venous blood was collected from each participant on day 3 and 21 of their menstrual cycle at early hours of the day between 8.00 am- 10.00 am under aseptic conditions. The day 3 samples were used for FSH, LH, PRL, E2 estimation while day 21 samples were used for progesterone estimation. Blood was centrifuged at 3,000 rpm for 10 minutes, and the serum was aliquoted into cryovials and stored at -20 °C until analysis.

2.2. Determination of Hormonal Parameters

The quantitative determination of follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL), estradiol (E2), and progesterone (PG) levels was performed using solid-phase enzyme-linked immunosorbent assay (ELISA) kits supplied by Calbiotech Inc., USA. The assays were designed to ensure high specificity and sensitivity, utilizing the streptavidin-biotin sandwich detection system.

All reagents, controls, and calibrators were used according to the manufacturer's protocols and batch information. The following kit lots and reference numbers were employed: FSH (Lot: FSS5287; Ref: FS232F), LH (Lot: LHS5205; Ref: PG3625), PRLactin (Lot: PRS5392; Ref: PR234F), Estradiol (Lot: ESG5362; Ref: ES180S), and Progesterone (Lot: PGS5374).

2.2.1. Assay Procedure for Follicle-stimulating Hormone (FSH)

For FSH determination, 50 µL of patient serum, standards, and controls were pipetted into microplate wells in duplicate, followed by 100 µL of enzyme conjugate reagent specific for FSH. The plates were incubated at room temperature (20-25 °C) for 60 minutes. Subsequently, the contents of the wells were aspirated and washed five times using an automated plate washer with 350 µL of wash buffer per cycle. The wells were blot-dried with absorbent paper. A volume of 100 µL of TMB substrate was added to each well and incubated in the dark for 15 minutes. The reaction was halted by adding 50 µL of stop solution, and absorbance was read at 450nm within 15 minutes using a microplate reader. Results were calculated and expressed in international units per milliliter (IU/mL).

2.2.2. Assay Procedure for LH, Prolactin, and Estradiol

The protocol for LH, PRL, and E2 followed a similar pattern with minor volume modifications. Briefly, 25 μ L of serum samples, standards, and controls were dispensed into designated wells in duplicate. Each well received 100 μ L of the respective hormone-enzyme conjugate reagent. The assay mixture was incubated for 60 minutes at room temperature. After incubation, plates were washed five times with 350 μ L of wash buffer per well, blot-dried, and 100 μ L of TMB substrate was added. Following a 15-minute incubation, 50 μ L of stop solution was added to terminate the reaction. Absorbance readings at 450 nm were recorded using a microplate reader, and hormone levels were calculated from standard curves.

2.2.3. Assay Procedure for Progesterone

For progesterone measurement, 20 μ L of patient serum, controls, and reference standards were added to each well in duplicate. Subsequently, 100 μ L of working HRP-progesterone conjugate and 50 μ L of biotin-progesterone reagent was added. The assay mixture was gently mixed and incubated for 60 minutes at room temperature. The microplates were washed five times with 300 μ L of wash buffer, followed by drying with absorbent paper. A 100 μ L volume of TMB substrate was added to each well and allowed to react for 15 minutes before the addition of 50 μ L of stop solution. Absorbance was recorded within 15 minutes at 450 nm. The final results were extrapolated from a standard curve and expressed in IU/mL.

2.2.4. Quality Assurance and Validation

All samples and controls were run in duplicate to ensure precision. Internal quality control materials provided in the kits were used to validate assay performance across each batch. Calibration curves exhibited strong linearity ($R^2 > 0.98$), and assay sensitivity adhered to the manufacturer's specifications. Washing procedures were automated to minimize inter-well variability.

2.2.5. Ultrasonographic Diagnosis

Transabdominal pelvic ultrasonography was performed

using a GE LOGIQ e Ultrasound Machine with a 3.5 MHz convex probe. Fibroids were classified based on location: submucosal, intramural, and subserosal. Fibroid size and number were also documented.

2.3. Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics (mean $\pm$ SEM) were used to summarize participant characteristics. Independent t-tests and one way ANOVA compared mean values between groups. A p-value < 0.05 was considered statistically significant.

3. Results

Table 1 Shows comparative analysis of the hormonal profile of the individuals investigated in this study. It was observed that the mean levels of PRL ($P < 0.001$) and E2 ($P < 0.01$) were statistically significantly higher among women with leiomyomas compared to control subjects while the mean levels of PG ($P < 0.01$) were significantly lower among women with leiomyomas compared to controls. However, the means level FSH and LH show no significant difference between women with leiomyomas compared to controls ($p > 0.05$).

Table 2 Shows comparison of hormonal parameters in women with leiomyomas based on different BMI. It was observed that the mean values of FSH, LH, PRL, PG and E2 show no significant difference ($P > 0.05$) among women with leiomyomas across the group.

Table 3 shows comparison of hormonal parameters in women with leiomyomas based on parity. There was no significant difference in the means levels of FSH, PRL, E2 and PG ($P > 0.05$) between nulliparous leiomyomas women compared to multiparous women with leiomyomas. Also, the means levels of LH was significantly lower ($P < 0.05$) in nulliparous women with leiomyomas compared to the multiparous women with leiomyomas.

Table 4 shows comparison of hormonal parameters in women with leiomyomas based on ethnicity. The mean PRL (ng/mL) value was higher among the Igbo ethnic group compared to others, and this difference was statistically significant ($P < 0.05$). The mean levels of FSH, LH, E2 and PG show no significant difference ($P > 0.05$).

Table 1. Comparative Analysis of the hormonal profile of the individuals investigated in this study.

PARAMETERS	CONTROL $\bar{X} \pm SE$ (min-max) N=100	PATIENTS $\bar{X} \pm SE$ (min-max) N=300	P-VALUE
FSH (i μ /ml)	13.12 $\pm$ 1.665 (0.07-89.67)	15.62 $\pm$ 2.306 (0.07-669.0)	0.543
LH (i μ /ml)	12.37 $\pm$ 1.697 (0.39-90.44)	13.09 $\pm$ 0.742 (0.13-90.44)	0.654

PARAMETERS	CONTROL $\bar{X} \pm SE$ (min-max) N=100	PATIENTS $\bar{X} \pm SE$ (min-max) N=300	P-VALUE
PRL (ng/ml)	13.29 $\pm$ 0.632 (0.13-13.0)	20.30 $\pm$ 0.984 (0.13-91.72)	0.001
PG (ng/ml)	10.722 $\pm$ 1.132 (0.02-93.46)	8.101 $\pm$ 0.342 (0.02-30.30)	0.003
E2 (Pg/ml)	86.83 $\pm$ 8.599 (8.90-503.00)	110.73 $\pm$ 4.171 (10.53-503.00)	0.007

P> 0.05 = no significant difference, FSH=Follicle Stimulating Hormones, LH=Luteinizing Hormones, PRL=PRLactin, PG=Progesterone, E2=Eostradiol, $\bar{X}$ =Mean, SE=Standard Error, *SIG= Level of Significant, N=number of participants.

Table 2. Comparison of hormonal Parameters in Women with Leiomyomas based on different BMI.

PARAMETERS	18.5-24.9 kg/m ² $\bar{X} \pm SE$ (min-max) n=78	25.0-29.9 kg/m ² $\bar{X} \pm SE$ (min-max) n=160	30.0-34.9 kg/m ² $\bar{X} \pm SE$ (min-max) n=61	35.0-39.9 kg/m ² $\bar{X} \pm SE$ (min-max) n=1	P-VALUE
FSH (i μ /ml)	23.07 $\pm$ 8.790 (0.07 -669.0)	12.41 $\pm$ 0.844 (0.07 -84.25)	15.04 $\pm$ 1.863 (0.60 -62.90)	9.103 $\pm$ 1.697 (7.41 -10.80)	0.291
LH (i μ /ml)	14.25 $\pm$ 2.013 (0.39 -90.44)	12.28 $\pm$ 0.917 (0.13- 76.14)	13.97 $\pm$ 1.079 (0.90 -44.54)	9.766 $\pm$ 0.865 (8.90 -10.63)	0.643
PRL (ng/ml)	31.81 $\pm$ 11.50 (0.13 -44.31)	139.49 $\pm$ 1.189 (0.00-141.67)	19.10 $\pm$ 1.924 (0.13 - 21.65)	15.79 $\pm$ 6.808 (8.98 - 22.60)	0.856
PG (ng/ml)	8.466 $\pm$ 0.667 (0.02-21.20)	7.870 $\pm$ 0.485 (0.00-30.30)	8.016 $\pm$ 0.712 (0.05-20.84)	7.732 $\pm$ 0.132 (7.60-7.86)	0.913
E2 (pg./ml)	109.78 $\pm$ 9.161 (10.53 - 503.0)	108.31 $\pm$ 6.003 (11.0 -503.0)	117.94 $\pm$ 6.447 (19.51-231.0)	95.16 $\pm$ 13.84 (81.32-109.00)	0.832

P>0.05 = No Significant Difference, FSH=Follicle Stimulating Hormones, LH=Luteinizing Hormones, PRL=PRLactin, PG =Progesterone, E2=Eostradiol, $\bar{X}$ =Mean, SE=Standard Error, BMI=Body Mass Index, N=Number of Participants.

Table 3. Comparison of hormonal Parameters in Women with Leiomyomas based on parity.

PARAMETERS	MULTIPAROUS $\bar{X} \pm SE$ (min-max) N=95	NULLIPAROUS $\bar{X} \pm SE$ (min-max) N=205	P-VALUE
FSH i μ /ml	19.34 $\pm$ 6.996 (0.11-669.0)	13.90 $\pm$ 0.952 (0.07-84.25)	0.274
LH i μ /ml	15.58 $\pm$ 1.814 (0.60-87.96)	11.95 $\pm$ 0.677 (0.13-90.44)	0.022
PRL ng/ml	19.96 $\pm$ 1.773 (2.62-91.60)	20.48 $\pm$ 1.186 (0.13-91.72)	0.805
PG ng/ml	8.262 $\pm$ 0.574 (0.02-21.0)	8.027 $\pm$ 0.424 (0.05-30.30)	0.749

PARAMETERS	MULTIPAROUS $\bar{X} \pm SE$ (min-max) N=95	NULLIPAROUS $\bar{X} \pm SE$ (min-max) N=205	P-VALUE
E2 Pg/ml	99.41 $\pm$ 6.280 (10.53-321.55)	115.97 $\pm$ 5.336 (11.0-503.0)	0.065

P>0.05 = No Significant Difference, P<0.05 = Significant Difference, FSH=Follicle Stimulating Hormones, LH=Luteinizing Hormones, PRL=PRLactin, PG =Progesterone, E2=Eoestradiol, $\bar{X}$ =Mean, SE=Standard Error, BMI=Body Mass Index, N=Number of Participants.

Table 4. Comparison of hormonal Parameters in Women with Leiomyomas based on Parameters and Ethnicity.

PARAMETERS	URHOBO $\bar{X} \pm SE$ (min-max) n= 196	ISOKO $\bar{X} \pm SE$ (min-max) n= 9	IJAW $\bar{X} \pm SE$ (min-max) n= 29	IGBO $\bar{X} \pm SE$ (min-max) n=11	ITSHEKIRI $\bar{X} \pm SE$ (min-max) n= 22	ETHNICITY IN OTHER STATES $\bar{X} \pm SE$ (min-max) n= 33	P-VALUE
FSH (iu/ml)	17.63 $\pm$ 3.492 (0.07-669.00)	8.449 $\pm$ 1.476 ^b (3.23-17.74)	10.51 $\pm$ 1.127 ^b (3.00-25.00)	11.66 $\pm$ 3.329 ^b (2.62-42.76)	14.35 $\pm$ 2.295 ^b (3.90-56.61)	12.33 $\pm$ 1.803 ^b (0.30-56.61)	0.902
LH (iu/ml)	13.15 $\pm$ 0.937 (0.13-87.96)	14.29 $\pm$ 9.577 (0.39-90.44)	13.71 $\pm$ 2.265 (0.67-56.81)	8.601 $\pm$ 1.287 (3.74-15.84)	13.01 $\pm$ 1.652 (0.70-37.87)	13.51 $\pm$ 1.793 (0.40-46.56)	0.912
PRL (ng/ml)	19.53 $\pm$ 1.083 ^b (0.13-91.60)	16.35 $\pm$ 2.149 ^b (8.16-24.63)	21.37 $\pm$ 3.753 ^b (0.13-70.01)	34.49 $\pm$ 9.369 ^a (7.90-91.72)	15.35 $\pm$ 1.323 ^b (4.93-26.00)	23.35 $\pm$ 3.819 ^b (5.60 -91.60)	0.041
PG (ng/ml)	7.926 $\pm$ 0.412 (0.02-27.00)	10.02 $\pm$ 2.139 (4.27-20.81)	7.398 $\pm$ 1.089 (0.32-19.00)	8.348 $\pm$ 1.988 (0.12-18.60)	7.421 $\pm$ 0.801 (0.67-15.24)	9.484 $\pm$ 1.268 (0.12-30.30)	0.593
E2 (pg/ml)	108.34 $\pm$ 5.256 (10.53-503.00)	71.59 $\pm$ 25.22 (22.49-262.44)	130.96 $\pm$ 16.50 (23.13-503.00)	117.21 $\pm$ 18.35 (44.60-206.00)	114.5 $\pm$ 9.731 (14.90-214.34)	113.65 $\pm$ 11.19 (14.18-321.53)	0.387

P> 0.05 = no significant difference, P< 0.05 = significant difference, FSH=Follicle Stimulating Hormones, LH=Luteinizing Hormones, PRL=prolactin, PG =Progesterone, E2=Eoestradiol, $\bar{X}$ = Mean, SE=Standard Error,, BMI=Body Mass Index, N=Number of Participants.

4. Discussion

Uterine leiomyomas are the most common benign pelvic tumors in women of reproductive age, with heightened prevalence and clinical severity reported among women of African descent. The conventional understanding of their pathogenesis primarily centers around the proliferative roles of ovarian steroids-estradiol and progesterone. However, recent research has expanded focus to include other components of the endocrine axis, notably gonadotropins and prolactin, which modulate reproductive tissue physiology in concert with metabolic and demographic factors. This study, conducted in a Nigerian population, aimed to investigate the hormonal profiles of women with uterine leiomyomas and analyze differences across body mass index (BMI), parity, and ethnicity.

At the core of the findings is a distinctive hormonal signature in women with uterine leiomyomas. Specifically, serum prolactin ($P < 0.001$) and estradiol ($P < 0.01$) were significantly elevated, while progesterone levels were notably

reduced ($P < 0.01$) compared to controls. FSH and LH were also higher, although these did not reach statistical significance. These results affirm previous evidence suggesting that estradiol promotes fibroid growth through upregulation of progesterone receptor expression and stimulation of fibrogenic signaling cascades such as the PI3K/AKT and ERK pathways [8]. Elevated systemic estradiol has consistently been associated with larger fibroid size and increased disease burden in epidemiologic and clinical studies.

Prolactin pronounced elevation aligns with growing recognition of its mitogenic and angiogenic functions beyond lactation. Kirschen *et al.* (2021) [9] demonstrated that fibroid tissues not only express prolactin receptors but may also produce prolactin in a paracrine or autocrine fashion. In vitro studies further show that blocking prolactin significantly inhibits fibroid cell proliferation. A case-control study by Levy *et al.* (2013) [10] reported preoperative prolactin levels of 169.6 ng/mL in leiomyoma patients, dramatically higher than levels in controls (~19 ng/mL). This supports prolactin role as both a biomarker and potential therapeutic target in fibroid pathology.

Interestingly, progesterone—a hormone long implicated in leiomyoma proliferation—was significantly lower in affected women, despite elevated estradiol. This hormonal discordance may suggest luteal phase dysfunction or HPO axis feedback abnormalities in women with fibroids. Progesterone's role in fibroid growth is mediated by its receptor-dependent activation of proliferative and anti-apoptotic genes; thus, reduced systemic levels could reflect either downregulation in the luteal phase or a local increase in tissue-specific receptor activity unreflected in serum measurements.

The findings in Table 2 indicate that among women with leiomyomas, there was no statistically significant difference ($P > 0.05$) in the mean levels of FSH, LH, prolactin, progesterone, and estradiol across different BMI groups. This suggests that body mass index does not have a notable effect on the baseline levels of these reproductive hormones in women with fibroids. This outcome aligns with previous studies which have shown that while fibroid development is influenced by estrogen and progesterone, systemic hormone levels often remain unchanged regardless of BMI. For example, Cramer et al. (2011) [11] found no significant differences in plasma levels of estrone, estradiol, and progesterone between women with large fibroids and those without. Similarly, Deligdisch and Loewenthal (1970) [12] reported no significant changes in FSH, LH, and prolactin levels among women with leiomyomas when compared with controls. The reason for this may lie in the fact that fibroid growth is more closely related to increased expression of hormone receptors within the fibroid tissue rather than elevated circulating hormone levels. These receptors, particularly for estrogen, progesterone, FSH, and LH, tend to be overexpressed in fibroid tissues, making them more responsive even to normal hormone concentrations [13]. Therefore, while BMI may influence other aspects of reproductive health, it does not appear to significantly affect the hormonal profile of women with leiomyomas, and fibroid pathophysiology may be driven more by local tissue sensitivity than by systemic hormonal changes.

The comparison of hormonal parameters in women with leiomyomas based on parity revealed that there was no significant difference in the mean levels of FSH, prolactin, estradiol, and progesterone ($P > 0.05$) between nulliparous and multiparous women. However, luteinizing hormone (LH) levels were significantly lower ($P < 0.05$) in nulliparous women compared to multiparous women with leiomyomas. This suggests that while parity does not seem to influence most baseline reproductive hormone levels, it may be associated with differences in LH secretion.

This finding is consistent with previous studies that have shown parity has a modulatory effect on the hypothalamic-pituitary-ovarian axis. Pregnancy and childbirth are known to induce long-lasting changes in hormone regulation, particularly affecting gonadotropin secretion. For instance, Laughlin-Tommaso et al. (2011) [14] reported that parity has a protective effect against the development and progression of uterine fibroids, which may be related to hormonal adapta-

tions after pregnancy. Although most studies focus on the structural effects of pregnancy on the uterus, it is also recognized that parous women tend to have higher LH responses during certain phases of the menstrual cycle compared to nulliparous women [15].

Moreover, Okolo (2008) [16] noted that nulliparity is a well-established risk factor for uterine fibroids and proposed that repeated anovulatory cycles or unopposed estrogen exposure in nulliparous women may contribute to fibroid development. However, even with this heightened risk, systemic levels of estrogen and FSH remain within normal ranges, as also observed in the current findings. The significant difference in LH levels may reflect altered pituitary sensitivity or feedback mechanisms modulated by parity.

Another study by Wise et al. (2005) [17] using data from the Black Women's Health Study also supported the observation that multiparous women have a lower incidence of fibroids, and hormonal variations between nulliparous and parous women may partially explain this trend. While the study focused primarily on incidence, it emphasized that hormonal factors play a key role in the pathogenesis of leiomyomas, even though systemic hormone levels like estradiol and progesterone often remain similar.

Overall, the present findings suggest that parity does not cause major shifts in baseline levels of reproductive hormones like FSH, estradiol, prolactin, or progesterone. However, LH appears to be more sensitive to parity status, likely due to changes in pituitary regulation following childbirth. This underlines the importance of considering reproductive history when assessing hormonal profiles in women with leiomyomas.

Among women with leiomyomas from different ethnic backgrounds, the average levels of FSH, LH, estradiol, and progesterone did not differ significantly, but the mean prolactin (PRL) level was significantly higher among the Igbo compared to other ethnic groups ($P < 0.05$). This suggests an ethnicity-specific elevation in prolactin among Igbo women with fibroids, while other reproductive hormones remain similar across ethnicities.

This finding aligns with research showing that systemic prolactin can vary among women with fibroids. A study of Nigerian women reported significantly higher prolactin levels in patients with uterine fibroids compared to postoperative or control groups, and noted a correlation between prolactin levels and fibroid burden [10]. A broader systematic review also found that serum prolactin was significantly elevated in leiomyoma patients in case-control and cross-sectional studies [18].

Although prolactin witnessed differences by ethnic groups, differences could be due to genetic, environmental, or lifestyle factors affecting prolactin secretion or metabolism. In a Nigerian fertility clinic population, prolactin and estradiol were significantly elevated in infertile women, with no difference in LH or FSH, suggesting regional variations in hormone profiles [19].

Igbo women might have a predisposition to higher pituitary

prolactin output or decreased clearance. Hyperprolactinemia may worsen fibroid pathology, as local prolactin promotes cellular proliferation and fibrosis within fibroid tissue [20].

Although FSH, LH, estradiol, and progesterone are unaffected by ethnicity, the elevated mean prolactin in Igbo women with leiomyomas mirrors broader data linking fibroids and prolactin in Nigerian populations. This emphasizes the importance of incorporating prolactin assessment in fibroid evaluations, particularly in ethnic groups showing elevated levels.

5. Conclusion

This study explored the hormonal profiles of Nigerian women with uterine leiomyomas, analyzing variations across BMI, parity, and ethnicity. Key findings revealed significantly elevated levels of estradiol and prolactin, alongside reduced progesterone, suggesting possible luteal phase dysfunction or altered HPO axis regulation. While FSH and LH were elevated, these changes were not statistically significant overall. No hormonal differences were observed across BMI groups, indicating that fibroid-related hormonal changes may be independent of body weight. However, LH levels were significantly lower in nulliparous women, pointing to parity-related hormonal modulation. Notably, prolactin levels were significantly higher among the Igbo ethnic group, suggesting potential genetic or environmental influences. These findings align with previous studies highlighting prolactin's mitogenic role in fibroid pathology. The study emphasizes the importance of considering demographic and ethnic differences in hormonal evaluation and supports the need for personalized approaches to fibroid diagnosis and treatment.

6. Recommendation

Future research should incorporate longitudinal designs and molecular profiling to better understand the endocrine drivers of fibroid heterogeneity and to inform culturally and biologically tailored management strategies.

Abbreviations

HPO	Hypothalamic Pituitary Ovarian
BMI	Body Mass Index
LH	Lutenizing Hormone
PRL	Prolactin
FSH	Follicle Stimulating Hormone
E2	Estradiol
SEM	Standard Error of Mean
UL	Uterine Leiomyomas

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The data are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

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