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Full-Length Research Article

Endogenous Testosterone Predicts Cognitive Function in Women with Epilepsy of Child-Bearing Age

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Summary: Hormonal disruptions due to seizures and antiseizure medications (ASMs) might be responsible for the cognitive dysfunction in people with epilepsy. This study investigated the association between reproductive hormones and Cognitive Impairment (CI) in women with epilepsy (WWE). A cross-sectional study was conducted on 150 WWE aged 18–45 years. Epilepsy diagnosis was clinical and supported by electroencephalography (EEG). Cognitive function was evaluated using the Community Screening Instrument for Dementia (CSID). Serum levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH), progesterone, estradiol, prolactin, and testosterone were measured during the follicular and luteal menstrual phases. Analyses included Age group, which examined CI distribution across different age ranges, Mann-Whitney U tests for group comparisons, logistic regression for predictors, and receiver operating characteristic (ROC) curve analysis for diagnostic accuracy ($p \leq 0.05$). Of the participants, 28% had CI. The CI group exhibited higher follicular-phase LH ($p = 0.002$) and testosterone ($p < 0.001$), as well as high luteal-phase LH ($p = 0.039$) and high LH/FSH ratio ($p = 0.046$), but lower luteal-phase progesterone ($p = 0.039$), estradiol ($p = 0.042$), and prolactin ($p = 0.023$) compared to the non-CI group. Logistic regression identified testosterone as the sole significant predictor ($p = 0.004$; odds ratio = 0.555; 95% confidence interval, 0.373–0.825), indicating a 44.5% reduction in CI odds per unit increase. ROC analysis demonstrated moderate predictive accuracy for testosterone (area under the curve = 0.708; $p < 0.001$). Elevated follicular-phase LH and testosterone, luteal-phase LH alongside LH/ FSH, are associated with CI in WWE, with testosterone emerging as a key predictor. These findings indicate that hormonal factors play a role in cognitive impairment among individuals with epilepsy and suggest that focusing on hormone-based therapies might augment ASMs to improve treatment outcomes.

Keywords*: Cognitive impairment, women with epilepsy, luteinizing hormone, testosterone, Mann-Whitney U test, logistic regression*

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INTRODUCTION

Epilepsy affects about 50 million people worldwide, with women comprising roughly half of these cases. Women with epilepsy (WWE) face unique challenges due to interactions among seizures, antiseizure medications (ASMs), and reproductive hormones (Hophing et al., 2022). Women with epilepsy experience cognitive impairment, struggling with memory, attention, or problem-solving, which can be due to hormonal fluctuations during menstrual cycles, pregnancy, or menopause (Svalheim et al., 2015). Furthermore, frequent seizures or the activity of ASMs can disrupt hormone balance (Eddy et al., 2011). For instance, enzyme-inducing ASMs like carbamazepine increase sex hormone-binding globulin (SHBG), reducing free testosterone and estradiol, potentially affecting neuroprotection (Couper et al., 2025).

Hormones such as estrogen, progesterone, and testosterone have a significant influence on brain function. They affect neural connections, neurotransmitters, and regions like the hippocampus, which are critical for memory and learning

(McEwen & Milner, 2017). Estrogen can increase seizure risk by enhancing brain excitability, while progesterone often reduces it, acting as a natural anticonvulsant. However, ASMs can disrupt this balance, leading to conditions like polycystic ovary syndrome (PCOS) or irregular periods, which may impair cognition (Luqman Ogunjimi et al., 2021a; Zhou et al., 2012).

Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) also impact cognition. Elevated LH levels are associated with cognitive decline, possibly through receptors in the hippocampus and prefrontal cortex, which regulate learning and memory (Blair et al., 2015). In WWE, high LH may worsen cognitive deficits, particularly in temporal lobe epilepsy, where hormonal dysregulation is prevalent (Stoffel-Wagner et al., 1998). Testosterone's role is complex: normal levels support neural growth and reduce inflammation, but elevated levels, often seen in PCOS (affecting 10–20% of WWE), may increase seizure risk and cognitive issues (Höfer et al., 2013; Li et al., 2021).

Most research on hormones and cognition in WWE focuses on older populations or lacks menstrual phase-specific data; however, this study assesses hormonal interplay (FSH, LH, progesterone, estradiol, prolactin, testosterone) in WWE with and without cognitive impairment in the younger population..

SUBJECTS AND METHODS

Study Design and Participants: This was a hospital-based cross-sectional study conducted among 150 WWE aged 18–45 years from outpatient clinics at University College Hospital, Ibadan, Nigeria (UI/EC/15/0077), after due ethical approval. Epilepsy diagnosis was clinical and supported by electroencephalography (EEG) based on the 2017 International League Against Epilepsy (ILAE) and Clinical Neurophysiology Society's Standardized Critical Care EEG (Fisher et al., 2017; Hirsch et al., 2021). Participants had regular menstrual cycles (21–35 days) with predictable menstrual flow. Menstrual phase classification was based on self-reported menstrual calendars rather than objective ovulation confirmation. The follicular phase was defined as days 1–14 from the onset of menstruation and the luteal phase as days 15–28. (Herzog, 2015). Pregnant women were excluded, as were those in menopause or those with a background of mental health issues. The data on age, education, seizure type, duration, and ASMs were collected using a questionnaire.

Sample Size Determination: Sample size was determined using Pocock's formula for two-group mean comparison, assuming a 3.0-point difference in CSID scores, a pooled SD of 6.5–7.0, $\alpha = 0.05$, and 80% power. This gave an estimated minimum of 140–160 participants. A total of 150 participants was targeted, with bootstrap resampling (B = 1000) planned for robustness due to expected non-normality (Ogunjimi et al., 2021; Ta et al., 2009).

Cognitive Screening: Cognitive function was assessed using the Community Screening Instrument for Dementia (CSID), a culturally adapted screening tool validated for use in Nigerian and other African populations (Ogbimi et al., 2023). The CSID evaluates memory, language, attention, orientation, and executive function. Cognitive impairment was defined as a CSID score below two standard deviations of the mean composite cognitive score of the age- and education-matched Nigerian general population. (Ojo et al., 2012).

Hormone Sampling and Assays: Blood samples (10 mL) were collected into serum separator tubes (SST) during the follicular (days 1–15) and luteal (days 15–28) phases, tracked via menstrual calendars. All samples were drawn in the morning (8:00–10:00 AM) after overnight fasting to ensure uniformity and minimize diurnal variation in hormone levels. Samples were centrifuged at 1200 g for 10 minutes to obtain serum fractions, which were batched and stored at −20°C until analysis. Serum levels of FSH, LH, progesterone, estradiol, prolactin, and testosterone were measured using AccuBind ELISA kits from Monobind Inc., 100 North Pointe Drive, Lake Forest, CA 92630, USA, based on the competitive enzyme immunoassay principle. All standards, controls, and samples were assayed in duplicate with strict adherence to the manufacturer's protocol, as stated in (Luqman Ogunjimi et al., 2021b)

Statistical Analysis

The data obtained were entered in Microsoft Excel and then transferred to SPSS (Social Package for the Social Sciences, Version 22). SPSS v.22 was used for analysis. The test of normality was done using the Shapiro-Wilk test, $p<0.001$, and hormone data were non-normal, so Mann-Whitney U tests were used to compare levels between CI and NCI groups. Logistic regression was used to test which hormones predicted CI. ROC analysis checked the ability to identify CI. Significance was set at $p \leq 0.05$.

RESULTS

Figure 1 illustrates the distribution of cognitive impairment across different age groups, revealing a statistically significant association ($p = 0.003$). The numbers of cognitively impaired individuals were 7 (4.7%) in the <20 years group, 16 (10.7%) in the 21–30 years group, and 19 (12.7%) in the 31–40 years group. There was a significant relationship between age and cognitive impairment, with the highest proportion observed in the 31–40 years age group ($p = 0.003$). Of the 150 WWE, 42 (28%) had cognitive impairment (CI), while 108 (72%) were non-cognitively impaired (NCI).

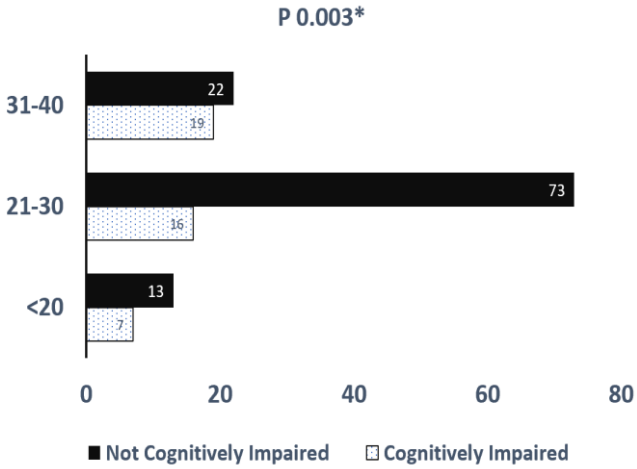

Figure 1:
Distribution of Cognitive Impairment Across Age Groups

The CI group had significantly higher mean ranks for follicular-phase LH (93.19 vs 68.62, p=0.002) and testosterone (97.98 vs 66.76, p<0.001). The cognitively impaired group had significantly higher luteal-phase LH (93.55 vs. 68.48, p = 0.039) and LH/FSH ratio (86.24 vs. 70.59, p = 0.046), and lower progesterone (63.74 vs. 80.07, p = 0.039), estradiol (63.95 vs. 79.99, p = 0.042), and prolactin (61.99 vs. 79.94, p = 0.023) than the non-impaired group (Table 1).

Table 1:
Hormone Profile Comparison: Cognitively Impaired vs. Not Cognitively Impaired (Mann-Whitney U)

Hormone Variable	Cognitively Impaired (N=42) Mean Rank	Not Cognitively Impaired (N=108) Mean Rank	p-value
Follicular Phase FSH (mIU/mL)	82.12	72.93	0.245
Follicular Phase LH (mIU/mL)	93.19	68.62	**0.002***
FP LH/FSH ratio (unitless)	78.67	72.85	0.457
FP Progesterone (ng/mL)	66.88	78.85	0.130
FP Estradiol (pg/mL)	64.39	79.16	0.060
FP Estrogen/Progesterone ratio (unitless)	76.01	71.77	0.581
FP Prolactin (ng/mL)	67.27	78.03	0.171
Luteal Phase (LP) FSH (mIU/mL)	79.11	74.10	0.526
LP LH (mIU/mL)	93.55	68.48	**0.039***
LP LH/FSH	86.24	70.59	**0.046***
LP Progesterone (ng/mL)	63.74	80.07	**0.039***
LP Estradiol (pg/mL)	63.95	79.99	**0.042***
LP Estrogen/Progesterone ratio (unitless)	80.75	72.74	0.308
LP Prolactin (ng/mL)	61.99	79.94	**0.023***
Testosterone (ng/mL)	97.98	66.76	**<0.001***

FP = Follicular Phase; LP = Luteal Phase; FSH = Follicle-Stimulating Hormone; LH = Luteinizing Hormone; LH/FSH = Ratio of Luteinizing Hormone to Follicle-Stimulating Hormone; Estrogen/Progesterone = Ratio of Estradiol to Progesterone. p ≤ 0.05 indicates statistical significance

Table 2:
Logistic Regression: Predictors of Cognitive Impairment

Hormone Variable	B	p-value	95% CI for OR (Lower–Upper)
Follicular Phase LH (mIU/mL)	-0.012	0.218	0.970–1.007
LP LH (mIU/mL)	-0.045	0.055	0.914–1.001
LP LH/FSH	0.000	0.987	0.994–1.006
LP Progesterone (ng/mL)	0.018	0.456	0.971–1.067
LP Estradiol (pg/mL)	0.002	0.204	0.999–1.006
LP Prolactin (ng/mL)	0.009	0.622	0.973–1.047
Testosterone (ng/mL)	-0.590	**0.004***	0.373–0.825

LP = Luteal Phase; LH = Luteinizing Hormone; FSH = Follicle-Stimulating Hormone; B = Regression coefficient; OR = Odds Ratio; CI = Confidence Interval. p ≤ 0.05 indicates statistical significance.

Logistic regression identified testosterone as the only significant predictor (B=-0.590, p=0.004, OR=0.555, 95% CI 0.373–0.825), suggesting a 44.5% reduction in CI odds per unit increase in testosterone. Other hormones, like follicular phase LH (p=0.218, OR=0.988) and luteal phase LH (p=0.055, OR=0.956), were not significant predictors.
Receiver operating characteristic (ROC) analysis was performed for all hormonally significant variables to compare their diagnostic performance for cognitive impairment. Testosterone demonstrated the highest discriminatory ability (AUC = 0.703), followed by luteal-phase LH (AUC = 0.671) and follicular-phase LH (AUC = 0.668). The LH/FSH ratio showed weak discrimination (AUC = 0.598). Luteal-phase progesterone (AUC = 0.377), estradiol (AUC = 0.393), and prolactin (AUC = 0.374) demonstrated poor discriminatory performance (Table 3 and Fig. 2)

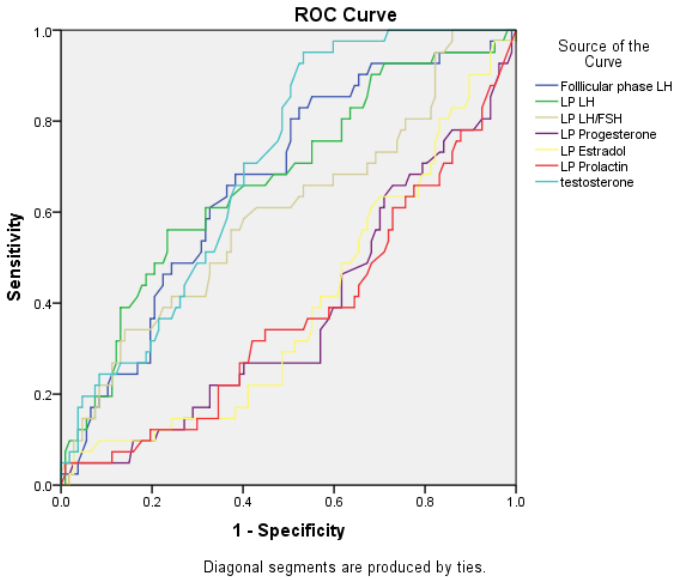

Figure 2:
ROC Curve Analysis: Testosterone as a Predictor of Cognitive Impairment

Table 3:
ROC Curve Analysis: Testosterone as a Predictor of Cognitive Impairment

Test Result Variable	Area Under the Curve (AUC)
Follicular Phase LH (mIU/mL)	0.668
Luteal Phase (LP) LH (mIU/mL)	0.671
LP LH/FSH	0.598
LP Progesterone (ng/mL)	0.377
LP Estradiol (pg/mL)	0.393
LP Prolactin (ng/mL)	0.374
Testosterone (ng/mL)	0.703

AUC=Area Under the Receiver Operating Characteristic (ROC) Curve.

DISCUSSION

This study interplayed between hormones and Cognitive Impairment in WWE. In this study, WWE with CI was associated with increased follicular phase luteinizing hormone (LH), luteal phase LH, luteal phase LH/FSH ratio, and testosterone, whereas lower luteal phase progesterone, estradiol, and prolactin levels were observed. Logistic

regression identified testosterone as the only significant predictor of CI. ROC analysis showed testosterone has a moderate ability to distinguish CI from NCI with 70.8% accuracy.

Elevated LH levels in the follicular and luteal phases of CI suggest disrupted cognitive function, likely due to overstimulation of hippocampal and prefrontal cortex receptors critical for memory and learning (Pletzer et al., 2019). A higher luteal phase LH/FSH ratio points to gonadotropin dysregulation, potentially worsening cognitive deficits, especially in temporal lobe epilepsy, where hormonal surges impair neural performance (Lai et al., 2020). Lower luteal phase progesterone, estradiol, and prolactin in CI indicate reduced neuroprotection, as these hormones suppress seizures and boost cognition; their deficiency may increase brain vulnerability during menstrual cycle phases linked to catamenial epilepsy (Maguire & Nevitt, 2019). While testosterone typically supports neural growth and reduces inflammation, in WWE, higher levels may disrupt neuroprotection by enhancing excitatory neurotransmission, particularly in temporal lobe epilepsy, where seizures further impair hippocampal function critical for memory and learning (Cutia & Christian-Hinman, 2023; Wang et al., 2025).

Prior research indicates that testosterone can positively influence cognition, particularly at moderate levels, by supporting neural growth, reducing inflammation, and improving memory and spatial abilities (Wahjoepramono et al., 2016). However, logistic regression in this study identified testosterone as a significant predictor of cognitive impairment, suggesting that higher testosterone levels may reduce the odds of CI. This study focuses on people of childbearing age, which may contribute to the observed differences between regression results and mean rank associations for testosterone. However, this study did not assess antiseizure medications (ASMs), such as carbamazepine, which may increase sex hormone-binding globulin (SHBG), potentially leading to a reduction in the neuroprotective effects of free testosterone (Couper et al., 2025; Kanojia et al., 2025). Elevated testosterone increases seizure frequency by enhancing brain excitability, particularly in temporal lobe epilepsy, and causes inflammation, both worsening cognition by affecting memory critical areas like the hippocampus (Reddy, 2013; Sha et al., 2023). Additionally, hormones like LH and estradiol are closely linked to testosterone, which can confuse the analysis, making it hard to isolate testosterone's true role in cognitive impairment (Li et al., 2024). The above reasons may explain the conflict where higher testosterone is associated with reduced CI odds in regression but higher mean ranks in the CI group. Testosterone's moderate discriminatory power in ROC analysis suggests it's a moderate biomarker for CI.

The participants were recruited from a single tertiary hospital in Nigeria, so the findings may not extend to women with epilepsy from other ethnic, geographic, or socioeconomic backgrounds. The cross-sectional nature of this study prevents the establishment of causality. The small sample size of women with cognitive impairment (CI) compared to those without limited the statistical power to detect subtle effects of other hormones. This study didn't assess the sex-hormone-binding globulin (SHBG) assay, which would have allowed for the measurement of free testosterone and estradiol, which may be more biologically relevant than total concentrations. The study did not control for the type, dose, or duration of ASMs, despite evidence that many drugs (e.g., carbamazepine) alter hormone metabolism and could confound the observed associations. Hormone measurements were taken only during one follicular and one luteal phase; hormonal fluctuations across multiple menstrual cycles were not captured. Despite these limitations, this study has laid the groundwork for understanding hormonal interplay and cognitive function. While other studies have always focused on the older age group, this study focuses more on the younger and childbearing age group. Furthermore, menstrual phase timing was based on calendar mapping rather than ultrasonographic follicular tracking or LH surge kits; however, the inclusion of only women with regular, predictable cycles helped mitigate potential phase misclassification.

Future studies should address this major limitation by including the ASM regimen as a covariate in statistical models (e.g., stratification or multivariable adjustment), collecting detailed ASM history (type, dose, duration, and enzyme-inducing vs. non-inducing status), and, where feasible, measuring SHBG to estimate free hormone levels. Despite these limitations, this study provides important preliminary evidence on the interplay between endogenous reproductive hormones and cognitive function in women of childbearing age with epilepsy

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