ISSN: 0975-833X

Available online at http://www.journalcra.com

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

International Journal of Current Research
Vol. 13, Issue, 09, pp.18774-18780, September, 2021

DOI: https://doi.org/10.24941/ijcr.42202.09.2021

RESEARCH ARTICLE

ASSESSMENT OF REPRODUCTIVE HORMONES IN OBESE INFERTILE WOMEN IN SOUTHERN (NIGER DELTA REGION), NIGERIA

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ARTICLE INFO

Article History:
Received 15th June, 2021
Received in revised form
24th July, 2021
Accepted 29th August, 2021
Published online 30th September, 2021

Key Words:
Body Mass Index,
Infertility, Obesity,
and Reproductive Hormones.

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ABSTRACT

Background: Obesity is a medical problem that increases the risk to many reproductive issues in women, and there is a high prevalence of obese women in the population attending the different fertility clinics in the Niger Delta Region of Nigeria. **Objective**: This cross-sectional study was carried out to assess reproductive hormones in obese infertile women. **Methods:** A total of 626 women comprising of 513 obese infertile women and 113 not obese women who serve as control were recruited for the study.Anthropometric measurements were taken and Body Mass Index were calculated. A non-fasting venous blood sample was collected from the subjects and analyzed for serum Estrogen, Luteinizing Hormone (LH), Follicle Stimulating Hormone (FSH), Progesterone, Inhibin, and Prolactin. **Results:** The result revealed that obese infertile women with primary and secondary infertility showed a statistically significant ($p<0.05$) increase in estrogen, LH, FSH, prolactin levels, and decreased progesterone and inhibin levels. However, women with secondary infertility had slightly higher levels of all analyzed hormones than primary infertility women. The study also revealed that hyperestrogenism was more prevalent among the obese women with primary infertility and secondary infertilitycompared with other gonadal disorders, but slightly higher in secondary infertility women. Infertility showed positive correlation with Body Mass Index. LH, FSH, E2, progesterone and prolactin showed a positive correlation withBMI in primary and secondary infertility women, while inhibin showed a negative correlation with Body Mass Index.**Conclusion:** Therefore, weight loss should be considered as a first line of treatment in obese women with hormonal imbalance.

Citation: Ferdinand Ezeiruaku Chukwuma and Onitsha Enebrayi Nelson. "Assessment of reproductive hormones in obese infertile women in southern (niger delta region), nigeria*", 2021. International Journal of Current Research, 13, (09), 18774-18780.*

INTRODUCTION

Obesity is a global problem, and its incidence worldwide has continuously escalatedduring the past two decades despite efforts to confront it (Haslam and James, 2005). The World Health Organization estimates that approximately one (1) billion people globally are overweight and that more than 600 million adults of these population are obese (WHO, 2016). Majority of this population is made up of women of childbearing age, and many of the risk factors associated with obesity can also lead to infertility (Vahratian, 2009; Pasquali*et al.,* 2003). According to World Health Organization, obesity is defined as a body mass index of ≥30 kg/m^2 (WHO, 2000). It is a condition characterized by excessive body fat that is closely associated with insulin resistance (Wyatt, 2013).

Obesity is classified according to the body mass index (BMI). A body mass index (BMI) between 18.5kg/m^2 and 24.9kg/m^2 is considered as normal, a BMI between 25kg/m^2 and 29.9 kg/m^2 is overweight and a BMI between 30kg/m^2 and 39.9kg/m^2 is obese, and a BMI value of 40kg/m^{2}and above is accepted as morbid obese (Arslan*et al.,* 2010). In Nigeria, the prevalence of obesity ranged from 8.1 percent to 22.2 percent, with overweight people accounting for 20.3 percent to 35.1 percent of the population (Innocent et al., 2013). The main causes of obesity problems are reduced physical activity, eating too many high-energy foods, lifestyle changes, and an imbalance between components in the diet (Murizah andRobert, 2008).Obesity has been associated to a variety of health problems, including hypertension, dyslipidemia, diabetes, cardiovascular disease (CVD), sleep apnea, respiratory disorders, osteoarthritis, and cancer, all of which

increase the likelihood of all-cause mortality (Hossain*et al.*, 2007; King and Wofford, 2000; Mertens and Van Gaal, 2000). Infertility is the failure to conceive despite regular, unprotected sexual intercourse after one year (WHO, 2015). It is one of the most common reproductive disorders in the low-income countries. Although the prevalence of infertility globally is difficult to determine, it affects one in four couples of developing countries (WHO, 2015).According to Boivin (2007) infertility prevalence is approximately 9–15% globally, and it is increasing continually. All kinds of assisted reproduction methods are affected by obesity including the use of gonadotropins in ovulation induction and *in vitro* fertilization (IVF) (Rafique*et al*., 2016).Obesity has a negative effect on reproductive potential due to physiological alterations in the Hypothalamic-Pituitary-Ovarian (HPO) axis (Kulvinder et al., 2018).It causes various hormonal changes in the reproductive system, making it difficult to manage infertility (Balen and Rutherford, 2007).Obese women often have hyperinsulinemia, which is a known stimulus for increased androgen production (Rachonand Teede, 2010). These androgens get converted to estrogen in periphery due to the presence of excess Adipose tissue (AT) via negative feedback on the HPO axis, and thus affecting gonadotropin production (Junghem and Moley, 2010). It had been reported in two (2) Saudi studies that a one-third of women needing assisted reproductive techniques (ART) are obese (Al-Malki*et al*., 2003).

Several studies have reported the link between obesity and a variety of obstetric and gynecological problems, including ovulation, irregular menstruation, infertility, miscarriage, and negative consequences of pregnancy (Jungheim *et al.,* 2013). The reduction in obesity-related infertility is due to the effects of different steps of each stage, including the implantation stage, which begins with follicle selection, due to the imbalance between the hypothalamus-pituitary-ovarian axis (Talmor and Dunphy 2015; Ramlau-Hansen *et al*, 2007). Caillon *et al.* (2015) reported that basal luteinizing hormone (LH), follicle-stimulating hormone (FSH), and estradiol levels are lower in obese women with the decrease in ovarian reserve when compared to normal weight women. A similar study by Jain et al. (2007) observed that luteinizing hormone (LH) level was significantly lower in obesewomen with oligoamenorreicwhen compared with the control group. Pergola *et al.* (De Pergola *et al.,* 2006) also confirmed significant relation statistically to lower Follicle stimulating hormone (FSH), luteinizing hormone (LH), inhibin B, and estradiol levels in the early follicular phase of overweight and obese infertile women.There are several reports that obesity influences the risks of sub fertility. It is not only by causing decreased fertility but also causes suboptimal responses to Assisted Reproductive Techniques (ART). Understanding the interplay between obesity and infertility especially female reproductive hormones is vital because it will help educate the populace of its effect on fertility. Thus, this study was carried out to assess the effect of obesity on female reproductive hormones in obese infertile women in the Niger Delta Region of Nigeria.

MATERIALS AND METHODS

Study Area: This study was carried out in Rivers and Bayelsa States, Niger Delta Region of Nigeria.

The samples for the study were collected from women that attended the tertiary health Institutions, University teaching hospital in Port Harcourt (UPTH), Niger Delta University Teaching Hospital (NDUTH) Okolobiri,Federal Medical Centre (FMC) Yenagoa, Bayelsa State, and other Fertility clinics in the States.

Study Population: This cross-sectional study was carried out on 626 women comprising of 513 obese women and 113 not obese women who serve as control. The study was carried out between the period of May, 2014 and June, 2018. They were women that presented for hormone evaluation in these hospitals in the age range of 18-40years. They were grouped into 297 and 216 obese women with primary and secondary infertility respectively. Included in the study were women with BMI of >25.0kg/m^2 which was considered to be obese and without any known medical/hormonal problems and any medications (Oral contraceptives or steroid hormones in the last 3 months). Also included were women whose partners had normal semen count gotten from the medical history. Excluded were women with known medical/hormonal issues and on medications and whose spouse were known to have low semen count.

Sample Collection and Preparation: The samples for this study were collected from the subjects who attend fertility clinic. About 5.0 ml of a single non-fasting venous blood was collected from the ante-cubital region from the subjects via venipuncture between 8:00 AM and 11:00 AM, to control for diurnal variations (Feldman et al., 2002) and dispensed into plain containers. The blood sample was allowed to clot properly and centrifuged at 1500rpm for 5minutes using bench centrifuge, and serum separated and stored frozen at $-20^{\circ}C$ until the time of assay.The measurement of the hormones was done within seven (7) days of sample collection.

Ethical Clearance: The essence and details of the study were explained to the subjects (obese infertile and apparently healthy women) and consent gotten before sample collection. Institution ethical clearance was gotten before the sample collection was performed from the committee responsible for research.

Analytical Methods: Body Mass Index:Obesity status of the subjects were confirmed after collecting their height and weight to calculate their Body Mass Index (BMI).The BMI calculated by dividing the weight of the respondents by their height in meters squared; (Weight (kg) / Height(m^2)).The height was measured with a Standiometer (2015), the subject standing erect without shoes, leg forming a V-shape and the back against a wall. This was recorded in meters to one decimal point. The body weight was measured using a known-weight standardized weighing scale; the participant stood on the scale without shoes and the weight was recorded in kilogram to onedecimal point. Analysis of Hormones: The serum progesterone, estrogen, follicle stimulating hormone, luteinizing hormone and Prolactin assays were done using standard Enzyme Linked Immuno-Sorbent Assay (ELISA) method (Amballi*et al*., 2007). The principleis based on the solid phase enzyme linked Immunosorbent assay (30). The components of the ELISA kit were specifically designed to analyze estradiol, progesterone, inhibin, FSH and LH.

Statistical Analysis: The data from this study were subjected to statistical analysis using SPSS package, version 23 and

presented as mean ± standard deviation. The student's t-test was used to test difference in mean values. 95% confidence level ($P < 0.05$) were used and considered significant.

RESULTS

The 513 obese infertile participants that comprised of 297 Primary (1^o) Infertility and 216 Secondary (2^o)Infertility with an average age of 29.33 and 33.10 respectively had distribution and characteristics of demographic variables among the three study groups, control, 1^o Infertility and 2^o Infertility summarized in the table below. From the table 3.1, the number of subjects with Primary and Secondary infertility is 513, with more cases of Primary (1^o) infertility. The mean values of Age of subjects with Primary (1^o) and Secondary (2^o) infertile women were 29.33 and 33.10 respectively. Body Mass Index (BMI) of Primary (83.40) and Secondary (81.91) Infertility was higher than the control group (22.43). Results from the table 3.2 have shown that the serum concentration of inhibin and progesterone were statistically ($p<0.05$) reduced in the obese subjects with primary and secondary infertility as compared with the control group. The mean values of Follicle Stimulating Hormone (FSH), Luteinizing hormone (LH), Estrogen (E2) and prolactin (PRL) of the obese infertile women with primary and secondary infertility were significantly ($p<0.05$) elevated as compared with the control group. However, the serum concentration of all analyzed hormones was found to be higher in the secondary infertility compared with the primary infertility.

From the table 3.3, the percentage of abnormal (high and low) inhibin (32.90%), FSH (45.34%), LH (41.84%) and Prolactin (38.57%) levels in obese women with Primary (1^0) infertility were significantly ($p<0.05$) lower than the normal (64.10%, 54.66%, 58.16% and 61.43%) respectively. However, the percentage of abnormal Estrogen (68.62%) and Progesterone (73.14%) levels in obese women with Primary infertility were significantly ($p<0.05$) higher than the normal (31.38% and 26.86%) respectively. From the table 3.4, the percentage of abnormal (high and low) inhibin (34.08%), FSH (42.37%), LH (38.94%) and Prolactin (41.25%) levels in obese women with Second (2^0) infertility were significantly ($p<0.05$) lower than the normal (65.92%, 57.63%, 61.06% and 58.75%) respectively. However, the percentage of abnormal Estrogen (60.89%) and Progesterone (69.89%) levels in obese women with Second (2^0) infertility were significantly ($p<0.05$) higher than the normal (39.11% and 30.15%) respectively. From the table 3.5, the number of subjects suffering from Primary infertility is 297. The results revealed that the number of obese women with primary infertility who suffered from gonadal disorders for hypoestrogenisim 96(32.32%), Hyperestrogenism 202(68.01%), Hypergonadism 21(7.07%), Hypogonadism 83(27.95%), Hypergonadotropic 47(15.82%), Hypogonadotropic 31(10.44%) and Amenorrhoea 41(13.80%). All were statistically significant ($p<0.05$) compared with the normal. The study also found that obese women with primary infertility suffers from hyperestrogenism 202(68.01%) more than any other gonadal disorders. Whereas, obese women with primary infertility have the least gonadal disorder Hypergonadism 21(7.07%). Results from thetable 3.6 revealed that the number of subjects suffering fromSecondary infertility is 216. It showed that the number of obese women with Secondary infertility who suffered from gonadal disorders for hypoestrogenisim 53(24.54%), Hyperestrogenism 154(70.73%), Hypergonadism 14(6.48%), Hypogonadism 67(31.02%), Hypergonadotropic 42(19.90%), Hypogonadotropic 34 (15.74%) and Amenorrhoea 23(10.65%) and were statistically significant ($p<0.05$) compared with the normal. The study also found that obese women with secondary infertility suffers from hyperestrogenism 154(70.73%) more than any other gonadal disorders. Whereas, obese women with secondary infertility have the least gonadal disorder Hypergonadism 14(6.487%). Results from the table revealed that FSH, LH, E2, Progesterone and prolactin levels in obese women with primary infertility showed a positive correlation with Body Mass Index (BMI). Whereas, Inhibin shows a negative correlation with BMI. Results from the table revealed that FSH, LH, E2, Progesterone and prolactin levels in obese women with secondary infertility showed a positive correlation with Body Mass Index (BMI). Whereas, Inhibin shows a negative correlation with BMI.

DISCUSSION

The prevalence of obesity and overweight are increasing steadily and has been observed as a global phenomenon. Majority of these population are women of reproductive age, and many of the risk factors that are linked to obesity may also predispose them to infertility (Vahratian, 2009; Pasquali*et al.*, 2003). In the current study, the number of subjects with primary (1^o) infertility and secondary (2^o) infertility is 513, with more cases of primary (1^o) infertility as shown in (table 3.1). Many researchers have similarly reported that primary (1^o) infertility is more common among women than secondary (2^o) infertility (Goynumer et al., 2008; ESHRE, 2006). Certain factors such as age and Body Mass Index (BMI) have been documented to negatively influencing time to pregnancy (ESHRE, 2006). The present study showed that the mean values of the age of obese women with primary (1^o) infertility (29.33) were lower than women with secondary (2^o) infertility (33.10). The Body Mass Index of women with primary infertility (83.40) and secondary infertility (81.91) was higher than the control group (22.43) as shown in (table 3.1). My finding is in agreement with Raque-Bogdan et al. (2015) and Friis et al. (2002) who documented that fertility declines with increasing age and Body Mass index.

Infertility is often caused by imbalance of reproductive hormones which is determined by blood test. Ovulatory disorders are often characterized by low concentration of LH and FSH and high concentration of prolactin (Jawad*et al.*, 2015). The levels of reproductive hormones of obese infertile women with primary infertility and secondary infertility compared with the control group are shown in (Table 3.2). In the present study, serum progesterone and inhibin levels were significantly ($p<0.05$) lower in obese infertile women with primary (1^o) and secondary (2^o) infertility as compared with the control group. This reduction could be attributed to the obesity, which influences the Hypothalamic-Pituitary Gonadal axis (HPG) by increasing free estrogen levels because of elevated conversion of androgens to estrogens in adipose tissue. Elevated estrogen diminishes Gonadotropin Releasing Hormone (GnRH) by the negative response. Therefore, the affected HPG axis creates an ovulatory or abnormal cycle and hence lowers the levels of progesterone hormone (Valderhaug*et al.*, 2015). This finding confirms the report by De-pergola et al. (2006) and Farah and Zena (2019) who documented a significant decrease in inhibin B and

Table 3.1. Baseline Characteristics of the Studied I nfertile Obese Woman (BMI ≥25kg/m^2) in Niger Delta Region of Nigeria

Groups	Number	Age (Yrs)	Age Range	Height (m)	Weight (Kg)	BMI (kg/m^2)
	113	28.94	18-40	1.52	51.82	22.43
	297	29.33	18-40	1.49	83.40	37.57
	216	33.10	21-40	1.57	81.91	33.30
	513	30.95	1-40	1.51		30.66

Table 3.2. The Mean ± S.D of the Measured Parameter in the 1^o and 2^o Obese Infertile Women with respect to Control in Niger Delta Region of Nigeria

Paramters	1^0 Infertility Mean±S.D(Min-Max)	2^{0}Infertility Mean±S.D (Min-Max)	Control Mean±S.D (Min-Max)	P-value
Inhibin B	3.91±1.93 (1.02-19.44)	4.58±2.83 (1.02-19.44)	6.15±3.10 (1.96-11.73)	P<0.05
FSH	10.15± 30.37 (0.43-58.95)	11.56±4.01 (1.02-48.46)	7.55±3.91 (1.56-37.90)	P<0.05
LH	8.57± 3.04 (0.21-93.40)	7.28± 3.12 (0.95-71.05)	6.33± 3.06 (2.40-44.54)	P<0.05
E2	19.55±5.83(13.58-158.15)	0.71± 0.36 (0.53-1.17)	14.55±9.03(10.15-57.16)	P<0.05
PROG	0.55± 0.27(0.25-2.76)	0.61.04±0.29(0.31-2.38)	0.71± 0.36 (0.53-1.17)	P<0.05
PRL	23.53±9.15(0.95-105.66)	25.60±8.40(1.03-96.88)	15.76±6.89(1.55-4.81)	P<0.05

Table 3.3. The Percentage of Abnormal Inhibin B, FSH, LH, E2, Progesterone and Prolactin in Serum of Obese Women (BMI ≥25kg/m^2) with 1^0 Infertility in Niger Delta Region of Nigeria

Parameters	Normal (%)	High (%)	Low (%)	Total Abnormal (n=297) (%)	P-Value
Inhibin B	64.10	8.99	23.91	32.90	0.022*
FSH	54.66	20.50	24.84	45.34	0.016*
LH	58.16	20,01	21.83	41.34	0.017*
E2	31.38	60.46	8.16	68.62	0.000*
PROG	26.86	0.95	72.19	73.62	0.000*
PRL	61.43	37.15	1.42	38.57	0.018*

Table 3.4. The Percentage of Abnormal Inhibin B, FSH, LH, E_2, Progesterone and Prolactin in Serum of Obese Women (BMI ≥25kg/m^2) with 2^{0}infertility in Niger Delta Region of Nigeria

Parameters	Normal (%)	High (%)	Low (%)	Total Abnormal (n=297) (%)	P-Value
Inhibin B	65.92	6.86	27.22	34.08	0.023*
FSH	57.63	18.96	23.41	42.37	0.018*
LH	61.06	16.83	22.11	38.94	0.027*
E2	39.11	60.48	9.41	60.89	0.000*
PROG	30.15	0.23	68.62	69.05	0.000*
PRL	58.75	39.28	1.97	41.25	0.012*

Table 3.5. Frequency distribution of Disorders of Gonadal Function in Obese Woman (BMI ≥25kg/m^2) with 1^0 Infertility in Niger Delta Region of Nigeria

Endocrine Disorder	1^0 Infertility n=297 (% Abnormal)	2^{0}Infertility n=297 (% Normal)	P-value
Hypoestrogenism	96(32.32)	201(69.78)	P<0.05*
Hyperestrogenism	202(68.01)	95(31.98)	P<0.05*
Hypergonadism	21(7.07)	276(92.93)	P<0.05*
Hypogonadism	83(27.95)	214(72.05)	P<0.05*
Hypergonadotropic	47(15.82)	250(84.18)	P<0.05*
Hypogonadotropic	31(10.44)	266(89.56)	P<0.05*
Amenorrhoea	41(13.80)	256(86.19)	P<0.05*

Table 3.6: Frequency distribution of Disorders of Gonadal Function in Obese Woman (BMI ≥25kg/m^2) with 2^0 Infertility in Niger Delta Region of Nigeria

Endocrine Disorder	1^0 Infertility n=216 (% Abnormal)	2^{0}Infertility n=216 (% Normal)	P-value
Hypoestrogenism	53(24.54)	163(75.46)	P<0.05*
Hyperestrogenism	153(70.73)	63(29.16)	P<0.05*
Hypergonadism	14(6.48)	202(93.52)	P<0.05*
Hypogonadism	67(31.02)	149(68.98)	P<0.05*
Hypergonadotropic	42(19.90)	174(80.56)	P<0.05*
Hypogonadotropic	34(15.74)	182(84.26)	P<0.05*
Amenorrhoea	23(10.65)	193(87.35)	P<0.05*

Table 3.7. Correlation of Anthropometric Measures (BMI) with Hormones in Primary infertility (1^0) Obese Women

Parameter/Hormones	Inhibin B	FSH	LH	E2	PROG	PRL
BMI (kg/m^2)	-0.289	+0.313	+0.095	+0.397	+0.296	+0.432

Table 3.8. Correlation of Anthropometric Measures (BMI) with Hormones in Secondary Infertile Obese Women

Parameter/Hormones	Inhibin B	FSH	LH	E2	PROG	PRL
BMI (kg/m^2)	-0.312	+0.305	+0.056	+0.433	+0.379	+0.511

progesterone levels in obese women. Estrogen levels were significantly ($p<0.05$) higher in obese infertile women with primary (1^o) and secondary (2^o) infertility compared with the control group as shown in (table 3.2). The estrogen levels in women with secondary infertility was slightly higher than primary infertility women. The increase in estrogen may be due to obesity, which is associated with sex hormone imbalance and low level of sex hormone-binding globulin (SHBG) (39). The exact cause of infertility among the obese women appears to be the absence of long anovulation due to hyperandrogenism caused by obesity (Hymavathi*et al*., 2016). Follicle Stimulating Hormone (FSH), and Luteinizing hormone (LH) levels were also significantly ($p<0.05$) higher in obese infertile women with primary (1^o) infertility and secondary (2^o) infertility compared with the control group as shown in (table 3.2). The FSH and LH levels in women with secondary infertility were slightly higher than primary infertility women. Elevated serum LH and FSH concentrations are characteristic of women with polycysticovarian syndrome (Hendrik*set al*., 2007). The primary function of luteinizing hormone (LH) is the regulation ofproduction of androgen in the theca interna, while follicle stimulating hormone (FSH) is responsible for the regulation of the growth and maturation of ovarian follicles, and stimulation of the aromatization of androgens to estrogens (Robin *et al*., 2011). Elevated LH/FSH ratios have beenassociated with obesity (Beydoun*et al*., 2012). Prolactin levels were found to be significantly ($P<0.05$) higher in obese infertile women with primary (1^o) and secondary (2^o) infertility compared with the control group as shown in (table 3.2). However, women with secondary infertility had a slightly higher prolactin than primary infertility women. Hyperprolactinemia adversely affects fertility potential by impairing GnRH plasticity, and thereby impeding ovarian function (Hivre*et al*., 2014). A high prevalence of obesity has been significantly linked with hyperprolactinaemia, and the relationship between hyperprolactinemia and increased body weigh has been documented.

The findings from this study also observed that progesterone, inhibin, FSH, LH and prolactin levels were found to be higher in the secondary (2^o) infertility compared with the primary (1^o) infertility. In the present study, the prevalence of abnormal inhibin, FSH, LH, and Prolactin levels in subjects with Primary (1^0) infertility (32.90%, 45.34%, 41.84%, 38.57% respectively) and secondary (2^o)infertility (34.08%, 42.37%, 38.94%, 41.25% respectively) were significantly ($p<0.05$) lower compared to the normal subjects (64.10%, 54.66%, 58.16%, 61.43%) and (65.92%, 57.63%, 61.06% and 58.75%) respectively as shown in (table 3.3 and 3.4). However, the prevalence of abnormal estrogen (68.62%, 60.89%) and progesterone (73.14%, 69.89%) levels in subjects with Primary infertility and Secondary infertility respectively were significantly ($p<0.05$) higher compared with the normal subjects (31.38%, 26.86% and 39.11%, 30.15%) respectively. The present study also showed that theprevalence of gonadal disorder; hypoestrogenisim (32.32%), Hyperestrogenism (68.01%), Hypergonadism (7.07%), Hypogonadism (27.95%), Hypergonadotropic (15.82%), Hypogonadotropic (10.44%) and Amenorrhoea (13.80%) were statistically significant ($p<0.05$) in obese women with primary infertility when compared with the normal (69.78%, 31.98%, 92.93%, 72.05%, 84.18%, 89.56% and 86.19% respectively) as shown in (table 3.5). The gonadal disorders; hypoestrogenisim (24.54%), Hyperestrogenism (70.73%), Hypergonadism (6.48%), Hypogonadism (31.02%), Hypergonadotropic (19.90%), Hypogonadotropic (15.74%) and Amenorrhoea (10.65%) were statistically significant ($p<0.05$) in obese women with secondary infertility when compared with the normal subjects as shown in (table 3.6). However, hyperestrogenism was found to be more prevalent in subjects with both primary infertility (68.01%) and secondary infertility (70.73%) compared with other gonadal disorders. While the least prevalent gonadal disorder was Hypergonadism in subjects with primary infertility (7.07%) and secondary infertility (6.487%). The prevalence of hyperestrogenism in obese women with infertility is due to the fact that obesity is associated with high estrogen levels, as would be expected from androgen aromatization in adipocytes (Belanger et al., 2002). It may also be due to a sex hormone imbalance and low levels of sex hormone-binding globulin (SHBG), which can increase the target tissue's exposure to free estrogen. In addition, the results as shown in (Table 3.7 and table 3.8) demonstrated a correlation between infertility and Body Mass Index (BMI), which confirm that obesity is a sign that increases risk factors of infertility. The correlation between primary infertility, secondary infertility and BMI is positively correlated with luteinizing hormone (LH), follicle stimulating hormone (FSH), Estrogen (E2), progesterone and prolactin, whereas inhibin showed a negative correlation with Body Mass Index in both primary infertility and secondary infertility. These findings suggest either a disruption of estradiol-mediated adipose tissue formation or reduced sensitivity to estradiol. The positive correlation between prolactin levels and Body Mass Index in infertile obese women is not yet clear, but it could be due to the stimulation of lipogenesis or to the dysregulation of the dopaminergic tone of the central nervous system (Shibli-Rahhal and Schlechte, 2009).

CONCLUSION

On our study; the secondaryinfertility group had significantlyhigher BMIs, waist circumferences, hip circumferences, and waist-hip ratios than the primary infertility group. The primary and secondary infertility groups showed similar hormone levels. In the primary infertility group, no significant correlation was observed between hormonal factors and anthropometric measurements. By contrast, in the secondary infertility group, prolactin levels demonstrated significant positive correlations with body In the study, obese infertile women with primary and secondary infertility shows similar hormonal variations with respect to control group.

In both primary infertility and secondary infertility women, it showed a statistically significant increase in estrogen, LH, FSH, prolactin levels, and decreased progesterone and inhibin levels. All the analyzed hormones were slightly higher in the obese women with secondary infertility than primary infertility women.The study revealed that hyperestrogenism was found to be the most prevalent gonadal disorder in women with primary infertility and secondary infertility compared with other gonadal disorders, but slightly higher in secondary infertility than primary infertility women. The result demonstrated a correlation between infertility and Body Mass Index (BMI). The correlation between primary infertility, secondary infertility and BMI is positively correlated with LH, FSH, E2, progesterone and prolactin, whereas inhibin showed a negative correlation with BMI in both primary and secondary infertility women.

Acknowledgement

We sincerely appreciate the management of University of Port-Harcourt Teaching Hospital, Niger Delta University Teaching Hospital (NDUTH) Okolobiri, Federal Medical Centre (FMC) Yenagoa, Bayelsa State, and other Fertility clinics in the States. We also thank the management and staff of De-Integrated Medical Laboratory Port-Harcourt.

Conflict of Interest: We declare that there is no conflict of interest regarding to the publication of this article

ABBREVIATIONS

AT= Adipose Tissues
ART= Assisted Reproductive Techniques
BMI= Body Mass Index
CVDs= Cardiovascular Diseases
E2= Estrogen
FSH= Follicle Stimulating Hormones
GnRH= Gonadotropin Releasing Hormone
HPG=Hypothalamus Pituitary Gonadotropin
HPO= Hypothalamic-Pituitary-Ovarian axis
LH= Luteinizing Hormone
PRL= Prolactin
SHBG= Sex Hormone Binding Globulin
WHO= World Health Organisation

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