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Assessment of pituitary and ovarian function in women receiving modern hormonal contraception

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ABSTRACT

Hormonal contraceptives induce changes in the hypothalo-pituitary-ovarian loop and this eventually results in their therapeutic effects. The study aimed to investigate the level of changes induced in the pituitary gland and the ovary by hormonal contraceptive agents. The sample size was 200. One hundred and fifty (150) women desirous of contraception who met the World Health Organization medical eligibility criteria were enlisted and equally divided into 3 groups, those on: (A) combine oral contraceptive pill containing levonorgestrel 0.15mg and ethinyloestradiol 0.03mg, (B) injectables of progesterone-only containing depo medroxyprogesterone acetate 150mg, (C) long-acting and contraceptive made up of progesterone alone containing 68mg of etonogestrel (Implanon) while the remaining 50 served as control (D). All data collected were analyzed using SPSS 23. Hormonal contraceptive administration resulted in reduced secretion of the gonadotropins (follicle-stimulating hormone and luteinizing hormone) from the pituitary gland in all the hormonal contraceptives employed in the study ($p = 0.000$). There was also reduced serum levels of estrogens ($p = 0.001$) and progesterone ($p = 0.000$) when compared with the controls. There were increased serum levels of prolactin with the hormonal contraceptives ($p = 0.000$) when compared. With the administration of hormonal contraceptives, there were no luteinizing hormone peak and no rise in serum progesterone concentration in the luteal phase in the women on hormonal contraceptives. This reflects that ovarian follicular development was stalled and ovulation was inhibited. There was also an elevation in serum prolactin level.

1. Introduction

Exogenous hormonal agents are among one of the most frequently administered medications in women of reproductive age.[1] Hormone therapy is widely used throughout the world by women of all ages for a myriad of reasons, ranging from oral contraception for family planning to ovulation induction for infertility to hormone replacement therapy in menopause to adjuvant therapy of tumours of the breast and uterus.[1]

Hormonal contraceptives have been used by at least 500 million women alive today[2] and over the past two decades, the contraceptive prevalence has increased from 47.7 to 49.0 per cent.[3] Hormonal contraceptives utilize synthetic progesterone-only or in combination with an estrogen. These drugs can be administered via several routes and these include oral, parenteral, transdermal, subcutaneous, transvaginal, vaginal and intrauterine implants.[4,5]

Hormonal contraceptives (HCs) contain synthetic analogues of the reproductive hormones estrogen and

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progesterone. These formulations act synergistically to prevent ovulatory and can also affect the endometrial lining which is the basis of its utilization in the treatment of a variety of gynecologic issues.[4,6]

"The combined hormonal contraceptives (CHCs) which contain synthetic estrogen and progestin are among the most commonly prescribed and well-researched types of medication in use."[7,8] Irrespective of the route of administration both the progestins and estrogen in the CHCs inhibit the hypothalamic-pituitary-ovarian axis, which regulates the reproductive cycle. "Progestins prevent pregnancy by; preventing the luteinizing hormone (LH) surge, thus inhibiting ovulation, causes the cervical mucus to thicken, decreases fallopian tube motility, and suppresses endometrial receptivity.[1,4,9–11] Estrogens prevent pregnancy by suppressing the production of follicle-stimulating hormone (FSH), and stops the development of the dominant follicle.[9,12] Progestin is responsible for the majority of both contraceptive action and side effects; the addition of estrogen helps prevent irregular or unscheduled bleeding.[10,12] The administration of exogenous synthetic estrogens and progestogens does alter the body's endogenous production of these hormones.

Progesterone-only methods include pills, injectables, implants, and intrauterine devices (IUDs). Regardless of the type and as reported by authors,[1,11,13] they act by affecting the hypothalamic-pituitary-ovarian axis resulting in the inhibition of ovulation through the suppression of gonadotropin-releasing hormone. They also cause endometrial atrophy.

Medroxyprogesterone acetate (DMPA) injection is available as a 150 mg/mL intramuscular injection or a 104 mg/mL subcutaneous injection given every 12 to 13 weeks. Its mechanism of action is similar to that of progestin-only pills. Implants and IUDs containing progestin, are together classed as long-acting reversible contraception (LARC). The single-rod implant (Implanon), contains 68 mg of etonogestrel and it can last for three (3) years.[12]

The hormonal contraceptives inhibit the natural secretion of estrogen and progesterone, basically by blunting the changes in serum concentration of these hormones that occur necessarily in a normal menstrual cycle. Hormonal contraceptives manipulate the hypothalamic-pituitary-ovarian feedback loop, culminating in the stalling of ovarian follicular maturation and eventually, prevention of ovulation,[10,14,15] and as a consequence, there is nil rise in serum estrogen concentration that occurs in the first half of the normal menstrual cycle.[16] Since there is no ovulation, there is also no formation of the corpus luteum, consequently, there is no increase in serum concentration of progesterone seen in the second half of the menstrual cycle.[17] Therefore, women on hormonal contraceptives**,** exhibit lower serum estradiol and progesterone levels than do women having natural menstrual cycles,[9,18,19] and these persist even after the hormonal contraceptives have been discontinued.[20,21]

As reported above, hormonal drugs are exogenous synthetic analogues of estrogens and progesterone, they cause distortions in the endogenous productions of gonadotrophin-releasing hormone (GnRH), the gonadotrophins [follicle stimulating hormone (FSH) and luteinizing hormone (LH)], prolactin, estrogen and progesterone through the interplay of positive and negative feedback mechanisms. This study set out to investigate the level of disruption of the endogenous hormone levels by hormonal contraceptives.

2. Materials and Methods

2.1. Study area

The study was conducted at the Family Planning Clinics of the Diete-Koki Memorial Hospital, Opolo-Epie, Yenagoa, and the Niger Delta Teaching Hospital Okolobiri, all in Bayelsa State, Southern Nigeria. These tertiary health facilities are the most patronized of all the Bayelsa State Government-owned hospitals. Bayelsa state is located in the Niger Delta Region of Nigeria, bordering Delta and Rivers States.

2.2. Study population

A total of two hundred (200) women were used for the study. One hundred and fifty (150) women who expressed the desire to use the contraceptive methods listed in this study were recruited using a non-random sampling technique and they were allocated into three groups of fifty (50) each after they were assessed to have met the World Health Organization medical eligibility criteria; (A) those on combining oral contraceptive pill containing levonorgestrel 0.15mg and ethinyloestradiol 0.03mg (levofem by Dkt international), (B) those on injectables of progesterone-only containing depo medroxyprogesterone acetate 150mg (Depo-Provera® Contraceptive Injection Pharmacia USA), (C) those on long-acting contraceptive made up of progesterone alone containing 68mg of etonogestrel (Implanon®, Schering-Plough, New Jersey, USA) and the remainder (D) served as control, as these are women not on any form of contraception. Blood samples were collected from all the women that had used the contraceptives after 2 cycles in group A, after 2 months in groups (B) and (C) and from the control.

2.3. Inclusion and exclusion criteria

Inclusion criteria include ages between 18 and 40 years and not on any hormonal contraceptive drug. Excluded from the study were women <18 and >40 years, women with polycystic ovarian syndrome, evidence of kidney, liver, cardiovascular, or adrenal disease; uncontrolled

hypothyroidism; diabetes; hyperprolactinemia; severe arterial hypertension; patients with endometriosis, uterine fibroid with menorrhagia and pelvic inflammatory disease; history of bleeding dyscrasias, thromboembolic disease or thrombophilia; pregnancy and lactating women.

2.4. Sample collection and preparation

Samples were collected at the late follicular phase (between days 11 to day 13) for assessment of estrogen, FSH and LH while the samples for progesterone and prolactin was collected on day 21 of the menstrual cycle. Samples were collected following the standard operating procedure of vein puncture and transferred into plain containers. The collected sample was allowed to clot and centrifuged at 3500rpm for 5 minutes. The supernatants collected were immediately analyzed for the hormones essential to the study.

2.5. Laboratory analysis

Enzyme-Linked immunosorbent assay (ELISA) was the choice method for the hormonal assay. This method targets antigen capture in samples using a specific antibody and target molecule detection/quantitation using an enzyme reaction with its substrate. All the procedural steps as established in the standard operating procedures (SOP) of Accu-bind were strictly followed. The hormones analyzed include luteinizing hormone (LH), follicle stimulating hormone (FSH), estradiol, progesterone and prolactin.

2.6. Ethical approval and informed consent

The ethical approval was obtained from the Committee on Research and Ethics of the Niger Delta University, Wilberforce Island, Bayelsa State, Diete-Koki Memorial Hospital, Opolo-Epie, Yenagoa, Bayelsa State and Niger Delta Teaching Hospital Okolobiri, Bayelsa State.

2.7. Statistical analysis

Data analysis was performed using Microsoft Excel version 10 and IBM statistical package for social science (SPSS) version 23/. Statistical significant value was pegged at $p< 0.05$.

3. Result

Table 1 expressed the percentage allocations of 25% of subjects per group of the four groups.

Table 3 was multiple comparisons of the study hormones of the four groups. Luteinizing hormone (LH) concentration was significantly higher in the control when compared to the other three groups of pills, injectable and implants. Furthermore, the LH concentrations of the pills were significantly lower than that of the injectable and the implants. Similarly, the FSH concentration was higher in the control when compared to pills, injectables and implants. FSH level was lower when compared to the level for injectable and implants. Prolactin concentration was lower when compared to other groups. Similarly, implants prolactin concentration was lower when compared with injectable. The concentration of progesterone was higher in the control group when compared to other groups. On the contrary, the concentration of Progesterone in the pill was higher when compared with the injectable and implants. Estradiol concentration was higher in the control when compared with pills, injectables and implants.

4. Discussion

The study compared the mean concentrations of some endogenous reproductive hormones (LH, FSH, prolactin, progesterone and estradiol) concentration in women on hormonal contraceptives (pills, injectables and implants) to those, not on hormonal contraceptives (Tables 2 and 3). A comparison of the hormonal profiles within the various types of hormonal contraceptive groups and the control revealed a lot of hormonal disruptions (Table 3). The discourses on the impact of the various contraceptives on hormonal profiles are presented based on individual hormones below:

The concentration of LH significantly decreased in all the contraceptives when compared to the control. Similarly, the decrease of LH in the pills group was significantly more pronounced when compared to the injectables and implants. As stated by Bickerstaff and Kenny "the mechanism of action for the positive feedback effect of estrogen involves an increase in GnRH receptor concentrations. The high levels of circulating estrogen in the late follicular phase of the ovary act via the positive-feedback mechanism to generate a peri-ovulatory LH surge from the pituitary in the normal menstrual cycle. However, the use of the combined oral contraceptive pill, which artificially creates a constant serum estrogen level in the negative-feedback range, induces a correspondingly low level of gonadotrophin hormone release"[22] and as a consequence, there is no LH surge and this accounts for the low levels of LH compared with the controls. The progestins have been shown to prevent the LH surge and ovulation.[23] Similar findings were reported by other researchers.[10,19,24]

Luteinizing hormone plays a crucial role in stimulating the ovarian follicles in the ovary to produce oestradiol, initiate ovulation and is integral to the formation of the corpus luteum.[24] These attributes are essential for pregnancy to take place. The non-occurrence of the pre-ovulatory surge of LH resulting from the use of contraceptives is one of the mechanisms of action involved in achieving contraception. Measured by the concentrations of LH in this study, the pills group is adjudged to be more effective considering the level of deficit. This finding agrees with the reports of Carlstrom et al[9] and Balogh et al.[20]

Table 1: Representation of women on hormonal contraceptives and control group

Female categories (18-40 years)	**Number of samples (%)**
1. Control	50 (25%)
2. Pills	50 (25%)
3. Injectables	50 (25%)
4. Implants	50 (25%)
Total	200 (100%)

Table 2: Mean concentration of some reproductive hormones in various contraceptive applications

Parameters	**Controls** (mean± SD)	**Pills** (mean± SD)	**Injectable** (mean± SD)	**Implants** (mean± SD)
LH (IU/L)	9.25±0.170	4.85±1.11	8.14±2.10	7.38±2.30
FSH(IU/L)	8.31±0.19	6.91±0.63	8.16±2.93	5.12±1.82
Prolactin (ng/ml)	19.85±2.71	30.94±8.45	31.56±8.13	22.72±0.63
Progesterone (ng/ml)	2.85±0.67	1.76±0.78	0.98±0.91	1.10±0.63
Estradiol (pg/ml)	8.40±0.59	8.10±2.11	7.62±3.41	7.73±3.51

LH-Luteinizing hormones FSH-Follicle Stimulating Hormone

Table 3: Comparison of the mean concentrations of some reproductive hormones between study groups

Parameters	**Controls** (mean± SD)	**Pills** (mean± SD)	**Injectable** (mean± SD)	**Implants** (mean± SD)	**F value**	**P-value**
LH (IU/L)	9.25±0.170	4.85 ± 1.11^{a}	$8.14\pm2.10^{a,b}$	$7.38\pm2.30^{a,b}$	25.442	0.000
FSH (IU/L)	8.31±0.19	6.91 ± 0.63^{a}	8.16 ± 2.93^{b}	$5.12\pm1.82^{a,b,c}$	16.371	0.000
Prolactin(ng/ml)	19.85±2.71	30.94 ± 8.45^{a}	31.56 ± 8.13^{a}	$22.72\pm0.63^{b,c}$	13.523	0.000
Progesterone (ng/ml)	2.85±0.67	1.76 ± 0.78^{a}	$0.98\pm0.91^{a,b}$	$1.10\pm0.63^{a,b}$	26.717	0.000
Estradiol (pg/ml)	8.40±0.59	8.10 ± 2.11^{a}	7.62 ± 3.41^{b}	$7.73\pm3.51^{a,c}$	6.040	0.001

Significant concentration (<0.05) Control versus others=a
Pills versus others= b Injectableversus others = c

Follicle Stimulating Hormone (FSH) levels were significantly decreased in pills, and implants when compared to the plasma level of the controls. Similarly, the concentration of FSH was significantly lower in pills and injectable when compared to injectables. Injectables was not affected by FSH. The low FSH seen in pills and implants is majorly due to their effect in blunting the pulsatile release of GnRH from the hypothalamus ultimately causing an imbalance in the hypothalamus-pituitary ovarian loop.[15,16,23] Similar findings was reported by other authors.[9,20]

Serum prolactin concentrations were significantly elevated in pills and injectables when compared to that of the serum concentration in controls. In a similar vein, the concentration of prolactin was significantly lower in implants when compared to the pills and injectable groups. High levels of estrogen and progesterone inhibit the synthesis and release of prolactin from the anterior pituitary. The inhibition of ovulation by hormonal/progesterone-only contraceptives invariably means that the resulting low levels of estrogen and progesterone will cause increased secretion of prolactin. This finding has also been reported by other authors,[25,26] whilst other researchers reported no increment in serum prolactin levels.[10,27] The increased prolactin levels observed in this study corroborate the fact that it plays a role in the effectiveness of the hormonal contraceptives.[4,28,29]

Progesterone levels were significantly decreased in pills, injectable and implants when compared with the serum concentration of the control. Comparison between various hormonal methods showed a significant decrease of progesterone in injectables, and implants as compared to pills. This decrease is because the hormonal contraceptives effectively inhibited ovulation and as such there was no formation of the corpus luteum.[4,29] The stance of this study on decreased progesterone concentration is in accordance with reports by other authors.[9,11,14,19] Progesterone prepares the endometrium for an eventual pregnancy after ovulation. A sustained decrease thwarts ovulation and halts the preparation of the endometrial lining that creates a conducive environment for receiving the fertilized egg.

Estradiol (E2) levels were decreased in pills, injectables and implants when compared with the control. As noted by Bickerstaff and Kenny "production of oestrogen increases until it reaches the necessary threshold to exert a positive feedback effort on the hypothalamus and pituitary to cause the LH surge"[22] therefore in the normal menstrual cycle estrogen secretion is increased in the late proliferative phase. With the blunting of the pulsatile release of GnRH,

estrogen concentration would be low or reduced. In our study, this occurred with all the hormonal contraceptives and related finding was affirmed by other researchers. [10,30–33]

The above discourse clearly shows that contraceptive success is orchestrated by hormonal imbalances and the creation of an un-conducive environment for fertilization. Various contraceptives utilize arrays of alteration of the hypothalamus-pituitary-ovarian axis as the means in preventing pregnancies.

5. Conclusion

Different hormonal contraceptives induce and utilized several disruption mechanisms in the hormonal milieu in the hypothalamus-pituitary-ovarian loop either individually or synergistically to achieve their required objectives. The study has shown that during the administration of hormonal contraceptives FSH, LH, progesterone and estradiol serum concentrations were decreased. There were no luteinizing hormone peaks and no luteal phase levels of progesterone and this reflects that hormonal contraceptives as depicted in this study result in considerable suppression of pituitary-ovarian function.

6. Source of Funding

None.

7. Conflict of Interest

None.

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