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Chemical Pathology and study of Basic Hormonal Profile in Women with Polycystic Ovarian Disorder undergoing assisted reproduction treatment in South East Nigeria

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Abstract
Polycystic Ovarian Syndrome (PCOS) is a heterogeneous disorder frequently diagnosed in women attending fertility clinics in Nigeria. It is characterized by an ovulation, hyper androgenism and polycystic ovary, several factors have been associated with the path physiology of this disorder.
A prospective cross-sectional study was conducted in four selected fertility clinics in South-South part of Nigeria. The purposive sampling technique was used to recruit participants. A total of 198 women were recruited in this study, 126 (63.6%) women fulfilled the Rotterdam criteria for the diagnosis of PCOS, while 72 women (36.4%) without PCOS were included as control. Biochemical hyper-androgenism as described by the Rotterdam criteria was observed in 126(63.6%) of women with PCOS, Elevated Anti-mullerian hormone (AMH) 77(38.8%) and elevated Luteinizing hormone (LH) 76(38.4%) were observed in women with PCOS.
Follicle stimulating hormone (FSH) levels showed no difference in the two groups, this was not so for the other hormone of interest analysed, serum levels of Anti-mullerian hormone (AMH), Luteinizing hormone (LM) and Testosterone were all significantly higher in the PCOS group. The standard error of mean for AMH levels (4.420± 0.679) were significantly higher than the control group (1.462±0.241) and this showed a high significant levels of $p<0.001$.
The aim of the study was to look at Chemical Pathology Levels of Luteinizing Hormone (LH) Follicle Stimulating Hormone (FSH) Anti-mullerian Hormone (AMH) Testosterone in Polycystic Ovary Syndrome (PCOS) in Patients undergoing Assisted Reproduction Treatment.
Keywords: *Hormonal Profile, Polycystic Ovarian Syndrome, assisted reproduction*

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I. Introduction:

The ovary is the most well-studied tissue regarding AMH expression and function. The ovarian AMH expression is detected in granulosa cells of activated primordial follicles and is highest in preantral and small antral follicles. AMH expression is absent in follicular stages following follicle-stimulating hormone (FSH)-dependent selection, although h some expression remain in cumulus cells of pre-ovulatory follicles (1). Expression of the AMH-specific type II receptor (AMHR2) coincides with AMH expression, albeit that AMHR2 expression is also detected in the cells. Thus, AMH may affect both granulosa and the cell function. Studies using AMH knockout (AMHKO) mouse models revealed that AMH inhibits the primordial follicle recruitment and selection of follicles for dominance, two major steps in folliculogenesis. In the absence of AMH, more primordial follicles are recuited and FSH sensitivity was increased (2) furthermore, studies in the AMHKO mice suggest that AMH may act as an intraovarian inhibitor of follicular atresia (3).The effect of AMH on selection of follicles for dominance seems consistent across species. However, species difference may exist with regard to preantral follicular growth.in non-human primates, Xu and his colleagues showed that in vitro treatment of macaque secondary follicles with AMH during the first 3 weeks of culture advanced follicle antrum formation within a week, whereas treatment with an AMH neutralizing antibody delayed this process. Consistent with the increased growth, estradiol (E2) production of these secondary follicles was also increased. In contrast, in mice, AMH mostly acts as a survival factor for small preantral follicles. Importantly blocking AMH action in vitro through intra-ovarian infusion of an AMH neutralizing antibody for 4 days resulted in the growth of multiple antral follicles in most animals.in both the vitro and in vivo experiments, blocking AMH action in antral follicle increased E2 levels (4). These finding suggest that AMH may have a follicle stage- dependent effect on E2

production. Several studies have shown that AMH reduces FSH- induced aromatase (Cyp19al) expression and E2 Production in human antral follicles, and that in follicular fluid, an inverse relationship between AMH and E2 concentrations exists. Hence, it is suggested that AMH in humans acts as a gatekeeper of follicle growth by preventing premature selection and E2 production of small antral follicles. Species differences in FSH-dependency of preantral follicles may explain the observed differences in AMH effects on follicular growth. Although cultured macaque preantral follicles require FSH for survival These species differences should be taken into consideration when translating results to human, particularly when implication AMH in the pathophysiology of PCOS. In PCOS, where AMH level are increased, the AMH effect on follicular growth/survival and FSH sensitivity may be exacerbated leading to increased follicle number combined with follicular arrest. Several studies showed that the increased serum AMH level in PCOS is not only explained by the increased follicle number but also buy increased production per follicle compared to normal ovaries. In both follicular fluid and isolated granulosa cells obtained after controlled ovarian hyperstimulation for in vitro fertilization, AMH levels as well as AMH and AMHR2 expression were increased in sample from PCOS women compared to control women. Two additional studies in which the switch from a gonadotropin-independent to gonadotropin-dependent follicular stage was taken into account confirmed these finding. AMH expression and follicular fluid AMH levels decline in gonadotropin- dependent follicles in normal ovulatory women, whereas this did not occur in PCOS patients. Likewise, the coincided increase in E2 levels was absent in PCOS patient (11).This altered AMH expression may be the result of intrinsic granulosa cell dysregulation in PCOS. Both the cell and the granulosa cells of small antral follicles express higher levels of the luteinizing hormone (LH)receptor in PCOS women compared to normoovulatory women (12). Combined with the elevated LH level in PCOS, this leads to hyperstimulation of the cells and premature luteinization of granulosa cells. Interestingly, LH stimulation increased AMH expression in granulosa cells of PCOS women but not in normal ovulatory women (13). Treatment with 5-α- dihydrotestosterone yielded similar results. Furthermore, although estogens suppress AMH expression, mediated via estrogen receptor β, innormoovulatory women, this suppression was not observed in granulosa cells of anovulatory PCOS women (14). Combined these results suggest a failure in the downregulation of AMH expression in gonadotropin-dependent follicular stage in PCOS, which may contribute to a failure in follicular growth.

II. STUDY AREA

This study was carried out in selected fertility clinics across South-South and South- East Nigeria, and were independent private or public organization and were equipped with needed facilities and expertise for in-vitro fertilization treatment and PCOS diagnosis. Ethical clearance was obtained from the Federal University Teaching Hospital Ethnic and Research committee. Consent was obtained from study participation is voluntary and the confidentiality and privacy of all the participant was respected they were assured that there was no penalty for refusal or withdrawal from participant. Information on medications, menstrual and clinical history was collected by administrating a structured questionnaire, cross sectional study design was utilized in this study. The purposive sampling technique was utilized in this study, this involved identifying and selecting study participant who fit the inclusion criteria for this study and recruiting them based on their availability and willingness to participate in the study.

STUDY POPULATION

The study group was taken from patient receiving in-vitro fertilization treatment at the Assisted Reproductive Clinic of the Hospital

INCLUSIVE CRETERIA

The inclusive criteria as based on the definition of PCOS adopted at the joint consensus meeting of the American society for Reproduction Medicine and the European society of Human Reproduction and Embryology (ASRM/ESHRE), CRITERIA

1. Oligo-and /or an ovulation
2. Hyperandrogenism (clinical and /or biochemical) and
3. Polycystic ovaries with the exclusion of other aetiologies. (Rotterdam ESHRE/ASRM,2004).

EXCLUSION CRITERIA

This study excluded women who have any of the following condition:

1. History of chronic hypertension
2. Known autoimmune disorder
3. Women who did not give constant
4. Diabetes mellitus or treatment with oral glucocorticoids

5. Congentital adrenal hyperplasia.

SAMPLE SIZE DETERMINATION
The sample size was calculated using the Cochran formula for sample size determination.

Blood sample collection
Day 3 blood sample (5 mL) was withdrawn from each participant with minimal stasis from the antecubital vein using a dry, sterile disposable syringe and needle. The blood were dispensed into sterile plain tubes and 5ml tubes, The specimens were labeled with the subject's identification number, the sample in the sterile plain tubes were allowed to clot and centrifuged at 3000×g for 10 mins. The sera extracted were stored at -20^{o} C until processing.

Hormonal profile analysis
Day 3 serum FSH, LH, AMH, and Testosterone were measured using automated immunoassay analyzer (mini VIDAS Biomerieux, France).

Test principle
FSH, LH AMH and testosterone assay for VIDAS Biomerieux combines any enzyme immunoassay sandwich method and a final fluorescent detection and this combination of methods is known as Enzyme Linked Fluorescent Assay (ELFA). The solid phase Receptacle (SPR) serves as the solid phase as well as the pipetting device for assay. Reagent for these assay are ready-to-use and pre-dispensed in a sealed reagent.

Stored serum sample and reagents were brought to room temperature. Sample were transferred into the well containing alkaline phosphatase-labelled antibodies (conjugate).The sample/conjugate mixture were cycled in and out of the SPR several times to increase the reaction speed, then the antigen bind to antibodies coated on the SPR and to the conjugate forming a 'sandwich'. Unbound component are eliminated during the washing steps. The final detection steps require the substrate (4-Methyl- umbellifryl phosphate) to be cycled in and out of the SPR. The conjugate enzyme catalyzes the hydrolysis of this substrate into a fluorescent product (4-methyl- umbelliferone).The fluorescence of which is measured at 450nm. Then the intensity of the fluorescence is proportional to the concentration of antigen present in the sample. At the end of the assay, results were automatically calculated by the instrument in relation to the calibration curve stored in memory and then printed out.

Test procedure
Serum sample and reagent were brought to room temperature The external instrument barcode reader was used to scan the barcode for each lot of reagent kit. The sample were gently mixed and 100µL (200µL for AMH) of sample were pipetted into wells in the reagent strip. The SPR and strips for each sample was inserted into the VIDAS instrument making sure that the colour labels with the assay code on SPR and the reagent strip matches. The assay was initiated and the assay performed automatically 12 samples at a run by the VIDAS instrument

Quantitative determination of antibodies (Anti-TPO and Anti-Tg)
Thyroid auto antibodies were measure using automated fluorescent immunosorbet assay (FIA) analyser (mini VIDAS Biomerieux,France).Processing of samples were done according to manufacturer's instruction.

III. RESULTS

A total of 198 women attending fertility clinics of the hospital was recruited for this study and the study subjects was divided into two groups according to the Rotterdam diagnostic criteria for PCOS. 126(63.6%) women who met the Rotterdam criteria for diagnosis of PCOS were grouped as the PCOS group while, 74 (37.4%) apparently healthy women who had male factor infertility were recruited and grouped as the control group. The baseline demographic characteristic of the study of population is shown in Table 1. The study group age ranged from 26 to 53 yrs and there was no significant difference between the mean ages of the PCOS and the control group with mean ± SD which was 29.46±4.3 for PCOS group vs 30.44±3.1 for the control group. The mean body mass index (BMI) in the PCOS (25.37±5.9). group were significantly higher than mean BMI of the control group (24.29±3.8) on the other hand, the mean number of menstrual cycle/yrs in the PCOS group (5±4.63) were significantly lower than that of the control group (5±4.63 and 9±3.04).

Table 2 shows the hormonal profile analysis in the PCOS and control group, follicle stimulating hormone (FSH) levels showed no difference in the two groups, this was not so for the other hormone of interest analysed, serum levels of Anti-mullerian hormone (AMH), Luteinizing hormone (LM) and Testosterone were all significantly higher in the PCOS group. The standard error of mean for AMH levels (4.420± 0.679) were significantly higher than the control group (1.462±0.241) and this showed a high significant levels of $p<0.001$.

Table 3 shows the result of logistic regression analysis of hormones in women with PCOS, A positive and significant relationship was observed between AMH and testosterone (r= 0.838; P = 0.001). There was no correlation found between FSH levels and testosterone in women with PCOS, on the contrary, a negative correlation was observed between FSH and LH levels.

Table 1: Demographic characteristics of study subjects

Demographic Characteristics Value	PCOS (Mean ±SD)	Control (Mean ± SD)	P value
Mean age (years)	29.46±4.6	30.44±3.1	0.124
Number of menstrual cycle/year	5±4.63	9±3.04	0.001*
Body Mass index (kg/m^2)	25.37±5.9	24.29±3.8	
0.021*			

Value are mean± standard deviation (n=166) p< 0.05 = significant.

Table 2: Serum hormonal profile of study population.

PARAMETERS	PCOS (Mean ± SEM)	CONTROL (Mean ± SEM)	P-VALUE	*SIG
AMH	4.420± 0.679[a]	1.462±0.241	0.000	
FSH	13.33±2.501	11.96±1.625[b]	0.644	
LH	27.54± 1.978[a]	12.11±1.109	0.000	
TESTOSTERONE	2.574± 0.356[a]	0.75±0.207	0.000	

P<0.001= very high significant difference, P> 0.05 = no significant difference, [a]significant, [b]No significance LH: Luteinizing Hormone; FSH: Follicular Stimulating Hormone; AMH: Anti-Mullerian Hormone

Table 3: Correlation between hormones in women with PCOS

Hormone	Testosterone Correlation coefficient	LH coefficient
AMH	0.838[a]	0.86[b]
FSH	0.018[b]	-0.047[a]

[a]significantcorrelation, [b]Nosignificance correlation

IV. DISCUSSION

AMH has been found to contribute to the creation of androgen excess symptomatic of PCOS. AMH itself is synthesized by the follicles from primary-follicular stage to antral follicle stage but not by primordial follicles. AMH is responsible for the self-selected of monthly dominant follicle by preventing the recruitment of primordial follicles from the inherent ovarian and also by sending a negative feedback signal to pre antral follicles to become resistant to lose its FSH-receptors, this in turn also reduces AMH production (Kruszynska and Slowinska, 2017) There it can be said that AMH controls the rate at which primordial follicles in the ovarian pool is expended and also controls the selection of dominant follicle in an ovarian cycle (Kushnier et al., 2017) AMH is also associated with increased generation of many antral follicles arrest seen in (Kushnier et al., 2017). Regarding Antimullerian Hormone (AMH),this study confirms the result by cook et al (2002) who established a marked increase in the serum AMH level in women with PCOS, AMH concentrations were significantly elevated in women with PCOS compared to the control group in this present study, data from this study adds to existing information regarding AMH and its role in the etiology of PCOS by showing a remarkable association between AMH levels and IL-6 in women with PCOS (Figure 1),results in this study shows a strong linear regression slope different from that seen in the controls. From these results, one can deduce that the marked increase in AMH concentration may be directly linked to the altered cytokine profile and chronic low grade follicular inflammation detected in women with PCOS in this study. Also elevated level of AMH as detected in this study may not be surprising given that one may link this phenomenon to the fact that women with PCOS also present with early follicle excess, and it is known that AMH production corresponds to the number of follicles present in the ovaries at any given point during their reproductive years. This study revealed that elevated levels of AMH also correlated with elevated level of testosterone in PCOS, this agrees with a previous study of women with PCOS, in which AMH was positively correlated to androstenedione and testosterone levels in the serum of women with PCOS .This study propose that the association between AMH and androgen excess i.e testosterone excess may be as a result of the catalytic effect of follicles testosterone and other androgen on follicles inducing multiple growth of primordial follicles which in turn induces excess production of AMH in the respective developing follicles.in this study, results established underiable relationship between androgen and AMH which appears to be discriminatory to women with PCOS as this pathern was not observed in the healthy controls Furthermore, using data from our results, we also propose that elevated levels of AMH observed in this study may be directly linked to the absence of aromatase activity induced by FSH which is attributed to induce the follicular growth arrest in PCOS, elevated levels of AMH observed in this study agrees with the work of cook et al, who successfully correlated decreased serum FSH with elevated AMH in women with PCOS (17).

Concerning Luteinizing hormone (LH), in the study, it was observed that there may be a link between LH and elevated AMH levels in PCOS, this study, noticed a marked increase in LH levels more than 3 times in contrast to the LH levels measured for the control group. Till data there are unanswered questions about the correlation between LH and AMH, it is unclear if elevated levels of AMH directly affects the production of LH in women with PCOS (18). Not withstanding, recent studies have observed that elevation or amplification of pulsatile LH in the theca/granulosa cells of growing follicles can be the cause of follicules arrest of growing follicles typical in PCOS .in this regression analysis model noted a positive correlation between serum LH and AMH levels in apparently healthy fertile women. LH receptor specific for LH have been discovered mostly on the surfaces of small sized ovaries predominant in women with PCOS, these receptor were not found in larger ovaries of women without PCOS, this phenomenon could be the main physiological component responsible for follicular growth arrest (19).However, this explanation of follicular growth arrest is still debatable given that some studies have disputed this claim and recorded no evidence of association between follicular arrest elevated LH levels.on the other hand, some studies successfully established a statistical significance between AMH and LH levels, this study also found clear statistical difference between AMH and LH in women with PCOS whereby elevated AMH levels corresponded with LH elevations in PCOS its was independent of circulating FSH levels. The elevated levels of serum LH in PCOS group on this study can be said to have been a factor in increasing AMH levels. However, in this study no significant correlation was observe between follicular fluid AMH and serum LH levels in both the PCOS and control group. Similarly, there was no significant association found between AMH levels and elevated levels of LH in this study. Although our statistical logistical regression analysis found no association between AMH levels and LH, it is still probable that there could be a link found between these two biological entities in a larger study. Nonetheless, this study noted a statistically significant elevation in AMH levels in the PCOS group. Indicating that the elevations in serum AMH previously recorded in other studies may not be only as a result of multie AMH producing small follicles seen in PCOS. Irrespective of the relationship between AMH and other circulating hormones. This study observe that women with PCOS have an altered AMH levels different from normal ovulating women and this could be admissible physiologically.

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