Original Article

Correlation of Anti-mullerian Hormone (AMH) and Thyroid Stimulating Hormone (TSH) in Patients with Polycystic Ovary Syndrome

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ABSTRACT

Background: PreeclampsiaPolycystic ovary syndrome (PCOS) is the commonest endocrine abnormality in women of reproductive age. The prevalence of PCOS is variable between different populations and ranged from 6% to 13%. It is a syndrome of ovarian dysfunction (oligo-ovulation or anovulation) along with the cardinal features of hyperandrogenism or hyperandrogenemia and Polycystic Ovary morphology (PCO) by ultrasound. Thyroid dysfunction is associated with Polycystic Ovary Syndrome (PCOS) in women of reproductive age. Anti-Müllerian hormone (AMH), a known biomarker of ovarian function, may be affected by impaired thyroid function; however, the relationship between AMH and thyroid stimulating hormone has not been elucidated.

Aim: To establish a correlation between Thyroid Stimulating Hormone and Anti-Mullerian Hormone in patients with PCOS.

Methods: In this study, we recruited 120 Egyptian females diagnosed as PCOS by Rotterdam's criteria from October 2018 to March 2019. Patients aged 20-40 years were included in the study. Patients with known chronic illness or autoimmune diseases as systemic lupus erythematosus (SLE), rheumatoid arthritis were excluded from the study. Patients who underwent thyroid operations, e.g. total or partial thyroidectomy, were also excluded. We assessed patients' Age, BMI, FSH, LH, E2, TSH, FT3, FT4, AMH as independent variables. Patients were monitored by transvaginal ultrasound examination to evaluate the antral follicular count.

Results: AMH levels were positively correlated with TSH level (p-value 0.014), however; it is weak correlation. There was a strong correlation between AMH concentration and AFC (p-value<0.001).

Conclusion: There is a weak positive correlation between AMH and TSH in women of reproductive age with PCOS.

Key Words: Anti-Mullerian hormone (AMH), polycystic Ovary Syndrome (PCOS), thyroid stimulating hormone (TSH).

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INTRODUCTION

The most prevalent cause of anovulation, infertility, and hyperandrogenism in women is polycystic ovary syndrome. In fact, 5–10% of reproductive-age women are impacted[1].

Many women are completely ignorant of their vulnerability. However, there is no universal agreement on the classification of the disease, or even what a polycystic ovary is. It's encouraging to see a trend away from the term "polycystic ovarian illness" and toward the more widely recognized "polycystic ovary syndrome." This supports the notion of PCOS as a combination of indications, symptoms, and endocrine problems, as well as the condition's heterogeneity[2].

PCOS is a condition of ovarian dysfunction (oligo-ovulation or anovulation) with the cardinal symptoms of hyperandrogenism or hyperandrogenemia and Polycystic Ovary morphology (PCO) by ultrasonography, according to the Rotterdam Consensus Workshop (2003). As a result, no one diagnostic criteria (such as hyperandrogenism or PCO) is adequate for clinical diagnosis of PCOS. Despite extensive attempts to discover the origin, the pathophysiology of PCOS remains unknown, however evidence is mounting suggesting the primary abnormalities of PCOS are largely ovarian in nature. Hyperandrogenism, in particular, is becoming more like the 'heart' of PCOS, with the initial effect being disrupted folliculogenesis[3].

Theca Cells are fundamentally hyperactive for genetic and/or epigenetic causes, resulting in an intra-ovarian androgen excess. Given the relevance of androgens in tiny follicle formation, intra-ovarian hyperandrogenism has been identified as the primary cause of the disease's follicular excess[4,5]. This follicular surplus has a significant impact on oligo-anovulation, via processes that are

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unknown but seem to be independent of any systemic hormonal effect[6,7]. Indeed, the negative association between the frequency of tiny (2–5mm) and big (6–9mm) antral follicles in PCO vs. normal ovaries implies that the former inhibits the latter through follicle-to-follicle interactions that have yet to be understood but would be worsened in PCOS[8]. The truth is that the tinier follicles there are, the more growth arrest occurs, and the chances of ovulation decrease. Surprisingly, serum Anti-Mullerian hormone (AMH) in PCOS is linked to the number of (2–5mm) follicles found by ultrasound, but not to the number of (6–9mm) follicles[9].

This is to be anticipated, since this is the follicle type in which the Granulosa Cells express the most AMH. As a consequence, we proposed that follicular stoppage may be caused by an increased AMH tone in the selected follicles' microenvironment[10]. It's not only an issue of follicle quantity when it comes to PCOS's disrupted folliculogenesis. Aside from this excess, we believe that androgens, Follicle stimulating hormone (FSH), AMH, and Estradiol all play a role in Granulosa cell dysregulation (E2)[11]. Antimullerian Hormone (AMH) is a dimeric glycoprotein that was isolated and refined in (1989) as a Mullerian inhibitory agent[12,13]. Granular cells release AMH only from primary pre-antral follicles and tiny antral follicles (size 4–6mm). AMH production decreases with each step of follicle development, eventually becoming undetectable in follicles (8mm) AMH levels have been related to the amount of tiny follicles and consequently the ovarian reserve[14].By restricting the reaction to Follicle stimulating hormone (FSH), AMH avoids an excess of primordial follicle recruitment, preventing follicle selection and causing follicular arrest in the small antral phase[15]. Without AMH, primitive follicles will recruit considerably more quickly, resulting in an influx of expanding follicles until the main follicle at a younger age exhausts. A greater level of AMH in PCOS patients may indicate the severity of the condition, and it is the most accurate diagnostic for PCOS diagnosis compared to other hormones. AMH is now widely regarded as the most reliable marker for ovarian follicle development and function, and it is considered a gold standard for PCOS diagnosis[16-19].

Thyroid dysfunction is a highly frequent endocrine disease in females of reproductive age, and hypothyroidism may be caused by monthly abnormalities and an-ovulation. Thyroid disease may affect follicular development and maturation, according to findings relating to irregular menstrual cycles and an-ovulation in hypothyroidism. Hypothyroidism is known to induce a reverse phenotype equivalent to PCOS by reducing ovarian reserves, causing early attrition in the pool of ovarian follicles. Thyroid Stimulating Hormone (TSH) levels may be influenced by hypothyroidism[20].

The Aim is to determine correlation between Anti-Mullerian Hormone (which serves as a biomarker for ovarian reserve) Thyroid Stimulating Hormone, Luteinizing Hormone and Antral follicular count.

PATIENTS AND METHODS

This study was conducted at Cairo university Obstetrics and Gynecology hospital from October 2018 to March 2019. The study included 120 patients diagnosed as PCOS based on Rotterdam criteria, ESHRE/ASRM (2003), by the presence of two of the following conditions: oligo-ovulation or anovulation, hyperandrogenemia, hyperandrogenism and polycystic ovaries detected by Ultrasonography with the presence of twelve or more follicles measuring 2-9mm in diameter, and/or at least one enlarged (>10cm^3) ovary.

Patients aged 20-40 years were included in the study. Patients with known chronic illness or autoimmune diseases as systemic lupus erythematosus (SLE), rheumatoid arthritis were excluded from the study. Patients who underwent thyroid operations, e.g. total or partial thyroidectomy, were also excluded.

On the 2nd day of the menstrual cycle (either spontaneous or induced progesterone- in-oil injection in amenorrhoeic patients), serum and blood samples were collected for FSH, LH, E2, AMH, as well as TSH, FT3, FT4.

On the 3rd day of the menstrual cycle, patients were monitored by transvaginal ultrasound examination to evaluate the antral follicular count. Ultrasound was done using MEDISON SONOACE X6, Transvaginal probe[2D] G50/P90/105dB, FA6/FS12, MI12/TIb0.3. All the collected data were revised for completeness and logical consistency. Pre- coded data was entered on the computer using Microsoft Office Excel Software Program (2010).

Pre-coded data was then transferred and entered into the Statistical Package of Social Science Software program, version 21 (SPSS) to be statistically analyzed.

For quantitative variables (Using Kolmogorov-smirnov *Z* test) variables that were normally distributed were described as mean and SD. While variables that were not normally distributed were described as median, IQR (interquartile range).

For qualitative variables, they were described as frequency and percentage. Spearman Correlations done between not normally distributed quantitative variables, where p value of significant correlation if $p<0.05$. r value for the strength of the correlation $r=0$ no correlation, 0-<0.25 weak correlation, 0.25-0.75 moderate, >0.75 strong correlation.

RESULTS

This study was conducted at Cairo university Obstetrics and Gynecology hospital from October 2018 to March 2019. The study included 120 patients with PCOS as one group to determine correlation between Anti-Mullerian Hormone, Thyroid Stimulating Hormone, Luteinizing Hormone and Antral follicular count (Table 1).

Table 1: Baseline demographic data of the study participants:

Variable	Value
Age	30±4.6
Weight	67.5±12.5
Height	1.6±0.1
BMI	26.9±5.1

The analyzed data were collected and tabulated and the following results were obtained (Table 2).

Table 2: Hirsutism percentage among the study participants:

Variable		Value
Hirsutism	Hirsute *n*(%)	81(67.5)
	Non-Hirsute *n*(%)	39(32.5)

There were 81 patients (67.5%) with Hirsutism while 39 patients (32.5%) without Hirsutism (Table 3).

Table 3: Menstrual pattern among the study participants:

Variable		Value
MenstrualRegularity	Regular *n*(%)	36(30%)
	Irregular *n*(%)	72(60%)
	Amenorrhea *n*(%)	12(10%)

There were 36 patients (30%) with regular cycles, 72 patients (60%) with irregular Cycles and 12 patients (10%) with amenorrhea (Table 4).

Table 4: FSH, LH and Estradiol among the study participants:

Variable	Value
FSH* (mean±SD)	6.46±4.6
LH** median (IQR)	6.20(4.95-8)
E2** median (IQR)	40.00(31.95-52)

The Mean FSH was 6.46±1.6 among the study group The median value for LH was 6.2 ranging from 4.95-8 among the study group, The median value foe E2 was 40.00 ranging from 31.95-52 among the study group (Table 5).

Table 5: Thyroid profile among the study participants:

Variable	Value
TSH** median (IQR)	2.310(1.44-3.6)
FT3** median (IQR)	0.284(0.235-0.355)
FT4* (mean±SD)	1.27±0.95

The Median value for TSH was 2.310, ranging from 1.44-3.6 among the study group The Mean FT4 was 1.27±0.95 among the study group (Table 6).

Table 6: AMH and AFC among the study participants:

Variable	Value
AMH** median (IQR)	5.32(4.41-7.67)
AFC** median (IQR)	26.5(21.5-39)

The Median Value for AMH was 5.32, ranging from 4.41-7.67 among the study group. The Median value for AFC was 26.5, ranging from 21.5-39 among the study group (Table 7).

Table 7: Correlation between AMH and LH, TSH, AFC among the study participants:

		LH	TSH	AFC
AMH	*r**	0.013	0.223***	0.838****
	P value	0.887**	0.014	<0.001

There is no correlation between AMH hormone and LH (*p*-value 0.887) among the study group. There is weak correlation between AMH and TSH (*p*-value 0.014) among the study group. There is strong correlation between AMH and AFC (*p*-value <0.001) among the study group (Figures 1-3).

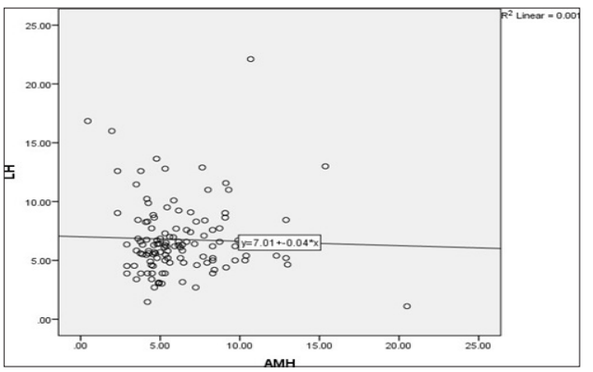

Fig.1: Correlation between AMH and LH.

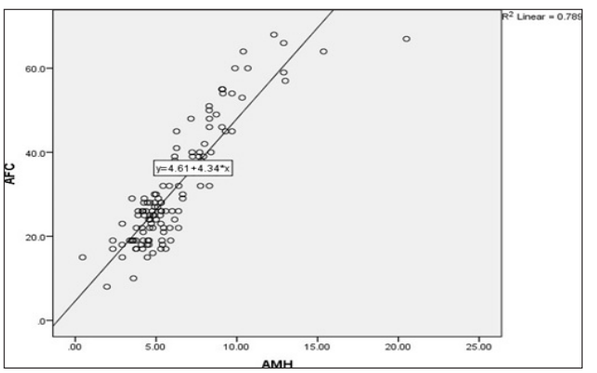

Fig. 2: Correlation between AMH and TSH.

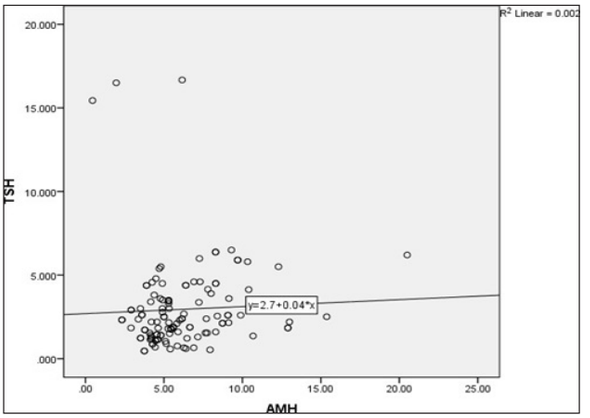

Fig. 3: Correlation between AMH and AFC.

DISCUSSION

The correlation between AMH and thyroid function has remained unresolved. We in this study attempted to predict out correlation between thyroid function, and AMH in respect to PCOS. Not Many studies have been made in this field, so limited data are available to compare our results with.

In this context it is important to note that this study was performed on women diagnosed as Polycystic Ovary Syndrome by Rotterdam criteria, when two out of the following three features are present: oligo ovulation and/or anovulation, clinical and/or biochemical signs of hyperandrogenism, and polycystic ovaries on ultrasound examination (the presence of 12 or more follicles measuring 2–9mm in diameter).

This study was conducted at Cairo university Obstetrics and Gynecology hospital from October 2018 to March 2019. The study included 120 patients with PCOS as one group to determine correlation between Anti-Mullerian Hormone, Thyroid Stimulating Hormone, as well as Antral follicular count.

The results of this study showed a weak positive correlation between Thyroid stimulating hormone and AMH concentration (*p*-value 0.014) which disagrees with other studies. A study by Samal Hakeem *et al.,* Jaf[21]., (2018) in which Eighty women were included between February 2015 to June 2017, divided to four groups, control group, Polycystic Ovary Syndrome group, Hypothyroid group, and Polycystic Ovary Syndrome + hypothyroidism group, the women age was ranged between (26-40) years. The study showed a negative correlation between AMH and TSH ($p \leq 0.001$) among the Polycystic Ovary Syndrome group which disagrees with our study. This result also disagrees with A. Weghofer *et al.*,[22]., (2016) which their studies investigated 255 infertile women between July 2009 and march 2014 to determine whether thyroid function, affects Functional Ovarian Reserve (FOR) within what is considered normal thyroid function (TSH, 0.4-4.5μIU/mL) by assessing AMH levels in reference to TSH levels, stratified for TSH < or ≥3.0μIU/mL This study showed that Women with TSH <3.0μIU/mL presented with significantly higher AMH compared to those with TSH ≥3.0μIU/ml ($P = 0.03$), which means the increase in TSH is reflected by a decrease in AMH and consequently the functional ovarian reserve.

Another study by Moh'd Nizar Battikhi[23]., (2018) showed negative correlation between AMH and TSH and also between AMH and patient's age, in which 36 females were recruited between (2016) and (2017) divided into three age group where AMH, TSH and FT4 where assessed as independent variables. Significant negative correlation was found between AMH and TSH ($p<0.004$), also negative correlation was observed between AMH and age ($p<0.005$).

Another study by Kuroda *et al.,*[24]., (2014) in which 67 Japanese females aged 30-39 were recruited between (2012) and (2013). Age, BMI and AMH, prolactin, TSH and FT4 levels of all study participants were assessed as independent variables. Results showed that both thyroid-stimulating hormone (TSH) levels and patient age were negatively correlated with AMH levels ($p = 0.036$ and 0.003 respectively) which also disagrees with our study.

Another study by Michalakis *et al.,*[25]., (2011) also reported diminished ovarian reserve and decrease in AMH in patients with elevated serum TSH levels, which also disagrees with the results of our study. In addition, A Study by L. Meng *et al.,*[26]., (2017) on female rats, reported that AMH level was decreased with a high TSH level and both AMH and TSH had an inverse relationship. This association could be belonging to, TSH negatively effects on Follicles Ovarian Reserve (FOR) leading to significant decrease in AMH.

CONCLUSION

There is strong positive correlation between AMH and Antral follicular count which reflects the functional ovarian reserve. There is weak positive correlation between AMH and TSH in women with polycystic ovary syndrome. There is a negative correlation between AMH and LH in women with Polycystic Ovary Syndrome. Considering the pilot nature of this study and the results of other studies, further studies with much larger study populations are needed to verify its findings.

CONFLICT OF INTERESTS

There is no conflict of interests.

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