Relation of Lutenizing hormone and Antimullerian hormone among PCOS patients

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DEDICATION

I dedicate this work to my beloved parents who were there for me with their support and encouragement, to their loving tears and beautiful smiles that without them I couldn't be here.

Acknowledgement

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Abstract

Background:

Polycystic ovarian syndrome (PCOS) is known to be one of the most prevalent endocrine disorders affecting 15–20% of reproductive age women and is a primary cause of infertility.

Aims:

The aim of the present study was to investigate the relationship between serum LH and AMH levels in Iraqi women with PCOS.

Patients and method :

A total of 25 patients diagnosed as PCOS were enrolled in the study and blood samples were taken for laboratory evaluation of hormones.

Results:

PCOS patients had an average age of 24 years with majority of them having more than one parity. About 72% of them had secondary infertility and 48% were overweight. LH median readings were 14.4 (IU/L) , FSH median readings were 10.59 (IU/L) and AMH median readings were 8.6 (ng/ml).

Conclusions:

LH and AMH are positively related in PCOS patients.

Introduction:

Polycystic ovary syndrome (PCOS) is a set of symptoms due to elevated androgens in females[1]. Signs and symptoms of PCOS include irregular or no menstrual periods, heavy periods, excess body and facial hair, acne, pelvic pain, difficulty getting pregnant, and patches of thick, darker, velvety skin[2]. Associated conditions include type 2 diabetes, obesity, obstructive sleep apnea, heart disease, mood disorders, and endometrial cancer[1].

PCOS is due to a combination of genetic and environmental factors[3]. Risk factors include obesity, a lack of physical exercise, and a family history of someone with the condition. Diagnosis is based on two of the following three findings: no ovulation, high androgen levels, and ovarian cysts. Cysts may be detectable by ultrasound[4]. Other conditions that produce similar symptoms include adrenal hyperplasia, hypothyroidism, and high blood levels of prolactin[4].

PCOS has no cure. Treatment may involve lifestyle changes such as weight loss and exercise.[5] Birth control pills may help with improving the regularity of periods, excess hair growth, and acne.[6] Metformin and anti-androgens may also help. Other typical acne treatments and hair removal techniques may be used.[6] Efforts to improve fertility include weight loss, clomiphene, or metformin. In vitro fertilization is used by some in whom other measures are not effective.

PCOS is the most common endocrine disorder among women between the ages of 18 and 44.[7] It affects approximately 2% to 20% of this age group depending on how it is defined. When someone is infertile due to lack of ovulation, PCOS is the most common cause.

Signs and symptoms

Common signs and symptoms of PCOS include the following:

- Menstrual disorders: PCOS mostly produces oligomenorrhea or amenorrhea, but other types of menstrual disorders may also occur.[7]
- Infertility: This generally results directly from chronic anovulation (lack of ovulation).[7]
- High levels of masculinizing hormones: Known as hyperandrogenism, the most common signs are acne and hirsutism(male pattern of hair growth, such as on the chin or chest), but it may produce hypermenorrhea (heavy and prolonged menstrual periods), androgenic alopecia (increased hair thinning or diffuse hair loss), or other symptoms.[7]
- Metabolic syndrome: This appears as a tendency towards central obesity and other symptoms associated with insulin resistance.[7] Serum insulin, insulin resistance, and homocysteine levels are higher in women with PCOS.[8]

Women with PCOS tend to have central obesity, but studies are conflicting as to whether visceral and subcutaneous abdominal fat is increased, unchanged, or decreased in women with PCOS relative to reproductively normal women with the same body mass index. In any case, androgens, such as testosterone, androstanolone (dihydrotestosterone), and nandrolone decanoate, have been found to increase visceral fat deposition in both female animals and women.[9]

Pathogenesis:

Polycystic ovaries develop when the ovaries are stimulated to produce excessive amounts of androgenic hormones, in particular testosterone, by either one or a combination of the following (almost certainly combined with genetic susceptibility[10]).

- the release of excessive luteinizing hormone (LH) by the anterior pituitary gland
- through high levels of insulin in the blood (hyperinsulinaemia) in women whose ovaries are sensitive to this stimulus

The syndrome acquired its most widely used name due to the common sign on ultrasound examination of multiple (poly) ovarian cysts. These "cysts" are actually immature follicles not cysts. The follicles have developed from primordial follicles, but the development has stopped at an early antral stage due to the disturbed ovarian function. The follicles may be oriented along the ovarian periphery, appearing as a 'string of pearls' on ultrasound examination.

Women with PCOS experience an increased frequency of hypothalamic GnRH pulses, which in turn results in an increase in the LH/FSH ratio.[11]

A majority of women with PCOS have insulin resistance and/or are obese. Their elevated insulin levels contribute to or cause the abnormalities seen in the hypothalamic-pituitary-ovarian axis that lead to PCOS. Hyperinsulinemia increases GnRH pulse frequency, LH over FSH dominance, increased ovarian androgen production, decreased follicular maturation, and decreased SHBG binding. Furthermore, excessive insulin, acting through its cognate receptor in the presence of component cAMP signalling, upregulates 17α-hydroxylase activity via PI3K, 17α-hydroxylase activity being responsible for synthesising androgen precursors. The combined effects of hyperinsulinemia contribute to an increased risk of

PCOS.[12] Insulin resistance is a common finding among women with a normal weight as well as overweight women. [7]

Adipose tissue possesses aromatase, an enzyme that converts androstenedione to estrone and testosterone to estradiol. The excess of adipose tissue in obese women creates the paradox of having both excess androgens (which are responsible for hirsutism and virilization) and estrogens (which inhibits FSH via negative feedback).[13]

PCOS may be associated with chronic inflammation, with several investigators correlating inflammatory mediators with anovulation and other PCOS symptoms. [47][48] Similarly, there seems to be a relation between PCOS and increased level of oxidative stress.[14]

It has previously been suggested that the excessive androgen production in PCOS could be caused by a decreased serum level of IGFBP-1, in turn increasing the level of free IGF-I, which stimulates ovarian androgen production, but recent data concludes this mechanism to be unlikely.[15]

PCOS has also been associated with a specific FMR1 sub-genotype. The research suggests that women with heterozygous-normal/low FMR1 have polycystic-like symptoms of excessive follicle-activity and hyperactive ovarian function.[16]

Diagnosis:

Not everyone with PCOS has polycystic ovaries (PCO), nor does everyone with ovarian cysts have PCOS; although a pelvic ultrasound is a major diagnostic tool, it is not the only one.The diagnosis is straightforward using the Rotterdam criteria, even when the syndrome is associated with a wide range of symptoms.

Rotterdam:

In 2003 a consensus workshop sponsored by ESHRE/ASRM in Rotterdam indicated PCOS to be

present if any 2 out of 3 criteria are met, in the absence of other entities that might cause these findings[17].

1. oligoovulation and/or anovulation
2. excess androgen activity
3. polycystic ovaries (by gynecologic ultrasound)

The Rotterdam definition is wider, including many more women, the most notable ones being women without androgen excess. Critics say that findings obtained from the study of women with androgen excess cannot necessarily be extrapolated to women without androgen excess.[18]

Androgen Excess PCOS Society

In 2006, the Androgen Excess PCOS Society suggested a tightening of the diagnostic criteria to all of the following:[7]

1. excess androgen activity
2. oligoovulation/anovulation and/or polycystic ovaries
3. exclusion of other entities that would cause excess androgen activity

Differential diagnosis:

Other causes of irregular or absent menstruation and hirsutism, such as hypothyroidism, congenital adrenal hyperplasia (21-hydroxylase deficiency), Cushing's syndrome, hyperprolactinemia, androgen secreting neoplasms, and other pituitary or adrenal disorders, should be investigated.

Management

The primary treatments for PCOS include: lifestyle changes and medications.[19]

Goals of treatment may be considered under four categories:

- Lowering of insulin resistance levels
- Restoration of fertility
- Treatment of hirsutism or acne
- Restoration of regular menstruation, and prevention of endometrial hyperplasia and endometrial cancer

In each of these areas, there is considerable debate as to the optimal treatment. One of the major reasons for this is the lack of large-scale clinical trials comparing different treatments. Smaller trials tend to be less reliable and hence may produce conflicting results.

General interventions that help to reduce weight or insulin resistance can be beneficial for all these aims, because they address what is believed to be the underlying cause.

As PCOS appears to cause significant emotional distress, appropriate support may be useful.[20]

Prognosis

A diagnosis of PCOS suggests an increased risk of the following:

- Endometrial hyperplasia and endometrial cancer (cancer of the uterine lining) are possible, due to overaccumulation of uterine lining, and also lack of progesterone resulting in prolonged stimulation of uterine cells by estrogen.[21]
- Insulin resistance/Type II diabetes. A review published in 2010 concluded that women with PCOS have an elevated prevalence of insulin resistance and type II diabetes, even when controlling for body mass index (BMI).[21] PCOS also

makes a woman, particularly if obese, prone to gestational diabetes.

- High blood pressure, in particular if obese or during pregnancy.
- Depression and anxiety[7].
- Dyslipidemia – disorders of lipid metabolism — cholesterol and triglycerides. Women with PCOS show a decreased removal of atherosclerosis-inducing remnants, seemingly independent of insulin resistance/Type II diabetes.
- Cardiovascular disease,[21] with a meta-analysis estimating a 2-fold risk of arterial disease for women with PCOS relative to women without PCOS, independent of BMI.
- Strokes[21]
- Weight gain.

Aim of Study:

The aim of the present study was to investigate the relationship between serum LH and AMH levels in a sample of Iraqi women with PCOS.

Patient and Methods:

1. Patient:

This study was approved by the Gynecology and Obstetrics department at Al-Nahrain university college of medicine. A total of 25 patients who first visited the higher institute for the diagnosis of infertility and assisted reproductive techniques for irregular menstruation and infertility between October 2018 and April 2019.

Inclusion criteria:

- Patients who gave written informed consent.
- Patients with known case of PCOS.
- Age between 18-35.

Exclusion criteria:

- Patients with congenital adrenal hyperplasia.
- Patients with androgen secreting tumor .
- Patients with cushing syndrome

All PCOS patients were diagnosed using the 2003 Rotterdam criteria (2 out of 3) as follows: 1) oligo-anovulation (menstrual cycle of >35 days); 2) clinical and/or biochemical signs of hyperandrogenism; and 3) PCO as identified by ultrasonography. Clinical hyperandrogenism was defined by the presence of hirsutism (modified Ferriman-Gallwey score >8) ,and biochemical hyperandrogenism was defined as an elevated serum androgen level (total testosterone >0.68 ng/mL, and/or free testosterone >1.72 pg/mL). PCO on ultrasonography was defined as follows: 1) the presence

of ≥12 follicles in each ovary measuring 2-9 mm in diameter; and/or 2) increased ovarian volume (>10 mL). All participants met the ultrasonographic criteria for PCOS.

2. Measurement of serum anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone levels:

Blood samples were collected from all participants in tubes without anticoagulants on the same day of ultrasonography in the early follicular phase. Sera were obtained by centrifugation for the determination of serum hormone levels. Serum hormone levels, including AMH, FSH and LH were measured using enzyme immunoassays.

Results:

A total number of 25 patients with PCOS were enrolled in this study.

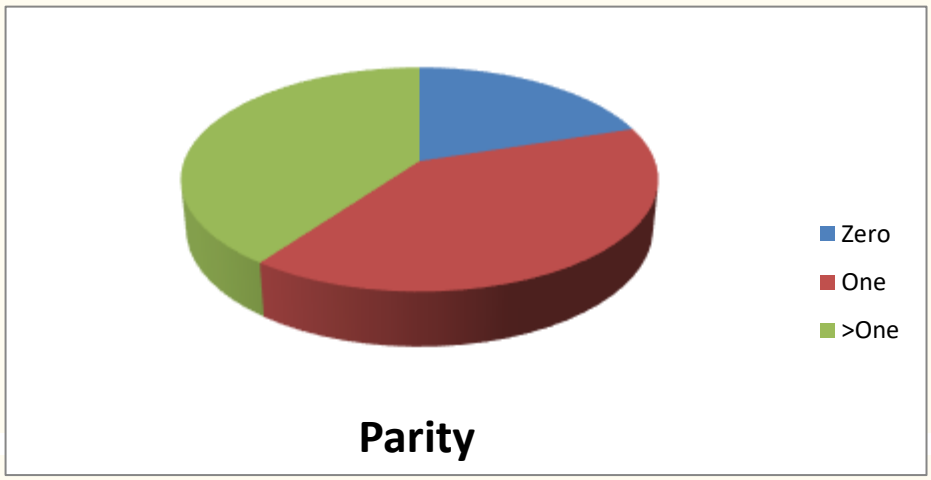
- Their ages range from 18 and 35 with median age of 24. (table1).
- (28%) of them had Zero parity,(32%) of them had 1 parity and (40%) had parity more than 1 (table2).
- (28%) had primary infertility and (72%) had secondary infertility.
- (40%) of them had normal BMI, (48%) of them were overweight and (12%) were obese
- The LH readings ranged from 7.3 and 22.7 with median of 14.4 (IU/L).
- FSH readings ranged from 7.1 and 14.3 with median of 10.59 (IU/L).
- AMH readings ranged from 3.9 and 11.2 with median of 8.6 (ng/ml)

Table (1): age distribution among primary and secondary infertile women.

Age	Primary infertility	Secondary infertility	total
18-25	4	6	10
25-30	3	9	12
30-35	0	3	3
35-40	0	0	0
Total	7	18	25

Table (2): number of parity.

	zero	one	More than one
Parity	7 (28%)	8 (32%)	10 (40%)

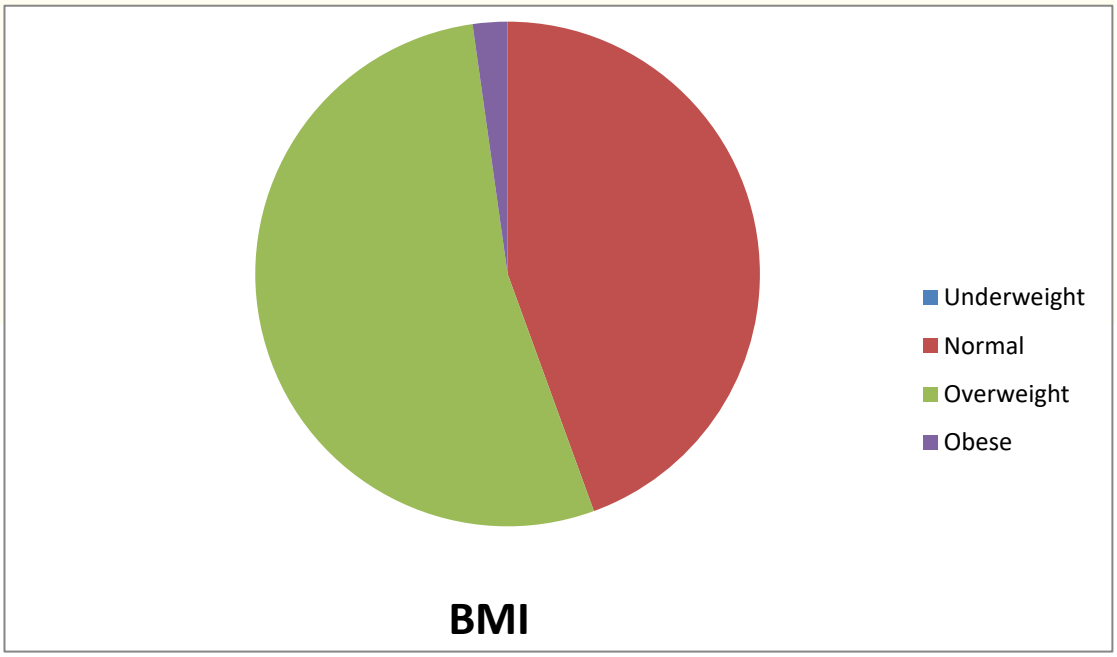


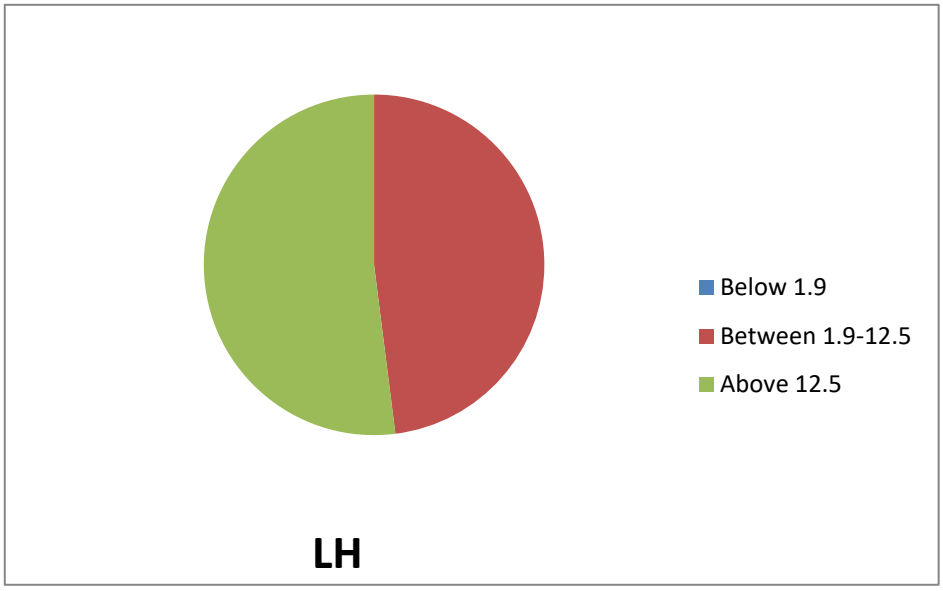
Variable	Primary infertility	Secondary infertility
LH level (IU/L)	12.95	15.27
FSH level (IU/L)	9.74	10.25
AMH level (NG/ML)	8.2	8.2
Age	24	25
BMI	24.6	27.2

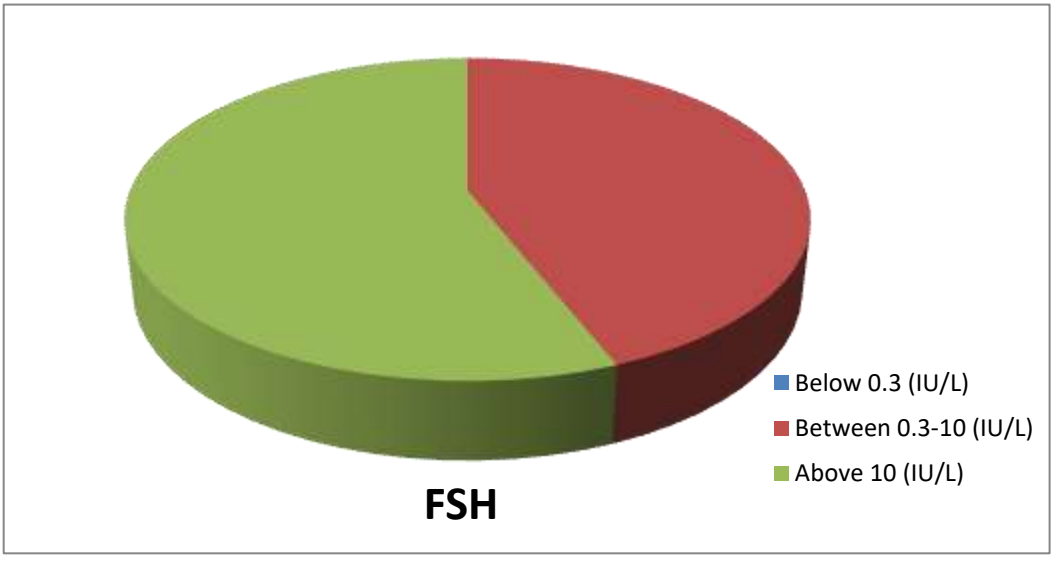
Table (3): hormonal and clinical parameters in PCOS patients.

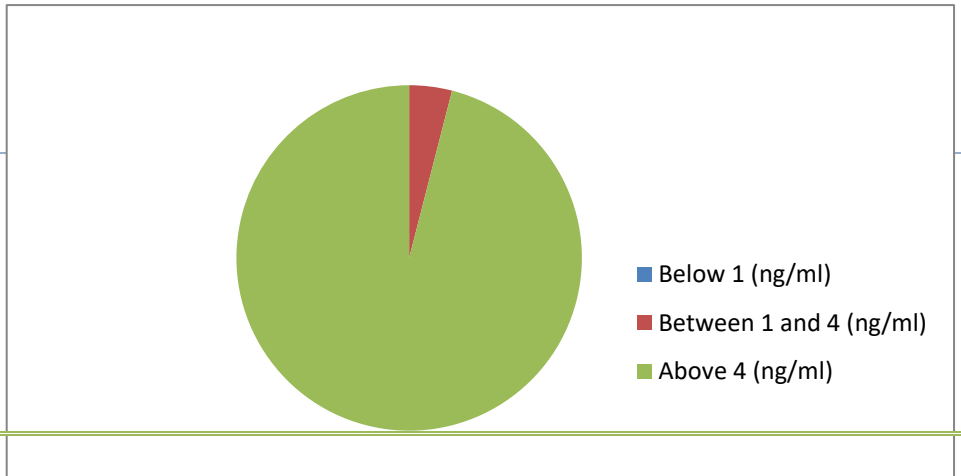
Table(4): BMI of patients with pcos.

BMI	Primary infertility	Secondary infertility	Total
Low	0	0	0
Normal	3	7	10
Overweight	4	8	12
Obese	0	3	3




Below 1.9
Between 1.9-12.5
Above 12.5
LH


Below 0.3 (IU/L)
Between 0.3-10 (IU/L)
Above 10 (IU/L)
FSH


Below 1 (ng/ml)
Between 1 and 4 (ng/ml)
Above 4 (ng/ml)

Discussion:

Increased AFC (≥12 follicles measuring 2.9 mm in diameter) and increased ovarian size (ovarian volume >10 cm^3) on ultrasonography are two cardinal features for the diagnosis of PCO which is similar to reports of Balen AH and Laven JS et al. in 2003. The AMH level that distinguishes normal volunteers who lack a polycystic ovary from PCOS with 95% specificity (>6.2 ng/mL) is similar to the cut-off level proposed by Dewailly et al. in 2003 for the diagnosis of PCOS (24). However, much higher AMH levels (>10.7 ng/mL) are required to comparably distinguish PCOS from asymptomatic women with a polycystic ovary, and sensitivity using this cut-point is poor.

The mean age of PCOS patients in the present study is 24 years old, this is believed to due to a declining number of antral follicles as the patients age advance which is similar to reports of Johnstone EB et al. in 2009 and Murphy MK et al. in 2006 [25, 26]. However, present study found no significant differences of age group among patients with PCOS.

I found that patients with PCOS were significantly correlated with the serum AMH and LH levels and AMH is regarded as the most useful marker of the status of ovarian reserve which is similar to studies of Lee JR et al. in 2009 and Van rooji IA in 2005. Women with PCOS have high AMH concentrations which is similar to studies of Pigny P et al.in 2003 , Laven JS et al. in 2004 and La Marca A et al. in 2004. The pathogenesis of PCOS has been attributed mainly to disordered folliculogenesis causing oligo-ovulatory cycles or impaired folliculogenesis with increased preantral and small antral follicle counts, which resulted in high serum AMH levels as reported by Visser JA et al. in 2005 and Sahmay S et al. in 2013. However, because of varying sensitivity and specificity, the optimal threshold remains unclear.

In the present study, the serum LH and AMH levels were highly correlated with PCOS, and these results were in agreement with those reported by Fanchin R et al. in 2003.

Conclusion:

This study found that LH and AMH are positively related in PCOS patients and this goes with the results of other studies regarding this subject.

Recommendations:

- PCOS is a common condition so patients should be fully aware about it.
- Patients should be educated about the importance of these hormones and their role in investigations.
- Lastly, We need longer duration and a more number of patients for further knowledge about the association of these hormones with PCOS.

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