Sudan Journal of Science and Technology
Journal homepage:
http://jst.sustech.edu/

# Anti- Müllerian Hormone and Lipid Profile in Sudanese Obese Women Diagnosed with Polycystic Ovary Syndrome

**Amel, A.A Sayed[*1] , Idris, O.F[*2], Salah, I. A[*3] , Amel, O.Bakhiet[*4]**

Department of Biology and Biotechnology, Faculty of Science and Technology, EL-Neelain University.
Department of Molecular Biology, Faculty of Science and Technology, EL-Neelain University.
Faculty of medicine , Omdurman Islamic University.
Deanship of Scientific Research-Sudan University of Science and Technology.

**ARTICLE INFO**

ARTICLE HISTORY
Received:20/2/2020
Accepted:12/7/2020
Available online:June2020

KEYWORDS:
Poly cystic ovary syndrome. Lipid profile, obese, fertile

**ABSTRACT**

As infertility binds to life style these days, increased lipid profile binds to diagnosis with poly cystic ovary syndrome, which can be diagnosed with AMH level. So this study aimed to assess AMH as a definitive diagnosis tool for PCOS and observing those results though such women with additional criteria, which was obesity. Measurement of all parameters was conducted in Alneelain University. Women were attended to fertility centers in Khartoum state-Sudan. So AMH and lipid profile (cholesterol, triglyceride, LDL and HDL were measured among case groups (obese PCOS and non-obese PCOS) and comparing data with other of control group involved fertile non –obese women. Data revealed that there was increase of AMH with lipid profile parameters except the HDL among PCOS women obese and non-obese.

## Introduction:

Poly cystic ovary is a heterogeneous endocrinopathy characterized by both reproductive and metabolic abnormalities (Rotterdam, 2004) . PCOS is a common hormonal abnormality in reproductive-age women affecting almost 7% of this population. The reproductive features of PCOS include menstrual irregularity and infertility (Sam and Dunaif, 2003). PCOS has metabolic characteristics that include prominent defects in insulin action and β-cell function, defects that confer a substantially increased risk for glucose intolerance and type 2diabetes (Sam and Dunaif, 2003) (Dunaif, 1999). Although PCOS is the most frequent endocrine disorder in women of reproductive age but its diagnosis remains one of the most challenging issues in endocrinology, gynecology, and reproductive medicine (Eshre, 2004). Anti- Müllerian hormone (AMH), also known as Müllerian inhibiting substance, has been mainly studied for its regulatory role in male sex differentiation. AMH, produced by the Sertoli cells of the fetal testis, induces the

regression of the Müllerian ducts, the anlagen of the female reproductive tract (Josso *et al*, 1993) (Lee and Donahoe, 1993). AMH is a homodimeric glycoprotein linked by disulfide bonds and a molecular weight of 140kD (Cate *et al*, 1986). The hormone belongs to the Transforming Growth Factor-β (TGF-β) superfamily. The gene encoding AMH is located in the short arm of chromosome 19 (Cohen-Haguenauer *et al*, 1987). AMH action is exerted through two receptors: type I receptor (AMHRI) and type II receptor (AMHRII) which are present on the AMH target-organs (gonads and Müllerıan ducts) (LaMarca and Volpe, 2006). The ovarian reserve refers to the number of primordial follicles, defined at birth (around 1 million). This follicular capital decreases gradually throughout reproductive life, with the continuous initiation of growth of some follicles, and then mostly their apoptosis. There are about 400 000 follicles in adolescents' ovaries (leading roughly to 400 ovulations), whereas only a thousand remains at the time of Menopause. Serum AMH concentration is strongly correlated with the number of growing follicles since it represents AMH secretion from all developing follicles.Serum AMH concentration is strongly correlated with the number of growing follicles since it represents AMH secretion from all developing follicles (Laven *et al*, 2004) (Pigny *et al*, 2003).

The word obesity is derived from the Latin obesus, which means "one who has become plump through eating." It may have first appeared in the writings of Thomas Venner in 1620 (Barnett, 2005). However, the negative effect of obesity on an individual's health has been known for a longer time and can be found in the writings of Hippocrates, Galen, and Avicenna (Abdel-Halim, 2005). Avicenna was probably among the first who described the relationship between obesity and male infertility in his encyclopedic medical book The Canon of Medicine. In a chapter entitled "The health disadvantages of excessive weight," Avicenna wrote, "this human (man) has a cold temperament; this is why he is infertile, unable to impregnate (women) and has low semen"(Avicenna, 2006). In modern times, the relationship between obesity and male infertility has been largely ignored until recently (Hedley *et al*, 2004) (Mokdad *et al*,1999). Interest in the rapid increase in obesity has brought to light the detrimental effects of obesity on health in general and on the reproductive function in particular.In women, the effects of extremes of body composition on reproductive function are readily evident by altered menstrual function and are well known and extensively studied (Pasquali *et al*, 2003).

Decrease in HDL-C and increase in TG levels are well known lipid profile characteristics in women with PCOS (Legro *et al*, 2001) (Wild *et al*, 1985) (Diamanti-Kandarakis *et al*, 1998).

**Material and method:**

This case control study involved, two case groups of infertile women, diagnosed with poly cystic ovary syndrome, attended to the fertility center, in Khartoum, they were at the reproductive age. The first group involved 55 obese PCOS women with BMI 31.4kg/$m^2$ and age's mean (29.9) years. The other group involved was 45 non-obese PCOS women with BMI 22.7 kg/$m^2$ and age's mean 28.8 years. A control group included 30 women, who were fertile with no obesity with BMI 21.6 kg/$m^2$and their age mean 27.1 years. Whole blood samples were collected at the morning as they were fasting. Serum was used for assessment of AMH and heparinized blood samples were used for the assessment of lipid profile (Cholesterol. Triglyceride, HDL and LDL). Reagents and device for the measurement were manufactured by Biosystem trade mark- Germany.

Laboratory work was conducted at Alneelain University-faculty of science-department of biochemistry.

Statistical analysis was performed using SPSSl7.0 statistical software. Measurement data were expressed as mean ± standard deviation (M ± S). Data were tested for normality and homogeneity of variance and compared using either t test (equal variances) or t' test (unequal variances). Significance level $\alpha = 0.05$, $P < 0.05$ was considered statistically significant.

**Result:**

A case control study involved 55 obese PCOS women and 45 non-obese PCOS women as case groups. The comparison of AMH levels among obese PCOS with non-obese PCOs groups revealed that increase level among the obese PCOS group giving increased significant difference as the P value 0.000, while lipid profile showed that increased cholesterol, triglyceride and LDL, while HDL was decreased each set gave high significant difference as P value for each 0.00 as well, (Table 1).

Table(1): Mean concentration comparison of AMH and lipid profile among PCOS obese case group and non-obese fertile control groups.

| Parameters | non-obese control (Mean±SD) | Obese PCOs (Mean±SD) | *P-value* |
|---|---|---|---|
| AMH (Pmol/l) | 2.99±0.91 | 9.20±4.10 | 0.000 |
| Cholesterol (mg/dl) | 162.53±12.16 | 217.06±19.01 | 0.000 |
| Triglycerides (mg/dl) | 93.85±17.89 | 125.45±16.55 | 0.000 |
| HDL (mg/dl) | 56.78±6.19 | 36.61±3.89 | 0.000 |
| LDL (mg/dl) | 87.04±11.38 | 155.75±16.23 | 0.000 |

**Significant difference as p value <0.005**

Comparing the data of AMH among obese PCOS and control groups gave the significance difference as AMH was increased among non-obese PCOS more than control giving increased significant difference, and comparing the data of lipid profile among non-obese PCOS and control groups gave also significance difference as, cholesterol, triglyceride and LDL were increased among non-obese PCOS than control, while HDL was low among the non-obese PCOS group than control giving increased significant difference (Table 2).

Table(2): Mean concentration comparison of lipid profile amongnon-obese control and non-obese PCOs

| Parameters | Non-Obese control (Mean±SD) | Non-Obese PCOs (Mean±SD) | *P-value* |
|---|---|---|---|
| AMH (Pmol/l) | 2.67±0.98 | 7.54±2.91 | 0.000 |
| Cholesterol (mg/dl) | 130.9±9.06 | 191.04±14.37 | 0.000 |
| Triglycerides (mg/dl) | 64.19±12.49 | 103.50±16.64 | 0.000 |
| HDL (mg/dl) | 51.96±6.35 | 35.12±4.03 | 0.000 |
| LDL (mg/dl) | 66.22±9.86 | 135.22±12.76 | 0.000 |

**Significant difference as p value <0.005**

**Discussion:**

In this study PCOS diagnosed women were targeted to be assessed for AMH and lipid, as obesity was one of the criteria for selection, through BMI and lipid profile the observation was conducted. BMI ($kg/m^2$) normal weight range (18.5-24.99) 21 and overweight (25-29.99), while obese >30.1.AMH among obese and non-obese women diagnosed with PCOS was elevated more than control group with involvement fertile of women with no obesity. Lipid profile assessed was included cholesterol, triglyceride,

LDL and HDL. Each of cholesterol, triglyceride and LDL were increased among PCOS women and HDL decreased than control, giving significant difference.

Many studies were involved both AMH and lipid profile among PCOS patients, as PCOS is connected to many disorders. This study in close agreement of study conducted at the same manner as total of 80 women diagnosed with PCOS were investigated for lipid profile parameters and other parameters and compared with 40 apparently healthy women. Cholesterol, triglyceride and LDL were increased and HDL was decreased among PCOS women than control giving significant difference (Richa Lath *et al*, 2015). Similar finding also were reported by Nimish *et al* 2016, who tested lipid profile among PCOS women with other parameters, their study it concluded that hyperandrogenism in PCOS may be additionally marked by raised LDL. Overweight/obese PCOS subgroup may be prone to dyslipidemia (Nimish *et al*, 2016). Though different studies, PCOS always binds to obesity and to prove that, a study conducted inversely, as it measured the lipid profile among PCOS women before and after taking drugs anti to the sugar in blood (metformin) and used to dissolve fatty material in human, it found that, decreased levels of cholesterol, triglyceride and LDL and increased in HDL after drug usage more than before as there was an improvement of lipid profile among PCOS patients (Geetika Singh *et al*, 2017). A n agreement of the present finding study also found in other study, as more than 70% women, lipid abnormalities such as low levels of high-density lipoprotein (HDL) cholesterol and high levels of triglycerides and low-density lipoprotein cholesterol were observed (DonthuKiranmayee *et al*, 2017).

**Conclusion:**

Obesity did not prevent women from being fertile, as to be, additional reasons should be involved, such as increased levels of prolactin, testosterone and presence of high AMH to diagnosis poly cystic ovary syndrome.

PCOS is related to increased cholesterol, triglyceride and LDL and decreased level of HDL.

**Recommendation**

Lipid profile assessment should be applied as routine laboratory work among PCOS women in order to control the side effects of diagnosis with PCOS and diminish complications related.

**Acknowledgement**

Thanks to Professor Amel Omer Bakhiet for her support and encouragement and for creating such good conditions to carry out this research. My also thanks to Dr. Elsir Abu Elhassan the head of the Dr. Elsir Abu Elhassan Fertility Center and the stuff of Alneel Fertility Center, Banoon Fertility Center, and Ashmeek fertility center.

**Reference:**

**Abdel-Halim, R.E. Obesity: 1000 years ago. Lancet. (2005);** 366: 204

Avicenna (2006).The disadvantages of excessive weight. In: Avicenna. The canon of medicine, book IV: diseases involving more than one member; the Cosmetic art. Rome: Medical Press, 1593:173–174.

**Barnett R. Obesity.Lancet. (2005);** 365: 1843.

**Cate RL, Mattaliano RJ and Hession, (1986).** Isolation of the bovine and human genes for MIS and expression of the human gene in animal cells. Cell 45: 685-698.

**Cohen-Haguenauer O, Picard Mattei JY and Mattei MG, (1987).** Mapping the gene for anti-Müllerian hormone to the short arm of human chromosome 19. Cytogenet Cell Genet 44: 2-6.

**Diamanti-Kandarakis E, Mitrakou A, Raptis S, Tolis G and Duleba AJ (1998).** The effect of a pure antiandrogen receptor blocker, flutamide, on the lipid profile in the polycystic ovary syndrome.J Clin Endocrinol Metab. 1998;83:2699–2705.

**DonthuKiranmayee, KothapalliKavya, YalamanchaliHimabindu MS, Professor ManneSriharibabu, GadiLeela Jaya Madhuri and SwargamVenu, (2017).** Correlations Between Anthropometry and Lipid Profile in Women With PCOS. J Hum Reprod Sci. 2017 Jul-Sep; 10(3): 167–172.

**Dunaif A, (1999).** Insulin action in the polycystic ovary syndrome. Endocrinol Metab Clin North Am. 28:341–359.

**Eshre R (2004)** ASRM-sponsored PCOS consensus workshop group, 2004 Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertility and Sterility 81: 19-25.

**Geetika Singh, NishatAfroz, NooraSaeed, SheeluShafiqSiddiqi, AaliyaEhsan and MohdRafey, (2017).** Study of Lipid Profile in Patients of Polycystic Ovarian Syndrome Before and After Metformin Therapy. Pacific Group of e-JournalsAugust.

**Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR and Flegal KM, (2004).** Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. JAMA. 2004; 291: 2847–2850.

**Josso N, Cate RL, Picard JYand Vigier B, di Clemente N, Wilson C, Imbeaud S, Pepinsky RB, Guerrier D, Boussin L, Legeai L and Carré-Eusèbe D, (1993).** Anti-mullerian hormone: the Jost factor. Recent Progress in Hormone Research 481–59.

**LaMarca A and Volpe A,(2006).** Anti-Müllerian hormone (AMH) in female reproduction: is measurement of circulating AMH a useful tool? Clin Endocrinol 64: 603-610.

**Laven JS, Mulders AG, Visser JA, Themmen AP, De Jong FH and Fauser BC, (2004).** Anti-Mullerian hormone serum concentrations in normoovulatory and anovulatory women of reproductive age.J Clin Endocrinol Metab. 89(1):318–23.

**Lee MM & Donahoe PK (1993).** Mullerian inhibiting substance: a gonadal hormone with multiple functions. Endocrine Reviews 14 152–164.

**Legro RS, Kunselman AR and Dunaif A (2001).** Prevalence and predictors of dyslipidemia in women with polycystic ovary syndrome. Am J Med.111:607–613.

**Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS and Koplan JP (1999).**The spread of the obesity epidemic in the United States, 1991–1998.JAMA. 1999; 282: 1519–1522.

**Nimish R. Halasawadekar, Jaiprakash B, Ramanand, Sunita J, Ramanand, Girish T, Raparti, Praveenkumar T, Patil, Ruchi D, Shah Arvind V and Kumbhar, (2016).** Serum lipid profile in non-polycystic ovary syndrome and polycystic ovary syndrome women: a comparative and correlational study. ijbcp2016 .

**Pasquali R, Pelusi C, Genghini S, Cacciari M and Gambineri A (2003).** Obesity and reproductive disorders in women. Hum Reprod Update. 2003; 9: 359–372.

**Pigny P, Merlen E, Robert Y, Cortet-Rudelli C, Decanter C, Jonard S, (2003).** Elevated serum level of anti-mullerian hormone in patients with polycystic ovary

syndrome: relationship to the ovarian follicle excess and to the follicular arrest. J Clin Endocrinol Metab. 88(12):5957–62.
**Richa Lath, Reshakiran Shendye and Aniruddha Jibhkate, (2015).** Insulin resistance and lipid profile in polycystic ovary syndrome. Asian Journal of Biomedical and Pharmaceutical Sciences.
**Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised (2003)** consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. FertilSteril. 2004;81:19–25.
**Sam S and Dunaif A. Polycystic ovary syndrome: syndrome XX? Trends Endocrinol Metab. (2003**;)14:365–370.
**Wild RA, Painter PC, Coulson PB, Carruth KB and Ranney GB (1985).** Lipoprotein lipid concentrations and cardiovascular risk in women with polycystic ovary syndrome.J Clin Endocrinol Metab. 1985;61:946–951.