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EVALUATION OF SERUM ANTI-MULLERIAN HORMONE LEVELS ON OXIDATIVE STRESS MARKERS AND THE ENVIRONMENTAL LIFESTYLE IN FEMALE CONTROL AND PATIENTS WITH POLYCYSTIC OVARY SYNDROME GROUPS

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June 2022

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ABSTRACT

EVALUATION OF SERUM ANTI-MULLERIAN HORMONE LEVELS ON OXIDATIVE STRESS MARKERS AND THE ENVIRONMENTAL LIFESTYLE IN FEMALE CONTROL AND PATIENTS WITH POLYCYSTIC OVARY SYNDROME GROUPS

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Master of Science in Chemistry

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Despite the fact that the most prevalent cause of infertility is polycystic ovary syndrome (pcos), infertility affects women around the world. It is known that PCOS is connected with oxidative stress, and this study was meant to explore the association between free radicals and AMH levels in the body; AMH is a good biomarker of PCOS. The study included 100 study participants, 50 of whom had pre-menstrual syndrome (PMS) and the other 50 were healthy controls. The participants were recruited from the Nineveh Health Center. There is a well-known relationship between PCOS and AMH elevation, Asignificant effect between MDA and AMH was observed in PCOS' blood samples, the PCOS samples with high Amh are significant associated with high GLU, The effect of high cholesterol levels on PCOS cases was not strong, aweak effect of low GSH concentration on AMH value was observed in PCOS' blood samples, there is a relative relationship between AMH and FSH, It Depends on the age for PCOS cases.

2022, 55 pages

Keywords: AMH Hormon, Polycystic Ovary Syndrome, Oxidative Stress Markers, Malondehyde, Glutathion, Vitamine D, Insulin resistance, Environmental Lifestyle

ÖZET

KADIN KONTROLÜ VE POLİKİSTİK OVARY SENDROM GRUBU OLAN HASTALARDA OKSİDATİF STRES BELİRTEÇLERİ ÜZERİNDEKİ SERUM ANTİMÜLLER HORMON DÜZEYLERİNİN VE ÇEVRESEL YAŞAM TARZLARININ DEĞERLENDİRİLMESİ

Aswan Adnan Abdulwahhab AL-SAFFAR
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Haziran 2022

Kısırlığın en yaygın nedeninin polikistik over sendromu (pkos) olmasına rağmen, infertilite tüm dünyada kadınları etkilemektedir. PCOS'un oksidatif stres ile bağlantılı olduğu biliniyor ve bu çalışmanın amacı vücuttaki serbest radikaller ve AMH seviyeleri arasındaki ilişkiyi araştırmaktı; AMH, PCOS için iyi bir biyobelirteçtir. Çalışma, 50'si adet öncesi sendromu (PMS) olan ve diğer 50'si sağlıklı kontroller olan 100 çalışma katılımcısını içeriyordu. Katılımcılar Nineveh Sağlık Merkezi'nden alındı. PCOS ile AMH yüksekliği arasında iyi bilinen bir ilişki vardır, PCOS'lu kan örneklerinde MDA ve AMH arasında önemli bir etki gözlenmiştir, Amh'si yüksek olan PCOS örnekleri yüksek GLU ile ilişkilidir, Yüksek kolesterol düzeylerinin PCOS vakaları üzerindeki etkisi PKOS'lu kan örneklerinde düşük GSH konsantrasyonunun AMH değeri üzerinde güçlü değil, zayıf etkisi gözlenmiştir, AMH ve FSH arasında nispi bir ilişki vardır, PKOS vakalarında yaşa bağlıdır.

2022, 55 sayfa

Anahtar Kelimeler: AMH Hormonu, Polikistik Over Sendromu, Oksidatif Stres Belirteçleri, Malondialdehit, Glutatyon, D Vitamini, İnsülin direnci, Çevresel Yaşam Tarzı

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Aswan Adnan Abdulwahhab AL-SAFFAR

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LIST OF SYMBOLS

-	Minus
%	Percent
/	Divide
+	Plus
°C	Celsius
µL	Microliter
cm	Centimeter
dL	Deciliter
g	Gram
IU	International Unit
kg	Kilogram
Kg/m^2	Kilogram-meter squared
m	Meter
mg	Milligram
mg/dL	Milligrams per deciliter
mIU	Milli-international Unit
min	Minute
mL	Milliliter
mmol/L	Millimoles per litre
ng	Nanogram
nm	Nanometer

LIST OF ABBREVIATIONS

A	Absorbance
AMH	Anti-mullerian hormone
Cho	Cholestrol
D	Vitamin
FSH	Follicle-stimulating hormone
GOD	Glucose oxidase
GSH	Glutathione
HDL	High density lipoprotein
IF	Intrinsic factor
IR	Insulin resistance
LDL	Low-density lipoprotein
MDA	Malondialdehyde
MI	Body mas index
OS	Oxidative stress
PCOS	Polycystic ovarian syndrome
ROS	Reactive oxygen species
VLDL	Very low-density lipoprotein

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1. INTRODUCTION

Couples struggling with infertility are among the most emotionally distraught people in the world. Even though it isn't deadly, it has been referred to be a life-altering and psychologically traumatizing issue. There are several issues that might contribute to infertility, and a reason may never be found. Polycystic ovarian syndrome (PCOS), which affects six to ten percent of premenopausal women, is a common cause of infertility in these people. High androgenism, hirsutism, and oligomenorrhea, or the lack of menstruation, are all symptoms of PCOS. Metabolic, hormonal, and cardiovascular disorders may coexist in the same person. This is plausible. Oxidative stress has been associated to insulin resistance and an increase in testosterone levels in patients (González and colleagues 2006).

N-acetylcysteine (NAC), which is known to replenish glutathione stores, has an antioxidant glutathione replenishment effect in patients with insulin resistance, as do hyperglycemia and insulin resistance found elevated levels of MDA and upregulated SOD activity in patients' controls (Fulghesu *et al.* 2002, Kuşçu and Var 2009). Patients with insulin resistance had the highest levels of MDA. Oxidative stress affects both men and women's reproductive lives because of an imbalance between reactive oxygen species (ROS) and antioxidants. oxidative stress may disturb the intracellular milieu, resulting in sick cells or cell death, and ROS can influence the cell's functions. As a general rule, antioxidants are able to counteract ROS generation by neutralizing existing free radicals and facilitating cell structure repair. Low amounts of oxidative stress may enhance certain reproductive functions, while high levels may cause infertility and adverse pregnancy outcomes.

Reactive oxygen species in excess and inadequate antioxidant status have been connected in studies on infertility, therefore restoring the reproductive microenvironment's antioxidant defenses makes sense as an approach to treating infertility. Glutathione, a natural antioxidant, shields other antioxidants in the body from glutathione's toxicity. It is found in both male and female gametes, although the degree

varies widely. Both male and female infertility are examined in this research, as well as the function of oxidative stress and the antioxidant effect of glutathione. Follicular growth in women seems to be regulated by the anti-Müllerian hormone (AMH), which is secreted by the granulosa cells, and its amount varies with the menstrual cycle, peaking at its highest level in the latter stages of follicular development (La Marca *et al.* 2005) Serum AMH levels, on the other hand, might vary widely among persons of the same gender and ethnicity (Dennis *et al.* 2012).

FSH does not seem to have a direct role in AMH production or release when estradiol and follicle-stimulating hormone (FSH) levels follow the anticipated patterns throughout pregnancy or early puerperium (La Marca *et al.* 2005). Serum AMH levels have been linked to polycystic ovary syndrome (PCOS), but the lack of an international standard for AMH assay, mainly due to technical difficulties, makes it difficult to define consensus thresholds and thus hinders the widespread use of this new ovarian marker in the diagnosis and treatment of PCOS (Dumont *et al.* 2015).

The blood AMH test for menopause prediction is expected to improve in the near future. It was recently shown that ovarian reserve testing and the time to menopause are linked (Broer *et al.* 2010). Women's AMH levels decline as they become older, according to a few researchers (Barbakadze *et al.* 2015).

1.1 Objectives of Study

We aim to describe the associations between demographic, lifestyle, reproductive factors and physical factors with anti-Mullerian hormone concentration and take advantage of linking the measurements to the pathological diagnosis and suggesting recommendations to maintain the hormone ratio within the normal range.

1.2 Specific

- Study the relationship between(AMH Hormone and stress marks and anti-oxidant) and the effect of levels of them on PCOS.
- Knowing the relationship between AMH and FSH and clinical parameters (Glugose, lipid profile, vit D).
- Study and detect the relationship between clinical parameters among them.

2. LITERATURE REVIEW

2.1 Polycystic Ovarian Syndrome (PCOS)

Complex, mysterious, and common: Polycystic ovarian syndrome (PCOS). When it comes to reproductive-age women, it's the most frequent endocrinopathy, affecting one in five (Teede *et al.* 2010). Women who have anovulatory menstruation for an extended period of time are at an increased risk of developing endometrial cancer. Obesity, hirsutism, and diabetes mellitus are also more common in women with anovulatory menstruation. Polycystic ovarian syndrome may be caused by a combination of hereditary and environmental causes (PCOS) (Escobar 2018). Menstrual abnormalities and increased testosterone levels are the most common symptoms. Anovulation may be caused by a wide range of health issues, including PCOS.

PCOS is a diagnosis of exclusion because of its wide range of possible causes. Menstrual abnormalities, aberrant ovarian size and shape on ultrasonography, and clinical or laboratory evidence of hyperandrogenism are all common symptoms for women who are affected. Insulin resistance, dyslipidemia, obesity, and cardiovascular risk factors are typically linked with metabolic dysfunction. PCOS includes a slew of significant side effects. Endometrial hyperplasia and endometrial cancer are more likely to occur if estrogen levels are increased. Hirsutism and metabolic syndrome are both made more likely when androgen levels are increased. Increased ovarian androgen production may be a result of hyperinsulinemia caused by insulin resistance. Cardiovascular disease, particularly hypertension, is a long-term consequence of androgen excess in men. Depression and anxiety are more common among pregnant women with PCOS, and their capacity to deal with life's ups and downs is also affected (Teede *et al.* 2011).

There is, however, substantial evidence that genetics may play a role in the development of PCOS. Hyperandrogenemia and hyperinsulinemia (common results in PCOS) have been shown to be inherited in families with the illness, indicating a probable genetic

component. Supporting the theory that genetics and environment are intertwined are environmental risk factors like obesity (Phee *et al.* 2010).

2.2 The Distinction Between Ovarian Cysts and Polycystic Ovaries (PCOS)

Understanding the distinctions between Polycystic Ovarian Syndrome (PCOS) and Ovarian Cysts is critical. In women with PCOS, the male reproductive hormone androgen is overproduced, resulting in an imbalance of hormones. The ovaries, which normally generate the hormones oestrogen and progesterone necessary to regulate the monthly cycle, are affected by this imbalance. To put it another way, this results in the menstrual cycle being disrupted, which leads to missing or fewer periods (González *et al.* 2019).

There are two types of ovarian cysts: those filled with fluid, which rupture and produce intense abdominal discomfort; and those filled with fluid that do not break and do not cause bleeding. Women with regular cycles are more likely to develop ovarian cysts. Ovarian cysts might present with no symptoms at all in some women. It's possible for ovarian cysts to remain functioning, despite the fact that they never burst and produce an egg. In addition to severe pelvic infections, other causes of ovarian cysts include aberrant ovarian cell growths, both benign and malignant. So Ovulation issues and increased testosterone production are all part of the polycystic ovarian syndrome, a metabolic condition. Cysts in the ovaries are filled with fluid and do not affect ovulation in any way. They might be asymptomatic and have no additional side effects (Miller 2021).

2.2.1 AMH(anti-mullerian hormone)

To further understand AMH's role in growth and development, we need to understand its structure as a two-component glycoprotein. Its principal purpose is to distinguish between men and women. The synthesis of AMH by fetal Sertoli cells during testicular development causes Mullerian duct regression. All of the reproductive organs of the

female reproductive system are developed without AMH (Munsterberg and Lovell 1991).

At 32 weeks gestation, human fetuses have been seen to express AMH in their ovaries (Rajpert *et al.* 1999). AMH immunostaining may be found in the granulosa cells of follicles in the early stages of development. The ovary's developing follicles produce AMH until they reach a size and differentiation stage at which they may be chosen for dominance by the gene's promoter (Durlinger *et al.* 2002). AMH expression initially appears in human antral follicles of 4–6 mm in diameter (Weenen *et al.* 2004).

AMH is expressed because it is created in follicles that have been drawn from the primordial follicle pool but have not been selected for dominance. AMH is not expressed by the theca cells or atretic follicles (Ueno *et al.* 1989, Munsterberg and Lovell 1991, Hirobe *et al.* 1994).

Researchers have discovered that the oocyte's stage of development (early pre-antral, late pre-antral, and pre-ovulatory follicles) affects the mRNA levels of the AMH gene in the granulosa cells. Oocyte-modulated gene expression in granulosa cells may play a significant role in coordinating intra- and interfollicular follicle development, as shown by these studies (Salmon *et al.* 2004).

The selection of follicles may be affected if AMH inhibits follicular FSH sensitivity (Durlinger *et al.* 1999, Gee and Hsueh 2000). When AMH expression is reduced in the ovaries, ovulation might occur earlier in the estrous cycle, making it easier for the body to respond to FSH (Durlinger *et al.* 2001, Visser *et al.* 2007). It's still unclear what function AMH plays in the human ovary, since few research have been done in vitro or in vivo to investigate this. Estradiol concentrations in follicular fluid from antral follicles have been related with AMH levels in recent investigations, according to the researchers (Andersen and Byskov 2006).

An association between AMH gene polymorphisms and estradiol levels in the follicular phase has recently been found, suggesting that AMH is involved in FSH-induced steroidogenesis, as shown by a recent study (Kevenaar *et al.* 2007).

2.2.2 The levels of AMH

At birth, females have essentially no measurable quantities of AMH. AMH levels peak in late adolescence, then decline progressively throughout reproductive life as the follicular reserve is depleted, and finally become undetectable after menopause (Guibourdenchee *et al.* 2003). For further information, see (VanRooij *et al.* 2004, La Marca *et al.* 2005b). The absence of measurable AMH levels in the study group 3–5 days following bilateral ovariectomy, according to the results of further research, suggests that the majority of circulating AMH originates from the ovaries (La Marca *et al.* 2005b).

There are no significant changes in AMH blood levels throughout the menstrual cycle, which suggests that it may be a unique endocrine indication for the investigation of ovarian function (Hehenkamp *et al.* 2006, La Marca *et al.* 2006b,Tsepelidis *et al.* 2007, Streuli *et al.* 2008)

Oscillations of 3 to 219 percent have been recorded in AMH's claimed intercycle oscillations (Wunder *et al.* 2008, Streuli *et al.* 2009). These variations are equivalent to those found in AMH's reported luteal phase oscillations (Wunde *et al.* 2008, Streuli *et al.* 2009, Streuli *et al.* 2008) , The clinical environment may consider the variability in blood AMH levels between and between cycles during a menstrual cycle to be low enough to allow for random scheduling of AMH testing. As a result of the pituitary being down-regulated by GnRH agonist pituitary (Mohamed *et al.* 2006) and the use of oral contraceptives, AMH levels seem unchanged when endogenous gonadotrophin secretion is dramatically lowered (La Marca *et al.* 2005a, Arbo *et al.* 2007, Somunkiran *et al.* 2007, Streuli *et al.* 2008). It is possible that this investigation will prove that FSH-independent ovulation may occur even when pituitary FSH secretion is suppressed.

FSH-independent growth of small follicles is compatible with AMH levels, according to this theory.

2.2.3 AMH with polycystic ovary syndrom

Studies show that women with polycystic ovarian syndrome (PCOS) had greater blood AMH levels than women who do not have PCOS (Cook *et al.* 2002, Pigny *et al.* 2003, La Marca *et al.* 2004b, Laven *et al.* 2004, Mulders *et al.* 2004, Eldar-Geva *et al.* 2005b, La Marca *et al.* 2006a, Wachs *et al.* 2007). Polycystic ovaries' increased production of granulosa cells and release of AMH is thought to be responsible for these results (Mulders *et al.* 2004). There is a 75-fold difference between the AMH levels in polycystic ovaries and those in normal ovaries (Pellatt *et al.* 2007). If folliculogenesis is not working properly, an excess of pre-antral and small follicles in the anterior area of the ovaries might be the source of increased AMH in PCOS patients (Wang *et al.* 2007). Women with PCOS who had regular menstrual cycles in their teenage years had higher AMH levels, which suggests that abnormal follicular development occurs before the clinical phenotypes of PCOS are visible in their prepubescent and peripubertal children. In terms of specificity and segregation, AMH measurements have been proven to be very useful (Pigny *et al.* 2006).

2.3 FSH

Follicle-stimulating hormone (FSH) is secreted by the anterior pituitary in response to hypothalamic gonadotropin-releasing hormone (GnRH) (Stamatiades *et al.* 2018). FSH has a crucial role in the development and reproduction of sex in both males and females. The hormone is made comprised of the glycoprotein dimer FSH's alpha and beta subunits. While the alpha subunit is present in TSH and hCG, as well as in LH and FSH, the beta subunit is only found in FSH.

For example, GnRH released into the hypophyseal portal circulation acts on anterior pituitary cells via G-protein-coupled receptors, resulting in the production by these

gonadotropic cells of FSH and luteinizing hormone (LH) that is then released into the peripheral circulatory system. The GnRH is induced to create more FSH by low pulse frequencies, whereas the LH is stimulated by high pulse frequencies. When GnRH is used constantly, the anterior pituitary releases less FSH and LH, which inhibits ovulation and estrogen synthesis in women. This is how GnRH agonists like leuprolide are used in the treatment of gynecomastia. FSH secretion is inhibited in women by the negative feedback from estrogen levels (Shaw *et al.* 2010).

In males, Sertoli cells release inhibin B in response to FSH, which acts as a negative feedback mechanism to restrict FSH production (Boepple *et al.* 2008). Fertility-promoting hormone (FSH) stimulates granulosa cells, which convert androgens generated by thecal cells into estradiol. Follicular Development in Women During the Menstrual Cycle. In the menstrual follicular phase of the cycle, FSH stimulates ovarian follicle development by promoting the growth of follicles. Estradiol and inhibin released by a dominant follicle induces the hypothalamus to release GnRH, which in turn promotes the production of gonadotropic hormones. After the LH spike that produces ovulation, FSH levels stay low for the rest of the luteal phase.

2.3.1 FSH with AMH and PCOS

According to earlier studies that demonstrated a negative association between FSH and AMH blood levels, the AMH level is strongly predictive of the FSH level and may be used as an independent predictor of ovarian reserve (Singer *et al.* 2009) According to another study, the amount of intrafollicular AMH (AMH) was shown to correlate adversely with the level of FSH (Follicle Stimulating Hormone) in ovulatory women who had IVF treatment (Dumesic *et al.* 2009).

Follicles are less sensitive to FSH when AMH is present (Durlinger and colleagues 2001) Increases in blood AMH concentration in women with polycystic ovarian syndrome (PCOS) have been observed to coincide with a 2- to 3-fold increase in the number of small follicles. A rise in AMH levels has been connected to the development of PCOS as a disease. Follicular sensitivity to FSH in the blood may be reduced in

PCOS, resulting in follicle selection failure and follicle arrest in the small antral phase with failure of dominance (Pigny *et al.* 2003, Laven *et al.* 2004).

The LH:FSH ratio is skewed in PCOS as a result of the hormone GnRH's irregular but persistent pulses. During GnRH pulses, the LH:FSH ratio is off, causing the ovaries' theca cells to release too much androgen but the granulosa cells to not produce enough aromatase to convert it into estradiol. This leads to a variety of problems (Stamatiades GA *et al.* 2018).

2.4 Oxidative Stress Biomarkers

According to Sies, oxidative stress is a "disturbance in the pro oxidant-antioxidant balances in favor of the former, resulting to significant damage". Reactive oxygen species (ROS) and free radicals are generated during metabolic and immune system activity, and this causes oxidative stress, which is both necessary for life and responsible for cell death, to worry researchers all over the globe. Complex organic compounds are formed in organisms by the oxidation process, which utilizes the H2O2 substrate for numerous enzymes (peroxidases) (Keshari *et al.* 2014, Apel and Hirt 2004).

Humans' principal defenses against ROS toxicity are antioxidants, including endogenous antioxidants like Catalase and superoxide dismutase (SOD), small proteins like thioredoxin and glutaredoxin, and chemicals like glutathione (Apel and Hirt 2004). Oxidative stress occurs when the concentration of ROS exceeds a particular threshold. Damage to DNA and other cellular components occurs when ROS concentrations exceed this threshold. DNA, Proteins, Carbohydrates, and Lipids are the building blocks of a well-balanced system in the human body. Damage to these biomolecules by ROS results in oxidative stress, which disrupts the metabolic state, cell growth, and development, all of which contribute to illness. When reactive oxygen species (ROS) are generated, oxidative stress causes damage to nitrogenous bases and strand breaks in DNA. radicals such as the superoxygen atom, the hydroxyl radical, and hydrogen peroxide (H_2O_2), for example] Hydroxyl radicals, superoxides, and hydrogen peroxide

are only a few of the radicals our bodies produce, with hydrogen peroxide standing out since it easily.

2.4.1 The role of stress markers in infertility and fertility

Aerobic metabolism produces ROS, or reactive oxygen species (ROS), which affect cellular function and may disturb the intracellular environment if antioxidants are not present to balance them (Agarwal *et al.* 2005). Some data suggests that ROS may have a role in the pathophysiology of both infertility and assisted reproduction (Agarwal *et al.* 2004).

Many oxidative indicators have been investigated, and greater total antioxidant concentrations have been associated to ovarian stimulation efficiency in infertile patients and pregnancy success. To maintain the cellular and extracellular redox balance, antioxidant enzymes of the glutathione system are essential (Erel 2004, Agarwal *et al.* 2005, Dalto 2017).

In contrast to viable oocyte donors and high-response patients, young patients undergoing ovarian stimulation cycles had greater quantities of antioxidant enzymes in their follicular fluid, according to Nunez-study (Nuez Calonge's *et al.* 2016). It seems that dietary deficiencies, hormonal imbalances, and physical microenvironmental variables such oxidative stress impact oocyte development and maturity (Agarwal *et al.* 2005).

2.5 Malondialdehyde

In the world of organic chemistry, you'll find the malondialdehyde (MDA) CH2(CHO)2. Malondialdehyde, a colorless liquid, is a highly reactive chemical found in enol. As an indicator of oxidative stress, it is found in abundance in nature. Polyunsaturated fatty acids undergo lipid peroxidation, resulting in the formation of malondialdehyde (Davey *et al.* 2005).

Platelets and other cell types and tissues use it to convert arachidonic acid to prostaglandin H2 via the thromboxane A2 production enzyme cyclooxygenase 1 or cycloxygenase 2. Malondialdehyde and 12-hydroxyheptadecatrienoic acid are the end products of this product's thromboxane synthase metabolism (Nair *et al.* 2009).

One of the numerous electrophile species, malondialdehyde, may be used to measure the rate of lipid peroxidation (Davey *et al.* 2005). A reactive aldehyde, MDA is an aldehyde. Malondialdehyde is formed when reactive oxygen species destroy polyunsaturated lipids. An aldehyde that reacts Many electrophile species, including MDA, generate toxic stress in cells and advanced lipoxidation end products (ALE) by creating covalent protein adducts, similar to advanced glycation end products (AGE) (Nair *et al.* 2009)

As a biomarker for oxidative stress, the generation of this aldehyde may be measured in an organism (Papac *et al.* 2016). When malondialdehyde combines with DNA, it forms adducts, the most common of which is M1G, which is mutagenic. The 2-amino pyrimidines are produced when malondialdehyde reacts with the guanidine group of arginine residue (Davey *et al.* 2005).

2.5.1 Malondialdehyde and its reaction

As a result of polyunsaturated fatty acid peroxidation and arachidonic acid metabolism, MDA is extremely reactive. When polyunsaturated lipids are degraded by reactive oxygen species (ROS), a reactive species known as MDA is formed. MDA has been found in animal tissues, particularly when there is an antioxidant shortage. It is a biological marker for oxidative stress. Arachidonic acid oxidation is the primary source of the lipid peroxide MDA found in animal-based dietary products, according to recent research. Blood platelets and serum contain MDA, which is one of the most often used biomarkers for lipid peroxidation, lipid peroxidation plays a role in the pathogenesis and tissue damage caused by a variety of toxic substances, and MDA may be used as a biomarker. MDA generates a DNA adduct after interacting with deoxyadenosine and deoxyguannosine in DNA, which have been linked to a number of chronic illnesses in

both people and animal systems, and there is evidence that lipid peroxidation levels in the kidney may be linked to renal toxicity. The carcinogenic heterocyclic compound pyrimido is the major one (1,2-a). Condensation of MDA and the guanidine group of arginine residues yields 2-aminopyrimidines; the laboratory detection and quantification of MDA is based on its ability to condense with two equivalents of thiobarbituric acid and give a fluorescent red derivative that can be assayed by spectrophotometry, or alternatively, 1-methyl-2-phenylindole with a higher selectivity can be used. Patients with keratoconus and bulloskeratopathy had a higher MDA level in their corneas. Tissue sections from people with osteoarthritis may also contain it (Singh *et al.* 2014).

2.5.2 MDA as indicator of lipid peroxidation

A biomarker for lipid peroxidation and oxidative stress is Malondialdehyde (MDA). The MDA assay has been utilized as a metric by various researchers for a variety of sample types. MDA and MDA-DNA adduct detection has been discovered to be a significant technique in determining the correlations between elevated levels of oxidative stress and the emergence of many diseases, including cancer. A viable method for assessing oxidative stress levels and determining their correlations with various disease patterns is MDA estimates (Roede *et al.* 2010).

An significant biomarker of lipid peroxidation may be found by measuring MDA levels in a variety of different biological systems. MDA-DNA adducts are formed as a result of endogenous MDA production and interaction with DNA under intracellular oxidative stress, making it an essential biomarker for endogenous DNA damage (Zhang *et al.* 2002). MDA is a TBARS (Thiobarbituric Acid Reactive Substances), which are a marker of lipid peroxidation, and may be measured in a variety of samples, including serum, plasma, or tissues. Various procedures are used to assess MDA levels in different samples, including serum, plasma, or tissues. The most often used technique for determining MDA is the TBA test (Singh *et al.* 2014).

2.6 Glutathione

Glutathione is one of the most significant antioxidants in the body, neutralizing free radicals and reactive oxygen species, as well as protecting vitamins C and E from oxidative damage. Antioxidants, detoxifiers, and the immune system's savior, glutathione is the most important of them all (Drigen 2000). Cellular and organismal health is dependent on GSH, which is an essential redox-active biomolecule. GSH levels in RBCs have been linked to a wide range of diseases, including but not limited to: hormonal imbalance (Taylor and colleagues 1996, Hazelton *et al.* 1983, Hazelton *et al.* 1980).

The availability of GSH in red blood cells (RBCs), on the other hand, has been linked to long life and excellent health in old people (Lang *et al.* 2002). Prior to using GSH levels as a therapeutic target or a diagnostic biomarker, there are significant obstacles to overcome. GSH levels in RBCs vary widely across healthy persons, independent of their age or method of testing, according to many studies. Intra-individual (i.e. inside a person) GSH levels in RBCs, on the other hand, remain rather consistent across time (Richie *et al.* 1996, Mills *et al.* 1994). A large amount of the variation in GSH, glutathione disulfide (GSSG) concentrations, and Ehc status in RBCs is attributed to genotypic variations.

2.6.1 Glutathione and female infertility

Folliculogenesis requires the presence of glutathione in order to protect eggs from oxidative stress. In the research, higher levels of glutathione in oocytes result in stronger and healthier embryos (Mukherjee *et al.* 2014). Another research found that the intracellular glutathione levels in women's ovaries are greater when they are younger (Kankofer *et al.* 2013). Premature aging of the ovaries and possibly ovarian cancer have been linked to glutathione deficiency (Lim *et al.* 2013).

Glutathione levels in the follicles of women undergoing IVF were shown to boost fertilization rates in another investigation (Tola *et al.* 2013). Previous studies have shown that Glutathione is an anti-aging antioxidant that may have consequences for egg health, which is among the oldest cells in our bodies (Fujii *et al.* 2005). follicle stimulating hormone's ability to shield developing embryos from damage is mostly due to its ability to increase glutathione levels (Tsai-Turton and Luderer 2006). Defending the reproductive system against oxidative stress is a fundamental function of glutathione, which is the cell's principal antioxidant. (Gardiner *et al.* 1998).

2.7 Insulin Resistance

Noninsulin-dependent diabetes mellitus is characterized by insulin resistance as a major pathological abnormality (NIDDM). Despite the fact that peripheral insulin resistance in PCOS patients was well-known by the mid-1980s, no one had examined the link between glucose tolerance and hyperinsulinemia when we started our study in 1987. During an oral glucose tolerance test, we discovered that obese PCOS women had considerably higher glucose levels than ovulatory hyperandrogenic and control women who were both the same age and weight as the obese PCOS women. According to the National Diabetes Data Group Criteria, 20% of obese PCOS women had impaired glucose tolerance or frank NIDDM (Dunaif and Book 1997).

2.7.1 Insulin resistance and PCOS correlation

Glucose intolerance is much more common in people with PCOS because of insulin resistance and insulin secretion irregularities, which are linked to obesity. PCOS-related insulin resistance is a significant contributor to noninsulin-dependent diabetic mellitus (NIDDM) in women, which is not surprising given the prevalence of PCOS. Serine phosphorylation of insulin receptors has been linked to insulin resistance in at least half of women with PCOS.

This anomaly is caused by an insulin receptor extrinsic factor, most likely a serine/threonine kinase, and is an essential new mechanism for human insulin resistance connected to insulin receptor signaling factors. There is evidence that the primary regulator of androgen production, serine phosphorylation, is modulated by this process. Some PCOS women may have insulin resistance and hyperandrogenism as a result of a single genetic flaw. Several recent investigations have shown evidence that insulin increases ovarian and pituitary steroidogenesis as well as LH secretion in PCOS by binding to its own receptor.

Indeed, the insulin deficiency seems to be selective, impacting glucose metabolism but not cell growth. To explore the ontogeny of abnormalities in glucose metabolism and to discover big three-generation kindreds in order to uncover NIDDM genes, PCOS is an ideal model. Despite the fact that the existence of lipid abnormalities, dysfibrinolysis, and insulin resistance would seem to put PCOS women at an increased risk of cardiovascular disease, suitable prospective studies are needed to verify this (Dunaif and Book 1997).

2.8 Obesity and BMI

In order to calculate a person's body mass index (BMI), weight in kilograms is divided by their height in meters squared. Adult obesity is defined by this formula in international standards. Due to its inability to properly assess fat mass, the BMI is an inaccurate indicator of body weight. Physiologically plausible methods may explain how obesity may impact AMH production. Those with obvious PCOS exhibited higher serum AMH levels than women without PCOS, despite their elevated AMH levels. Serum AMH levels and testosterone levels have been shown to have a positive correlation in patients with PCOS. In addition, the AMH levels of obese and overweight women were shown to be significantly lower than those of normal-weight women, as well. The BMI has the advantage of being simple, but it has limitations since it does not discriminate between fat and lean body mass in epidemiological research (Er Luo *et al.* 2021).

2.8.1 Obesity and BMI With AMH and pcos corelation

The findings of a prior research on the relationship between BMI and serum AMH were mixed, with some finding a negative association and others finding no such connection. As a consequence of these disparate findings, it is unclear if BMI can be utilized therapeutically to estimate blood AMH levels. The link between BMI and serum AMH has to be studied further with a bigger sample size, therefore further research is needed (Bahadur *et al.* 2021).

2.9 Lipid with AMH and PCOS Correlation

Obesity contributes to PCOS in part by stimulating lipolysis and altering lipoprotein lipase and hepatic lipase expression. Due to an increase in lipogenesis, an increase in triglyceride accumulation, and an increase in VLDL particle release by the liver, plasma triglyceride concentrations may rise. Hyperandrogenism, insulin resistance, and enhanced hepatic lipase activity all lead to an altered lipid profile and a rise in HDL-C particle catabolism. Changes in lipids may lead to an increased risk of coronary artery disease and early atherosclerosis. PCOS patients reported greater levels of AMH and cholesterol and triglycerides in comparison to control individuals in previous studies. They also had lower levels of HDL. Researchers discovered that having a higher AMH level increases one's chance of developing PCOS, hyperlipidemia, and other health issues.

Some 70% of women with PCOS were impacted by lipid abnormalities connected to insulin resistance, obesity, and lipolysis stimulation, as well as altered expression of lipoprotein and hepatic lipase. It is possible that the rise in plasma triglycerides is caused by an increase in lipogenesis, which results in increased liver secretion of VLDL particles and, as a result, a higher level of plasma triglycerides. Hyperandrogenism may also contribute to an elevated lipid profile, as it has been linked to an increase in hepatic lipase activity and may play a role in HDL-C catabolism, It is thought that LDL particles are atherogenic and have a significant connection to coronary artery disease because of the elevated risk of early atherosclerosis that they pose (Paneri *et al.* 2018).

2.10 Vitamin D

Most of the body's vitamin D comes from sunshine exposure; just 10% of vitamin D is derived from the food that we eat. Vitamin deficiency affects almost a billion individuals throughout the globe. It is crucial to note that the vitamin D receptor is found in many reproductive organs, and so has a significant impact on reproduction (Seifer and Tal 2016).

2.10.1 D vitamin and AMH correlation

The hypothesis is that vitamin D has a direct influence on the generation of AMH in women who are able to retain their ovarian reserve for a longer period of time (Pearce *et al.* 2015). Ovarian reserve and the ovarian response to ovarian stimulation have not been shown to be linked to vitamin D levels, which suggests that vitamin D may be a positive regulator of AMH synthesis (Fabris *et al.* 2017, Irani *et al.* 2014). As the ovarian reserve is maintained for a longer period of time, a significant seasonal vasopressin (Vasopressin) concentration has been discovered in these people (Revelli *et al.* 2009). It is possible that Vitamin D insufficiency may be linked to a reduced ovarian reserve in women of reproductive age, since serum AMH levels are positively connected with serum 25 hydroxy-vitamin D levels in women of childbearing age (Grzechocinska *et al.* 2013).

Serum AMH levels in women with PCOS are unusually high, and Vitamin D administration reduces this abnormally high AMH level, which may be one method by which Vitamin D improves the ovulatory dysfunction reported in women with PCOS. It is possible to employ Vitamin D as a therapeutic supplement for ovarian health in women of reproductive age if we can determine the function of supplementation, dosage, and duration as well as serum cutoff values for this inexpensive, readily available, and generally safe vitamin. Specifically in women with PCOS and low ovarian reserve, adequate research are needed to determine the impact of vitamin D in female reproductive health (Seifer and Tal. 2016).

3. MATERIALS AND METHODS

3.1 Subjects

Between january 2022 and march 2022, the chemistry and biochemistry Department of Health Nineveh/ jamhoury teaching /Oncology and nuclear medicine hospital conducted a prospective study. This study included 100 Iraqi women ranging in age from 20 to 55. The following categories were used to split people into groups.

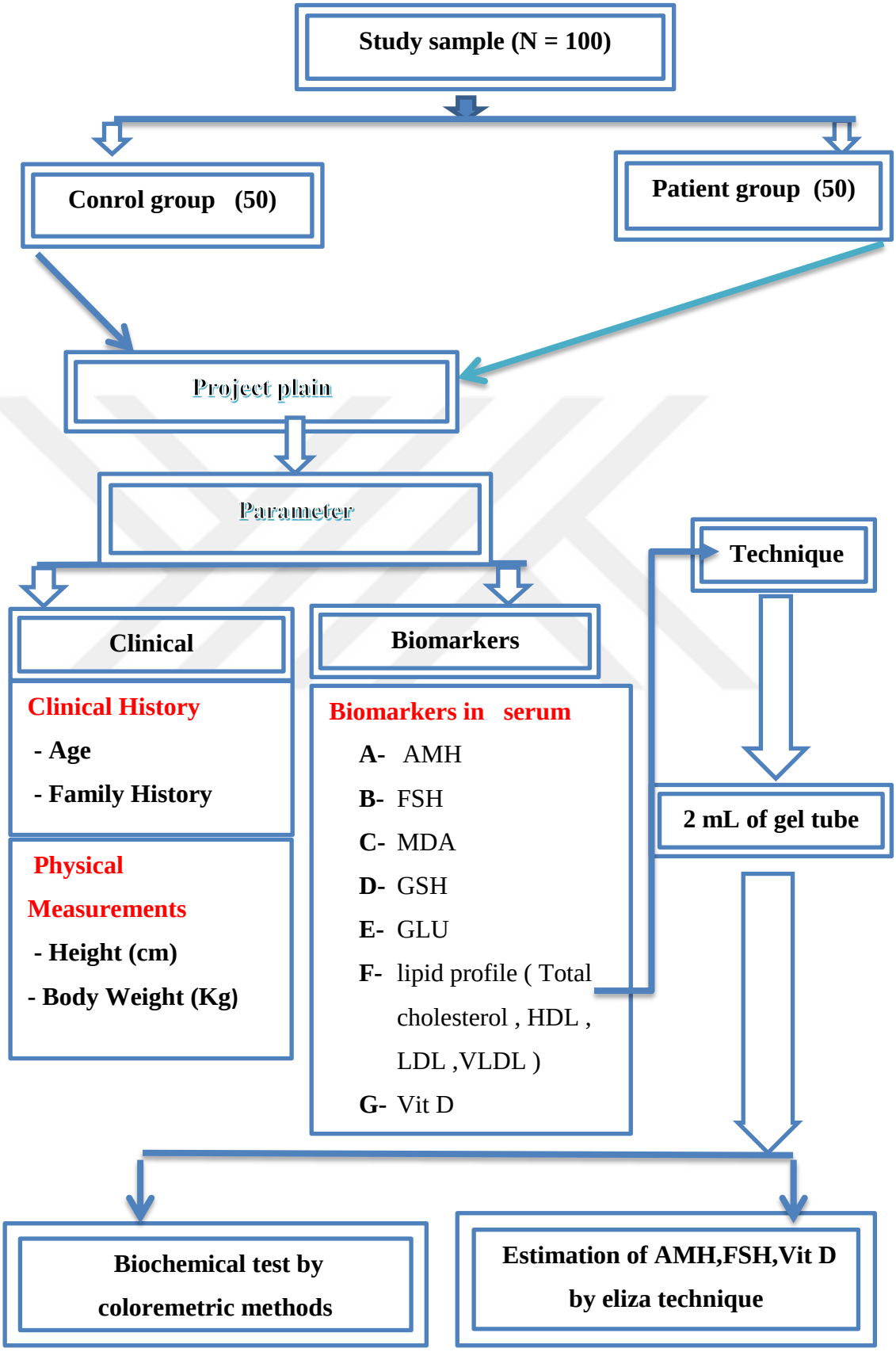
3.1.1 Patients groups (poly cystic ovary syndrome)

In january 2022 and march 2022, researchers at the Health Nineveh/ jamhoury teaching /Oncology and nuclear medicine hospitals collected study samples from patients. two groups of 100 Iraqi patients were enrolled in this study. We selected representative PCOS patients from a larger sample based on disease progression and data availability. PCOS was identified in 50 people lived at different areas (the city center and the Rural mountain areas) , all patients had PCOS. Take the patient's medical history (age, weight, Height and family history).

3.1.2 Healthy control group (normal women)

The control group who participated in this study include seventy five (50) apparently healthy women, from consultation clinic of jamhoury teaching /Oncology and nuclear medicine hospitals in the the Health Nineveh.

3.2 Diagram of Project



3.3 Materials

3.3.1 Instruments and equipment

General equipment and apparatus that were utilized during research are outlined in below Table 3.1.

Table 3.1 Researchers employed a variety of materials and compounds in their research

No	Equipment	Origin	Company
1	Deep freezer	Germany	Promega
2	Spectrophotometer	Japan	APEL,
3	Gel tubes	China	Xinle
4	Pipet.	Germany	Eppendorff
6	Centrifuge	Japan	H-19F Kokusan,
8	ELISA reader	Germany	Human
11	ELISA washer	Germany	Human
14	Incubation	China	Jrad
15	Plain tubes	Jordan	Sun
21	Tips (blue, yellow)	Jordan	AFCO,
22	Racks for plain tubes	China	Deans gate
23	Refrigerator	lebanon	Concord
25	Sterilized Latex Surgical Gloves	china	Deans gate
26	Cool Box	Germany	GenoMX
27	Water bath	Germany	Human,
28	Water distilater	Germany	GIF

3.3.2 Kits used in study

Lists the general kits studied Table 3.2.

Table 3.2 The kite that was utilized in the experiment.

No	Apparatus	Origin	Company
1	AMH Kit	Germany	Ansh
2	FSH Kit	Germany	Human
5	Cholesterol Kit	Germany	Human
6	GLU Kit	Germany	Human

3.4 Methods

Everyone who participated in the study was interviewed, provided samples, and asked questions about their family history, age, and weight, among other things. Blood samples were collected from patients, from each patient 5 ml in a disposable syringe. We placed the sample using a gel tube and 15 min at room temperature, Serum was collected and kept in the freezer (-20 °C) for future use after centrifugation at 3000 rpm for ten minutes until it's time for tests.

3.5 Calculation of Body Mass Index (BMI)

Obesity measurement and analysis It was determined by dividing the weight (kg) and height (m) squared values (m). BMI's equation (in meters):

BMI = Mass (kg)/ Height (m2)

BMI was categorized into the following:

- BMI less than 18.5 (underweight).
- BMI less than 24.9 (normal weight).
- Between 25 – 29.9 (over weight).
- More than 30 (class I) moderately obese.
- More than 35 (class II) severely obese.
- More than 40 (class III) very severely obese (Whitney and Rolfes 2005).

3.6 Estimation of (AMH Hormone) by ELISA

Prıncıple Of The Test : A sandwich-based ELISA, the DEMEDITEC AMH ELISA is an enzyme-linked immunosorbent test (ELISA) in solid phase. A monoclonal (mouse) antibody is used to coat the microtiter wells in order to target a specific antigenic site on the AMH molecule. During the first incubation, the AMH in the added sample binds to

the immobilized antibody. The enzyme conjugate, which contains an AMH antibody conjugated to horseradish peroxidase, binds to the AMH in the sandwich complex.

To detect unknown sample concentrations, OD measurements are plotted against concentrations of standards to form a standard curve. The amount of analyte in a sample is directly related to the color of the final product.

3.7 Estimation of FSH Hormone by ELISA

Principle: Human FSH ELISAs are only meant to be used by doctors. The initial phase of the ELISA for direct antigen detection is to combine a biotinylated monoclonal anti-FSH and a peroxidase-labeled anti-FSH enzyme conjugate to generate a particular immunocomplex, which binds to the surface of the wells at the conclusion of the incubation time. The absorbance of calibrators and specimens is measured using ELISA microplate readers or automated ELISA systems (such as those from HUMAN's Huma-Reader or ELISYS lines), and the concentration is evaluated by measuring the color, which turns yellow after stopping the reaction with the stop solution (step 2). Step 2 involves adding TMB/Substrate and measuring the resultant color, which becomes yellow once the reaction is stopped with the stop solution.

3.8 Measurement of MDA in Serum

Principle: MDA TBA2 is formed when malondialdehyde interacts with thiobarbituric acid (TBA), the malondialdehyde level in the blood can be identified using the modification approach proposed by (Schmedes and Holmer 1989).

3.9 Measurement of GSH in Serum

Principle: The tripeptide is a GSH (y-glutamyl cysteinyl glycine) (Glu-Cys-Gly), The y-glutamyl cysteine symptoms and GSH synthetases are produced together to generate cysteine and glycine, leading to two processes reliant on ATP.

Elman test use to measurment glutathion by using 4% sulfosalicylic acid solution and 5-5 dithio bis- 2 nitro benzoic acid in phosphate solution wth pH=8, Spectrophotometer wave lenght 412 nm.

3.10 Determination of Plasma Level of Glucose

Principle: To determine the concentration of glucose in a sample, peroxidase (POD) reacts with 4-aap and phenol to generate a colored complex whose intensity is proportional to the concentration of glucose in the sample produced by glucose oxidase (GOD).

3.11 Determination of Plasma Level of The Cholesterol

Enzymatic hydrolysis and oxidation are used to determine the cholesterol. In the presence of phenol and peroxidase, hydrogen peroxide and 4-aminoantipyrine are converted to indicator quinoneimine Equation 3.1, Equation 3.2 and Equation 3.3.

$$Cholesterolester + H2O \xrightarrow{CHE} cholesterol + fatty\ acid \quad (3.1)$$

$$Cholesterol + O2 \xrightarrow{CHO} cholestene - 3 - one + H2O2 \quad (3.2)$$

$$H2O2 + 4 - amino\ antipyrine + phenol \xrightarrow{POD} quinoneimine + 4\,H2O \quad (3.3)$$

4. RESULTS AND DISCUSSION

4.1 Statistical Analysis

When comparing two measuring methods graphically, we utilized the Bland Altman plot (also known as a difference plot) and the Spss plot for Windows version (25) The disparities (or ratios, depending on your preference) between the two methods are shown graphically versus the averages of the two methods.

It is possible to plot the differences against one of these two techniques as a reference, and then draw horizontal lines to depict the mean difference and limits of agreement, which are specified as a variance plus or minus 1.96 times a standard deviation of the differences.

The averages and standard deviations of continuous data were presented, whereas the percentages of categorical variables were expressed. Every statistically significant P (sig) less than 0.05 was considered. The t-test is utilized to essentially compare implies. Comparisons of subgroups were made using a post-hoc statistical test, In this study, the estimation of the correlation coefficient between the variables.

4.2 Comparison of the Mean Levels of Age, BMI, AMH, FSH, Glu, Ch, MDA and Gsh in Control and PCOS Groups

In this study, 100 women between the ages of 30 and 60 were compared characteristics into two PCOS and control groups, the mean difference in AMH between PCOS and control groups was highly significant, The mean AMH Difference was 5.46327 (P=0.000*,95%CI: 4.7337, 6.1928) with Std. Of 2.53999and T-test is (15.056) Table 4.1.

Furthermore, the mean difference between PCOS and control groups of FSH was highly significant, the mean FSH Difference was -14.96327**(P=(0.000*,95%CI: -23.0084, -6.9181) with Std. Of 28.00906, T-test is -3.740 see table ( ).

The data show that the age difference between PCOS and control groups was quite significant, the mean Age Difference was -3.02041*(P=0.033,95%CI: (-5.7826, -5.7826) with Std. Of 9.61659 andT-test is (-2.199), the Table 4.1 and Figure 4.1.

In contrast, there was non significant in the Mean difference between PCOS and control groups of BMI, the mean BMI Difference was -3.02041* (P=0.900,95%CI: -1.2158, 1.0724) with Std. Of 3.98317 and T-test (-0.126)the Table 4.1 and Figure 4.1.

Moreover, there was non statistically significant difference in the mean between PCOS and control groups of (GLU, CHO, MDA, and GSH), the mean Difference was (12.34694, 3.73469, 0.47426, 0.19902) (P=0.092, P=0.307, P=0.093, P=0.627) respectively, the other data are reported on the Table 4.1 and Figure 4.1.

Table 4.1 Comparison of one sample test of the mean difference for Age, BMI, AMH, FSH, Glu, Cho, MDA and Gsh.

	Test Value = 0					
	t	Std. Deviation	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
diff_age	-2.199	9.61659	.033	-3.02041	-5.7826	-.2582
diff_bmi	-.126	3.98317	.900	-.07168	-1.2158	1.0724
diff_Amh	15.056	2.53999	.000	5.46327	4.7337	6.1928
diff_fsh	-3.740	28.00906	.000	-14.96327	-23.0084	-6.9181
diff_glu	1.719	50.27821	.092	12.34694	-2.0946	26.7885
diff_cho	1.033	25.30133	.307	3.73469	-3.5327	11.0021
diff_mda	1.712	1.93899	.093	.47426	-.0827	1.0312
diff_gsh	.489	2.85024	.627	.19902	-.6197	1.0177

-Sig. (2-tailed) is *p*-value
-CI is Confidence Interval and the Confidence Interval of the Difference is 95%
-A negative value for the mean difference of control is bigger than the patient.
-The results in yellow colour mean highly significant

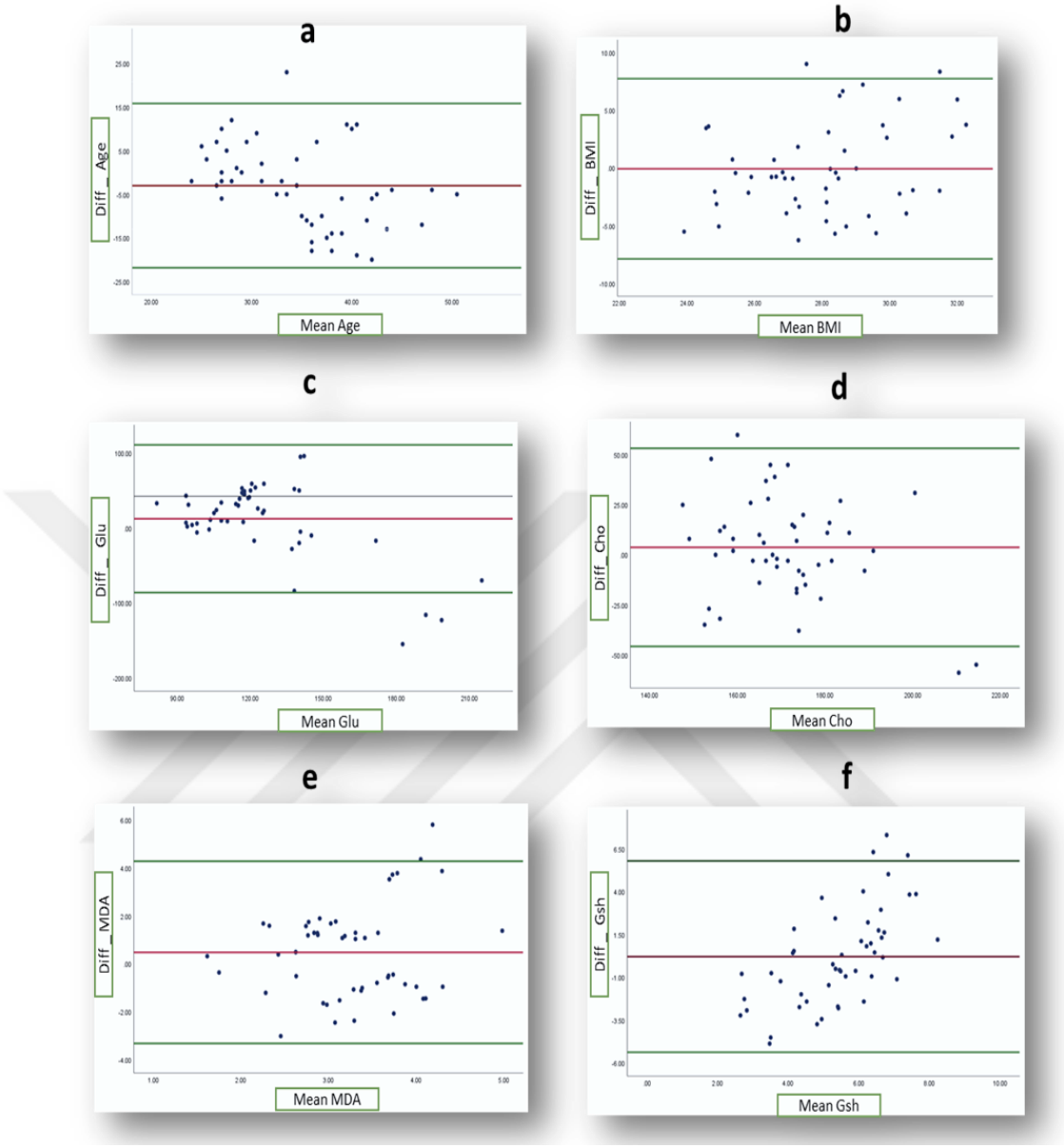


Figure 4.1 Comparison of one sample test of the mean difference for Age, BMI, AMH, FSH, Glu, Cho, MDA and GSH

Fig. A: -Upper and lower limits compared with the mean difference in age data
-Redline is The mean of the difference.
-Greenline, upper and lower limits of differences ((out layer)).
-If the values are close to the redline that means no statistically significant difference.
Fig. B: -Upper and lower limits compared with the mean difference in BMI data
Fig.C: -Upper and lower limits compared with the mean difference in GLU data
Fig. D: -Upper and lower limits compared with the mean difference in CHO data
Fig. E: -Upper and lower limits compared with the mean difference in aMDA data
Fig. F: -Upper and lower limits compared with the mean difference in GSH data

4.3 Correlations of One Sample Test of the Mean Difference Between Age, BMI, AMH, FSH, Glu, Cho, MDA and GSH

Using the R-value of 0.447 and p-value of 0.01, the study found a strong association between AMH and MDA Table 4.2.

Free radicals have an important physiological role in the female reproductive system, and high levels of free radicals may cause disorders of the female reproductive system, as has been previously shown by study (Ashok Agarwal *et al.* 2005).

As lipid peroxidation products, MDA has been related to a variety of chronic disorders in people and animal models of these diseases (Singh *et al.* 2014). Conversely, There was not significant correlation between AMH and (BMI, FSH, Glu, Cho) with the R-value of (0.128, 0.157, 0.140, 0.101) and p-value of (0.379, 0.282, 0.336, 0.491) respectively.

As for the result of the relationship between FSH and AMH hormones, it does not agree with previous studies, which showed a negative association between FSH and AMH serum levels(Singer *et al.* 2009). The reason for this may be attributed to that the women participating in the research are of different ages and conditions (perimenopausal, postmenopausal, fertility, infertility, PCOS).

There have been conflicting results from research on the relationship between BMI and serum AMH. Some have found a negative link while other studies have shown no correlation (Bahadur *et al.* 2021).

In earlier studies, women with and without PCOS were shown to have equal levels of insulin resistance when AMH was measured. However, recent research contradicts this finding, which might be attributable to the aging process or another factor (Luciano *et al.* 2009).

As previously reported, AMH levels in women were shown to have a negative link with Cho at the lowest AMH percentiles, whereas AMH levels in women at the highest AMH percentiles were found to have a positive correlation with HDL (Lim *et al.* 2021).

while, a negative correlation was found between AMH and (GSH, Age) the R-value of (-0.092, -0.147) and p-value of (0.0529, 0.314) respectively, the Table 4.2.

This is disagreed with research published in 1996 included that Significant correlation between lower levels of GSH in (RBC) and hormonal imbalance was indicated (Taylor *et al.* 1996). GSH's function may be dependent on a balance between reactive species formation and antioxidant defense, which suggests that the GSH/free radicals ratio should be greater. The result between AMH and Age is agreed with previous study that when age increases, AMH decreases(Naughton *et al.* 1992), Because fertility decreases.

The statistical estimates was clarified that a significant correlation between FSH and Age the R-value of 0.519 and p-value of 0.00the Table 4.2. The research published in 1992 agreed with our conclusion that the age has relationship with amh and fsh, when age increases, AMH decreases and FSH increases (Naughton *et al.* 1992).

Follicle growth and production are induced by FSH, which is why this occurs. Estrogen levels drop as a woman aged because fewer follicles need to be stimulated. As a result of the drop in estrogen levels, the brain's synthesis of the hormone FSH rises. FSH and GLU also showed a strong association (R=0.373, p=0.008), as shown in Table 4.2 (see below). In postmenopausal women with normal or impaired fasting glucose, the FSH concentration has been linked to insulin resistance, prediabetes, and diabetes in earlier research (Stefanska *et al.* 2019).

In contrast,there is not significant correlation between FSH and GSH with the R-value of 0.229 and p-value of 0.114. This is disagreed with previous study confirmed that Higher Gsh activity is associated with higher FSH level, but the difference may be due

to obesity in the abdominal area, as it is the main determinant of this relationship(Klisic *et al.* 2018).

Negative correlations are indicated between FSH and (Cho, MDA, BMI) with R-value of (-0.059, -0.159, -0.118) and p-value of (0.686, 0.276, 0.419) respectively, in Table 4.2. The research published in 2020 disagreed with current result, the data of this research suggest that higher FSH in postmenopausal women is related to higher levels of both Cho and LD (Serviente *et al.* 2019). The reason of difference for that the relation between lipids and FSH varies by age.

Furthermore, a 2014 study found that MDA had a positive connection with FSH, contradicting the conventional consensus that FSH and MDA are not linked (Ogunro *et al.* 2014).

The reason for the difference is due to the different values of fsh in different conditions (perimenopausal, postmenopausal, fertility, infertility, PCOS). (Kiddy *et al.* 1990) found an inverse correlation of FSH with BMI in obese PCOS, this agree with current study(Kiddy *et al.* 1990).

The data were expressed that a not significant correlation between GLU and (Age, BMI, Cho, GSH) the R-value of (0.208, 0.228, 0.233, 0.084) and p-value of (0.151, 0.116, 0.107, 0.568) respectively. The relationship between GLU and Cho may be seen in the chart below. In a 2017 study, researchers found that people with familial hypercholesterolemia had a reduced chance of developing type 2 diabetes if their LDL levels are elevated (the higher the LDL-C the lower the risk).

In contrast, the correlation between GLU and Age disagree with observation of some studies, that showed age had a direct effect on blood glucose level, The effect of age on blood glucose was also mediated by BMI (Fikriana and Devy 2018). found a positive correlation between fasting blood glucose level and BMI, This is inconsistent with the current results.

In addition, a 2013 study concluded that GSH depletion was not a significant factor in the etiology of diabetes Mellitus (Kalkan and Suher 2013). The mismatch in the results is due to the fact that the results depend on several factors like (abdominal fat mass, low physical activity, mitochondrial dysfunction, and hormonal changes). Whereas, there was a negative correlation in current research between GLUand MDA the R-value of -0.266 and p-value of 0.064.

According to the findings of SIR Okoduwa *et al.* (2013), a rise in the MDA level is linked to hyperglycemia because of the autooxidation of glucose, which generates free radicals. As a consequence, an increase in free radicals (such as MDA) and a decrease in antioxidant defenses may contribute to the development of diabetic problems (Okoduwa *et al.* 2013).

The reason for the difference in the results may be due to the level of free radicals and antioxidant defenses. According to the results obtained from the study, a strong significant association between Cho and BMI was discovered, with an R-value of 0.590 and a p-value of 0.00. Increasing BMI is linked to greater total cholesterol and low-density lipoprotein cholesterol, according to a study published in 2011 that also found the same result (LDL) (Lior *et al.* 2011).

It has been obtained a negative correlation with Cho and (Age, GSH) with the R-value of (-0.119, -0.048) and p-value of (0.417, 0.742) respectively. Published in 2001, a paper that supported our hypothesis that age-dependent reductions in cholesterol may be due to poor health status, and low cholesterol in older people may serve as a signal of poor health (Volpato *et al.* 2001).

Also, previous article found that an increased cholesterol level was associated with decreased concentrations of glutathione(Rajasekaran *et al.* 2005). Furthermore, the R-value of 0.085 and p-value of 0.0.563 indicate that there is a non-significant correlation between Cho and MDA, the Table 4.2.

This is confirmed by previous research that finding Insignificant positive correlation between MDA and total cholesterol(Rao and Kiran 2011). Through the final results of the study, it was found that a negatively strong significant correlation between MDA and Age the R-value of -0.372 and p-value of 0.008 .

Both gender and age were shown to have a substantial impact on malondialdehyde (MDA), with women showing a stronger dependency on their age than males (Pinchuk *et al.* 2019). In addition,a negative correlation between MDA and GSH with the R-value of -0.137 and p-value of 0.349, all data are in the Table 4.2.

According to another study on the association between MDA and GSH, the higher the MDA level, the lower the glutathione level in the body. This is in agreement with the findings of that study (Tualeka *et al.* 2019). Current research observed not significant association between MDA and BMI the R-value of 0.062 , p-value of 0.670, the Table 4.2.

This is not similar to a previous study, that indicates the concentration of MDA increased with increasing BMI. As well as, observations confirm that GSH had not significant correlation with(Age, BMI) the R-value of (0.137, 0.07) p-value of (0.235, 0.964) all data are reported in Table 4.2.

Conversely, another article found that glutathione peroxidase activity was tend to decreased with increasing age and body mass index.

Some samples from women with PCOS have higher cholesterol, triglycerides, LDL, and VLDL, as well as lower HDL values. AMH, cholesterol, triglycerides, LDL and VLDL levels, and HDL levels were all considerably higher in PCOS patients compared to control participants in previous research (Paneri *et al.* 2018).

This study (Grzechocinska *et al.* 2013), which found no significant differences between the PCOS and control groups in the levels of vitamin D in certain samples, hypothesized

that higher levels of vitamin D in PCOS patients might help them retain their ovarian reserve for longer periods of time (Grzechocinska *et al.* 2013). Moreover, vitamin D appears to be a positive modulator of AMH synthesis in other studies (Revelli *et al.* 2009).

Table 4.2 Correlations of one sample test of the mean difference between Age, BMI, AMH, FSH, Glu, Cho, MDA, GSH

		AGE	BMI	AMH (1.5-4)ng/ml	FSH (1.5_12.4)IU/L	GLU(90_120)mg/dl	Cho(130_150)mg/dl	MDA(2.02_4.65)mml/l	GSH(8.49_+6.3) mmol/l
AGE	Pearson Correlation	1	-.033	-.147	.519**	.208	-.119	-.372**	.173
	Sig. (2-tailed)		.822	.314	.000	.151	.417	.008	.235
	N	50	50	50	50	50	50	50	50
BMI	Pearson Correlation	-.033	1	.128	-.118	.228	.590**	.062	.007
	Sig. (2-tailed)	.822		.379	.419	.116	.000	.670	.964
	N	50	50	50	50	50	50	50	50
AMH (1.5-4)ng/ml	Pearson Correlation	-.147	.128	1	.157	.140	.101	.447**	-.092
	Sig. (2-tailed)	.314	.379		.282	.336	.491	.001	.529
	N	50	50	50	50	50	50	50	50
FSH (1.5_12.4)IU/L	Pearson Correlation	.519**	-.118	.157	1	.373**	-.059	-.159	.229
	Sig. (2-tailed)	.000	.419	.282		.008	.686	.276	.114
	N	50	50	50	50	50	50	50	50
GLU (90_120)mg/dl	Pearson Correlation	.208	.228	.140	.373**	1	.233	-.266	.084
	Sig. (2-tailed)	.151	.116	.336	.008		.107	.064	.568
	N	50	50	50	50	50	50	50	50
Cho (130_150)mg/dl	Pearson Correlation	-.119	.590**	.101	-.059	.233	1	.085	-.048
	Sig. (2-tailed)	.417	.000	.491	.686	.107		.563	.742
	N	50	50	50	50	50	50	50	50
MDA(2.02_4.65) mml/l	Pearson Correlation	-.372**	.062	.447**	-.159	-.266	.085	1	-.137
	Sig. (2-tailed)	.008	.670	.001	.276	.064	.563		.349
	N	50	50	50	50	50	50	50	50
GSH(8.49_+6.3) mmol/l	Pearson Correlation	.173	.007	-.092	.229	.084	-.048	-.137	1
	Sig. (2-tailed)	.235	.964	.529	.114	.568	.742	.349	
	N	50	50	50	50	50	50	50	50

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
The results in green colour mean highly significant

4.4 The Effect of Independent Variables (Subgroups) on AMH Using Regression

Logistics regression was used between AMH and subgroups, a posetively significant correlation was observed between AMH and GLU(B=0.280, *p*=0.059) (T=1.943) These data are also provided in Table 4.3. This is agreed with previous studies, Luciano G. Nardo *et al.*2009 indicated that AMH was similarly related to insulin resistance and

circulating androgens both in PCOS and non-PCOS subjects(Luciano *et al.* 2009). As well as, a posetively significant correlation was noticed between AMH and MDA (B=0.519,*p*=0.001)(T=3.506) are reported on the same Table 4.3.

Free radicals have a significant role in the female reproductive system, according to Ashok Agarwal *et al.* (2005), and this might lead to an increased risk of PCOS (Ashok *et al.* 2005). When it comes to measuring lipid peroxidation levels, MDA is one of the most often employed biomarkers. MDA plays an important role in the pathophysiology of many different kinds of tissue injuries and notably in the tissue damage inflicted by various harmful agents. A range of chronic disorders in people and animal systems have been linked to elevated levels of lipid peroxidation products (Singh *et al.* 2014).

As well as,there was a negatively correlation between AMH and (Age, Cho, GSH) (B= -0.010, B= -0.047, B=-0.046) (*p*=0.944, *p*=0.779, *p*=0.731) respectively, other data are shown in the Table 4.3. The previous study agreed with current result, The correlation between AMH and Age is reverse Because fertility decreases(Naughton *et al.* 1992).

There was a correlation found between AMH and Choesterol in the lower percentiles and a positive correlation found in the higher percentiles, based on the previous study's findings. Women with decreased ovarian function may have higher amounts of AMH if their lipid profile changes, according to these data (Lim *et al.* 2021).

Furthermore, research published in 1996 disagreed with current result included that there was asignificantly correlation between lower levels of GSH and hormonal imbalance(Taylor *et al.* 1996). The effect of GSH is depend on balance between the production of free radical and antioxidant defence. There was no significant correlation between AMH and BMI (B=0.061,*p*=0.714) the same Table 4.3. BMI and serum AMH had conflicting findings in a prior research, with some showing a negative correlation and others finding no correlation (Bahadur *et al.* 2021).

Table 4.3 The effect of independent variables (subgroups) on AMH using regression analysis

Coefficients[a]					
		Unstandardized Coefficients Std. Error	Standardized Coefficients Beta	t	sig
	(Constant)	4.237		.274	.785
	AGE	.052	-.010	-.070	.944
	BMI	.113	.061	.369	.714
	GLU(90_120)mg/dl	.014	.280	1.943	.059
	Cho(130_150)mg/dl	.024	-.047	-.283	.779
	MDA(2.02_4.65)mml/l	.262	.512	3.506	.001
	GSH(8.49_+6.3)mmol/l	.121	-.046	-.346	.731
Dependent Variable: AMH (1.5-4) ng/ml The results in red colour mean highly significant					

4.5 The Effect of Independent Variables (Subgroups) on FSH Using Regression Analysis

Logistics regression was applied between FSH and subgroups, there was a posetive significant correlation between FSH with Age(B=0.472, *p*=0.001) (T=3.590)the Table 4.4. Previous studies have shown that FSH rises with age (Naughton *et al.* 1992), and that this increase in FSH stimulates the ovarian follicles to grow and release estrogen (estradiol), estrogen levels decline as a woman matures because fewer follicles are activated. As a result of the decreased estrogen synthesis, the brain's production of FSH rises, which in turn causes an increase in FSH.

In addition, a posetively significant correlation was showed between FSH and GLU (B=0.339,*p*=0.013)(T=2.592) in the same Table 4.4. FSH levels were connected with decreased prevalence and incidence of type 2 diabetes and fasting insulin levels, according to a published study in 2017 (Elizabeth *et al.* 2017)

Whereas, a negative correlation was observesed between FSH and BMI(B= -0.211, *p*=0.163) as shown in the Table 4.4. This is confirmed by previous studiesthat found anegative association between FSH and BMI in obese PCOS (Kiddy *et al.* 1990).

Furthermore, there was no significant correlation between FSH and (Cho, MDA, GSH) (B=0.038, B=0.136, B=0.141)(p=0.803, p=0.310, p=0.254)other data as shown in the Table 4.4. A research indicated that FSH may be linked to lipid levels in postmenopausal women, and that the correlations between lipid levels and age may be influenced by these findings (Serviente *et al.* 2019).

FSH and MDA levels rose considerably throughout the perimenopausal and postmenopausal periods compared to the reproductive phase, according to another study (Ogunro *et al.* 2014).

For the correlation between FSH and GSH is disagreed with previous study, that confirmed Higher Gsh level is associated with higher FSH level, The reason for the inconsistency with the current results is due to obesity in the abdominal area, as it is the main determinant of this relationship (Klisic *et al.* 2018).

Table 4.4 The effect of independent variables (subgroups) on FSH using regression analysis

Coefficients[a]				
	Unstandardized Coefficients Std. Error	Standardized Coefficients Beta	t	sig
(Constant)	40.032		-1.903	.064
AGE	.493	.472	3.590	.001
BMI	1.071	-.211	-1.420	.163
GLU(90_120)mg/dl	.131	.339	2.592	.013
Cho(130_150)mg/dl	.230	.038	.251	.803
MDA(2.02_4.65)mml/l	2.471	.136	1.029	.310
GSH(8.49_+6.3)mmol/l	1.141	.141	1.157	.254
Dependent Variable: FSH (1.5_12.4)IU/L The results in blue colour mean highly significant				

5. CONCLUSIONS AND RECOMMENDATION

5.1 Conclusions

There is a well-known link between PCOS and elevated AMH levels. Polycystic ovarian syndrome (PCOS) has been linked to an increased AMH level by several investigations. PCOS blood samples showed a substantial correlation between MDA levels and AMH value.

Even during the menopausal years, there is some evidence that oxidative stress has an impact on a woman's reproductive lifespan and infertility. Many studies have shown that free radicals play an important role in the female reproductive system, precipitating diseases like miscarriage and gestational diabetes, which have been linked to the reproductive system. OS can develop when free radicals are produced in excess or antioxidant defense mechanisms are impaired (Ashok *et al.* 2005).

Elevated GLU levels were found in PCOS blood samples with high Amh levels. For the first time, it has been shown that insulin resistance may be linked to aberrant follicular growth in PCOS (Luciano *et al.* 2009). Polycystic ovaries (PCO), anovulation, hyperandrogenism and Insulin Resistance, dyslipidemia, hypertension, obesity, infertility, Type 2 diabetes, and many other complications have been shown to be directly and indirectly linked to increased AMH in the pathophysiology of reproductive and metabolic dysfunction in PCOS.

High cholesterol levels had a negligible impact on PCOS patients. PCOS has been linked to hyperlipidemia and other health issues, according to research. In comparison to control participants, PCOS patients had substantially higher levels of AMH, cholesterol, triglycerides, LDL, and VLDL, as well as lower levels of HDL, according to the studies (Paneri *et al.* 2018).

Low GSH concentration had little effect on AMH levels in PCOS patients' blood samples. According to research, Glutathione, a naturally occurring antioxidant, helps to preserve other antioxidants. Glutathione levels vary widely; it has been confirmed to play an important role in the fertilization process and early embryonic development; and it has been implicated in the preservation of germ cell biological value (Adeoye *et al.* 2018)

When AMH levels rise, the FSH level falls for individuals under the age of 40; however, when FSH levels fall for people beyond the age of 44, the opposite occurs. In light of the fact that FSH is secreted, researchers have shown FSH is an anti-menopausal hormone that increases the size of ovarian follicles and the production of estrogen. Because fewer follicles need to be stimulated as a woman gets older, her estrogen levels drop. Because there is less estrogen, the brain's synthesis of FSH increases because there is not enough estrogen to inhibit it.

When follicle stimulating hormone (FSH) levels are high in postmenopausal women, the hormone may interact with the LDL receptor in the liver and reduce the LDL receptor's levels, reducing the clearance of LDL cholesterol by endocytosis.

“The huge menopausal endocrine change, especially sharply raising the serum FSH levels, exhibited negative effects on lipid levels,” the researchers wrote. “the results of previous studies indicate that FSH may interact with its receptors in hepatocytes and reduce [LDL receptor] levels, which subsequently attenuate the endocytosis of [LDL cholesterol], resulting in an elevated circulating [LDL cholesterol] level.”(Song *et al.* 2015).

PCOS, high levels of AMH and FSH can lead to high blood sugar and type 2 diabetes. Insulin Resistance, dyslipidemia, obesity, infertility, type 2 diabetes and a range of other health issues are all linked to a high In FSH level.

Control blood samples were correlated with normal AMH values, often with normal values for cholesterol, FSH and Glu. The AMH value may decrease in control samples under the age of 40 years due to either ovarian failure, loss of ovarian function, or goiter. As for the control samples for ages between (50and53), the AMH drops below the normal limit with a high FSH value and an increase in cholesterol and GLU measurements.

Some studies have also found that FSH, which increases during menopause, is more strongly associated with health outcomes after menopause. The value of the MDA was within the normal limits in the healthy samples. No strong effect for vitamin D was observed on the PCOS cases. Vitamin D has been shown to have a favorable effect on the synthesis of AMH (Revelli *et al.* 2009).

5.2 Recommendations

1- Treating the symptoms of Polycystic ovarian syndrome, addressing the metabolic imbalances that underlie this condition and lowering one's chance of developing diabetes are all important aims for patients. If a patient wishes to get pregnant, the treatment plan is tailored according to her needs and typically starts with dietary and lifestyle changes.

Modifications in way of life:

Women with PCOS who are overweight or obese should begin by reducing their caloric intake and engaging in regular physical activity. This can help alleviate symptoms related to excessive androgen levels, insulin resistance, and decreased fertility.

Medications:

Treatment for PCOS may help control menstruation, ovulation, and the likelihood of insulin resistance in women, as well as help them become pregnant. The following are

examples of widely prescribed medications: (birth control pills, metformin, ovulation inducing medications, hair removal treatments)

Options for surgery:

Surgery is a possibility for people who have tried lifestyle modifications and medicinal treatment but have been unsuccessful in becoming pregnant.

2- When the level of the hormone AMH is low, this indicates a weak egg reserve, but the problem is that there are no ways to increase the levels of this hormone, but the chances of pregnancy can be increased if its levels are low by:(vitamin D supplements, L-arginine supplements, abdominal massage, getting rid of stress).

3- Also the following recommendations were be suggested:

The current research should be replicated with a bigger sample size at various universities. Efficacy and appropriateness of lifestyle management against anti-obesity pharmacological drugs and surgery for PCOS therapy should be examined in further study.

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