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**THE RELATIONSHIP BETWEEN OMENTIN-1 AND ADIPONIN IN
SERUM OF WOMEN WITH POLYCYSTIC OVARY SYNDROME
IN SAMARRA CITY**

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THE RELATIONSHIP BETWEEN OMENTIN-1 AND ADIPCIN IN SERUM OF
WOMEN WITH POLYCYSTIC OVARY SYNDROME IN SAMARRA CITY

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May 2022

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ABSTRACT

THE RELATIONSHIP BETWEEN OMENTIN-1 AND ADIPCIN IN SERUM OF WOMEN WITH POLYCYSTIC OVARY SYNDROME IN SAMARRA CITY

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Master of Science in Chemistry

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The study included evaluating the level omentin-1 and adipicin , as the study included a group of 90 blood samples, watching their ages ranged between (20-40) years, 60 samples infected with polycystic syndrome and 40 blood samples for healthy women , and PCOS samples were collected from samarra general hospital / salah al-din governorate, a bridge between 1/3/2021 to 1/7/2021. According to the findings of the present investigation, in comparison to the healthy group, the concentrations of omentin-1 and adipicin in the blood serum of PCOS patients were significantly higher. The levels of FSH, LH, and prolactin in the blood serum were significantly higher in the PCOS group compared to the healthy group, whereas the levels of these hormones were significantly lower. There was no significant difference between the PCOS group and the healthy group in terms of cholesterol or T.G levels, but HDL-C levels dropped significantly, while LDL-C and VLDL levels rose significantly.

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Keywords: Omentin-1, Adipicin, Polycystic ovary syndrome, Sera of women

ÖZET

SAMARRA İLİNDE POLİKİSTİK OVER SENDROMLU KADINLARIN SERUMLARINDA OMENTİN-1 VE ADİPCİN İLİŞKİSİ

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Çalışmaya omentin-1 ve adipicin düzeylerinin değerlendirilmesi dahil edilmiş olup, çalışma yaşları (20-40) yıl arasında değişen 90 kan örneği, polikistik sendrom ile enfekte 60 örnek ve sağlıklı kadınlar için 40 kan örneğinden oluşan bir grup ve PCOS örnekleri 1/3/2021 ile 1/7/2021 tarihleri arasında bir köprü olan samarra general hospital/ salah al-din governorate'den toplanmıştır. Mevcut araştırmanın bulgularına göre, PKOS hastalarının kan serumundaki omentin-1 ve adipisin konsantrasyonları sağlıklı gruba göre anlamlı olarak daha yüksekti. Kan serumundaki FSH, LH ve prolaktin seviyeleri PCOS grubunda sağlıklı gruba göre anlamlı derecede yüksek, bu hormonların seviyeleri ise anlamlı derecede düşüktü. Kolesterol veya T.G seviyeleri açısından PCOS grubu ile sağlıklı grup arasında anlamlı bir fark yoktu, ancak HDL-C seviyeleri önemli ölçüde düşerken, LDL-C ve VLDL seviyeleri önemli ölçüde yükseldi.

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Anahtar Kelimeler: Omentin-1, Adipicin, Polikistik over sendromu, Kadın serumu

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LIST OF SYMBOLS

μL	Microliter
cm^3	Cubic centimeter
mg	Milligram
mg/dL	Milligrams per decilitre
mL	Milliliter
mmol/L	Millimoles per liter
ng/mL	Nanograms per milliliter
nm	Nanometer



LIST OF ABBREVIATIONS

BAT	Brown Adipose Tissues
BMI	Body Mass Index
FSH	Follicle Stimulating Hormone
GnRH	Gonadotropin Release Hormone
HDL	High-density lipoprotein
IGF-1	Insulin-like growth factor-1
LDL	Low-density lipoprotein
LH	Luteinizing Hormone
mRNA	Messenger ribonucleic acid
PCOS	Polycystic ovary syndrome
PRL	Prolactin Hormone
SF-1	Activating steroidogenic factor
TG	Triglycerides
VLDL-C	Very Low-density lipoprotein cholesterol
WAT	White Adipose Tissues
WHO	World Health Organization

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1. INTRODUCTION

Menstrual irregularities and infertility are major issues for half of the world's women, making this branch of medicine one of the most difficult to understand. Infertility is a rare but serious disorder in which a woman is unable to conceive a child. In women or men, it prevents pregnancy, a disease that affects both women and men, and the methods of treating infertility are different due to its different causes. Infertility spreads in all countries of the world, and developing countries are among the areas in which this disease abounds (Abogar *et al.* 2005) According to the World Health Organization-WHO, Because human reproduction is a crucial part of life and the inability to have children has significant consequences, infertility in women is considered a global social problem by the World Health Organization (WHO 2020). This is because infertility in women is defined as an inability to conceive or sustain a pregnancy to the point of giving birth to a live child (Manju *et al.* 2020).

In Which infertility impact 10-25 % (48-180million) of couples of reproductive age all over the world. Infertility is divided into three types, including primary infertility, which affects the woman after a full year of her marriage, Without pregnancy despite the continuation of marital intercourse continuously without taking any contraceptives (Thomas *et al.* 2007). While the second type is a secondary infertility, which affects a woman after she has given birth to one or two children, an ectopic pregnancy, or one pregnancy that ends in miscarriage (Bansal *et al.* 2004) There is also a last type called unexplained infertility, which affects women without showing hormonal or genetic causes, and the real reasons behind this type of infertility are not known (Gillmer *et al.* 1999).

There are many reasons that can cause infertility for women, the most important of which are irregular menstruation, ovarian diseases, and a disorder in the rate of sex hormones, in addition to the disruption of the work of the hypothalamus gland (ACOG 2002, Genetic 2005), as happens Infertility as a result of fungal or bacterial infections or deviation from normal body weight, whether Over weight or underweight, which

weakens the effectiveness of the reproductive system of women due to the change in the level of hormones leading to disruption in the menstrual cycle and impaired ovulation. Additionally, PCOS, which is characterized by the development of cysts in the ovaries, may lead to infertility. A heterogeneous endocrine illness is a common cause of anovulatory and endocrine infertility in women (Jeshica *et al.* 2021).

Serious consequences in women are more likely when they have polycystic ovary syndrome (PCOS). As one in 5-6 females face serious complications related to infertility, irregular menstruation, stress, obesity and variation in the hormonal level in which is the main cause worldwide (Torie *et al.* 2016). A substantial family susceptibility to PCOS and the possibility that one or more genes are responsible for the disorder's diverse clinical and biochemical manifestations support the theory that PCOS is a genetic illness (Jiawei *et al.* 2016). The pituitary gland's sensitivity to GnRH is increased in women with polycystic ovary syndrome, which results in an increase in the concentration of Gonadotropin Releasing Hormone (GnRH) or a change in its secretion patterns. This leads in a rise in luteinizing hormone (LH) levels. In women with PCOS, high levels of triglycerides, low-density lipoprotein (LDL), and low-density lipoprotein HDL were found, as well as abnormalities in the levels of lipids in the blood (Mubarak 2018). Various factors, such as body mass, dietary habits, and ethnicity, influence a patient's fat content (Liu *et al.* 2019). Adipokines, which are released by white adipose tissue and play a key role in both energy storage and the production of a variety of physiologically active proteins, were also shown to be affected by ovarian syndrome in recent years (Ohushi *et al.* 2014, Riondino *et al.* 2014). Polycystic ovarian syndrome has been linked to a number of adipokines, including chemrin, omentin, and adiponin. Chemrin levels in the sera of PCOS patients were reported to be higher (Guvenc 2016). Blood glucose levels are positively connected with the BMI, however omentin-1 is adversely linked to it. A greater BMI is associated with lower levels of this hormone in the bloodstream. Omentin-1 levels decrease with an increase in BMI, which may be connected to an increase in blood glucose levels (Olumide 2014). Adiponin's connection to polycystic ovarian syndrome was also examined in research.

1.1 Aim of the Study

The present study aims to examine the levels of omentin-1 and adipicin in sera of women with PCOS as biochemical indicators for PCOS: Sex hormone levels (Follicle stimulating hormone – FSH, Luteinizing hormone – LH, and Prolactin) should be checked in the sera of women with PCOS. A woman's lipid profile (total cholesterol, triglyceride, low density lipoprotein-LDL, high density lipoprotein-HDL, and very low density lipoprotein-VLDL) should be examined in women with PCOS. Serum omentin-1 and adipicin should be correlated with the parameters under inquiry, and the association should be evaluated.

2. LITERATURE REVIEW

2.1 Ovaries

The ovaries are the female reproductive organs that store ,produce and release eggs into the fallopian tubes , and they are also responsible for the production of sex hormones, the ovary has an oval shape and white color and varies in size (between 0 5.0 cm in length, 1.0 5.0 cm in width and 3 1.0 cm in thickness) during the female reproductive age, The ovary is a mixed secretory gland, and its tissue consists of two areas.

The first region: Is the inner region, which is called the medulla, which is a rich central nucleus. The ovary's main portion, the connective tissue, is encircled by blood arteries. This is Tunica albuginea (Christian 2005).

The second region: It is the outer region and it is called the cortex, as this crust surrounds the pulp from the All sides except for the area of entry of blood vessels and nerves, and contains the ovarian cortex in women Mature on follicles in different stages of growth and development which are scattered everywhere From the stroma (containing connective tissue, interstitial cells and blood vessels (Sherwood 2013).

Each ovary surrounds a duct It is a fallopian tube, (the tube is 15 cm long and 1 mm in diameter) and this canal is lined by a membrane composed of ciliated cells, these cilia move in the upper part of the canal towards the uterus, opening two canals The fallopian tube is one opening on each side in the uterus. (Figure 2.1) shows Female reproductive system.

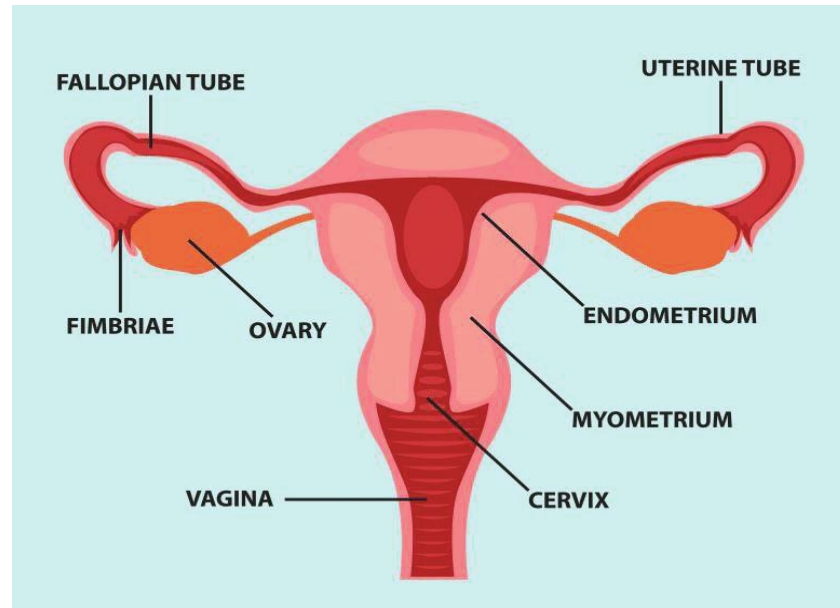


Figure 2.1 Displays the reproductive system of a woman (Seeley 1998)

Ovulation is a process of releasing mature egg from the ovary, through the fallopian tube, to be available to be fertilized and it is basic for pregnancy to occur. It's a complex process and starts at appropriate times by adequate amounts of specific hormones. The disturbance of hormonal regulation for any cause may result in ovulatory dysfunction. Follicles are like egg sacs that hold thousands of embryos, and when ovulation happens one follicle has to mature to maturity. Ovulation necessitates the creation of several hormones, all of which must be present at the appropriate quantities in order for a normal menstrual cycle to occur. Luteinizing hormone (LH) and follicle stimulating hormone (FSH) synthesis is necessary for the development of follicles (FSH). After the follicular phase, the egg is released around halfway through a woman's menstrual cycle.

2.2 PCOS Stands for Polycystic Ovarian Syndrome

Polycystic ovarian syndrome (PCOS) is a common cause of anovulatory and heterozygous infertility in women (Jeshica *et al.* 2021). According to the WHO, PCOS affects around 116 million women worldwide (WHO) (Bharathi *et al.* 2017). Ovarian hyperandrogenism and hyperplasia ovarian follicles are alternate names for the 1935 Leventhal and Stein description of polycystic ovary syndrome (Charnvises 2005) In

1970, the hypothalamic-pituitary axis was shown to be faulty, resulting in an imbalance in the production of luteinizing hormone (LH) and follicle stimulating hormone (FSH) (Rebar *et al.* 1976)

Theca interna, the inner follicle of polycystic follicles, surrounds the ovary in polycystic ovarian syndrome, causing it to have a diameter of 4-7 mm and a number of 6-10 cysts or more (Musmar *et al.* 2013).

Obesity is linked to insulin resistance in polycystic ovary syndrome, which is linked to a rise in androgen levels in the bloodstream that causes excessive hair growth, as well as hirsutism and acne vulgaris. This is an important cause of systemic complications such as type II diabetes mellitus, heart disease, and skin complications (Ibanez 2017, Polka 2017).

2.3 Symptoms of PCOS

The main symptoms of the disease include:

1- A significant number of ovaries with cysts in women with PCOS

During examination with ultrasound, it is usually noticed the presence of immature eggs whose size usually ranges from 0 to 5 millimeters, as the egg sac in normal cases in each menstrual cycle - its size ranges from 11 to 55 millimeters, but the presence of a large number of cysts in the ovary grows at a time. One causes all of them to stop growing, and therefore these eggs do not reach the appropriate size for the process of ovulation to occur, and therefore ovulation does not occur (Al-Hakeim 2010).

2- Hyperinsulinemia, which directly mimics LH and indirectly elevates GnRH, causes excessive androgens (Puttabyatappa and Padmanabhan 2018). Insulin resistance is a major problem in PCOS patients, putting them at an elevated risk for heart disease and diabetes (Rocha *et al.* 2019).

Obese women with PCOS are more likely to suffer from this symptom because of their extra fat. As a result of insulin resistance, the pancreas releases too much insulin to compensate, resulting in hyperinsulinemia, which may lead to diabetes. Insulin and insulin-like growth factor-1 (IGF-1) are important non-ovulatory stimulators of androgen synthesis, which in turn affects the manufacture of sex hormones. Each of these medications enhances the production of estrogen and testosterone in the ovaries and adrenal glands (by raising the CYP type 17A1 activity or activating the steroidogenic factor (SF-1), as well as its target genes) (Rosenfield 2016, Kinyua 2018).

In addition, insulin affects LH pulse amplitude and decreases hepatic sex hormone binding globulin -SHBG synthesis (Leckwyck 2016). Insulin resistance and hyperinsulinemia seem to be the most important factors in the development of PCOS symptoms from a PCOS perspective (Barber 2016). (Figure 2.2).

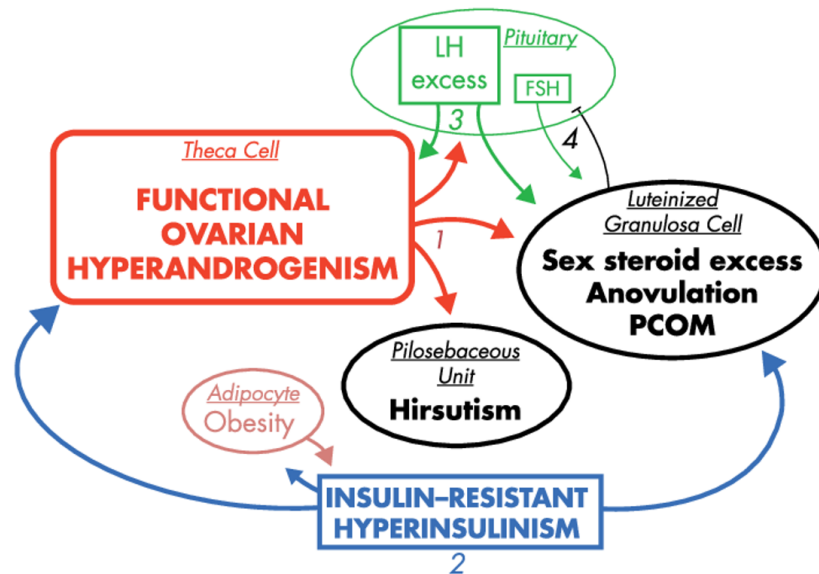


Figure 2.2 Hyperinsulinemia and pathogenesis of PCOS (Rosenfield 2016)

3- Extreme obesity :- It is what causes hormonal changes and the appearance of acne and coarse hair , and it is called functional disorders or phenotypic disorder (Yarak 2005). When obesity is combined with hyperinsulinemia, the lipid and glucose profiles of PCOS patients become even more abnormal. By activating LH, obesity increases

andro- gen production, which results in hyperandro- genism (Glueck and Goldenberg 2019).

4- Acne vulgarism:- In a study conducted on a number of women, it was found that women who have acne are exposed to PCOS compared to women without acne which is highly distributed between women with PCOS at arate 50 % ,whereas the formation of acne is correlated with increased blood androgen levels (Archer *et al.* 2004).

Other complications of PCOS are mainly weight gain, insulin resistance, heart diseases and blood vessels, hypertension, glucose intolerance (Terrie *et al.* 2015), lipid metabolism disorders (Dejager *et al.* 2001), Having a low HDL cholesterol level and a high triglyceride level (Pirwany 2001) .

5- Hyperandrogenism Androgens (male sex hormones) are produced by healthy ovaries in addition to the usual production of female sex hormones (estrogen). There is an increase in the patient's production of androgens when they have PCOS. The patient's level of PCOS is influenced by the amount of androgen being generated (Gleicher *et al.* 2018). Lack of ovulation, acne, hirsutism, and male pattern baldness are all caused by an overabundance of androgen, which is also responsible for the enlarged size of the clitoris and increased muscular mass (Ananya *et al.* 2018). In women with PCOS, hirsutism, acne, and androgenic alopecia are all clinical indicators of hyperandrogenism, which is characterized by elevated levels of testosterone. androgen overabundance may also cause weight gain, menstrual abnormalities, acanthosis nigricans, insulin resistance, and other symptoms (Figure 2.3) (Ashraf 2019)

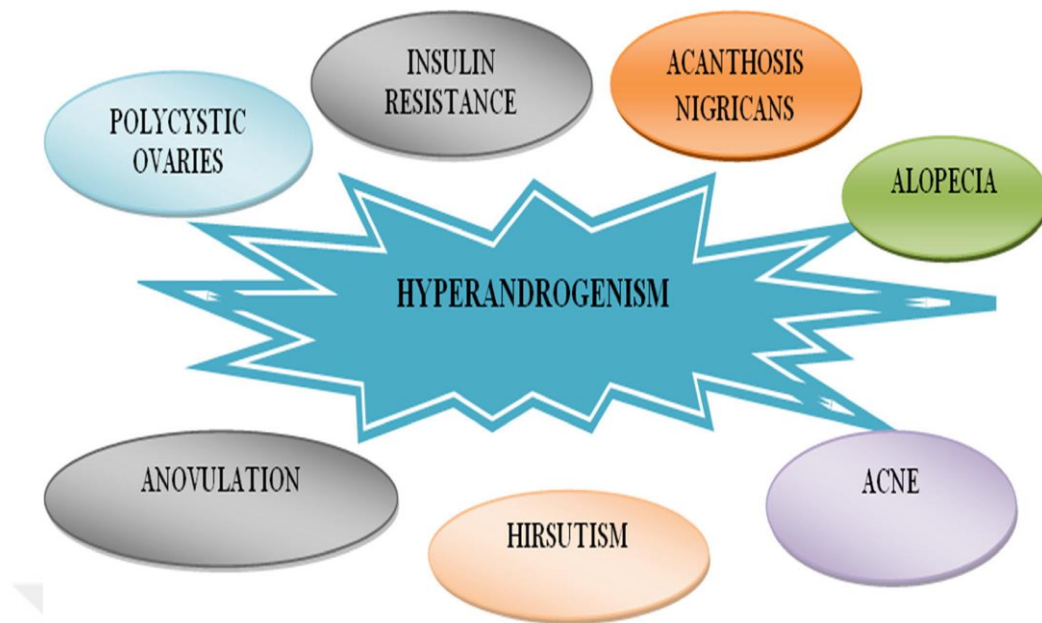


Figure 2.3 Hyperandrogenism in PCOS has important clinical consequences (Ashraf 2019)

2.4 Pathophysiology of PCOS

Despite the fact of the precise pathogenesis of PCOS is still unknown, it is agreed that the disorder arises from a culmination of genetic and environmental factors and that they adversely affect multiple body systems, across the hypothalamus-pituitary-ovarian axis. Adipokines have a significant role in the regulation of nearby metabolic activity (autocrine and paracrine function). Endocrine features are also evident in their ability to control the body's internal activities. Because of this, adipose tissue is a crucial component of the body's hormonal system (Kumari *et al.* 2019). It is possible that obesity and insulin resistance may be linked via the role of adipokines in insulin sensitivity (Figure 2.4) (Arner *et al.* 2018).

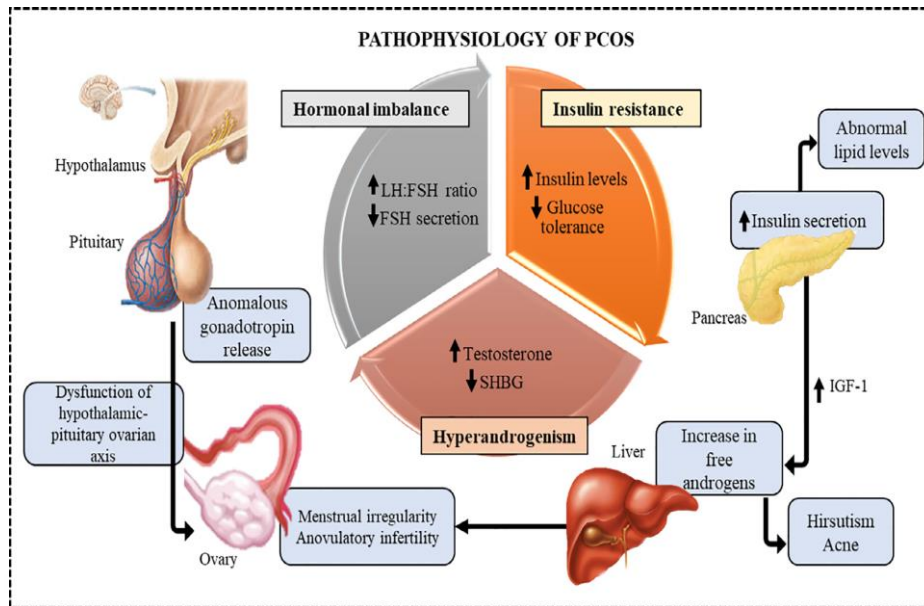


Figure 2.4 Pathophysiology of PCOS (Bulsaraa 2021)

2.5 Hormonal Disturbance in Polycystic Ovary Syndrome

To begin puberty, GnRH is released from the hypothalamic-pituitary-ovarian axis, which has been suppressed since childhood. Hormones released by the pituitary gland in response to GnRH pulse frequencies affect the ovarian tissues, such as granulosa cells and theca cells (Roe and Dokras 2011). DHEA and DHEAS, which are responsible for pubic and axillary hair formation, acne, and an increase in ovarian androgens, are also released by the adrenal gland. Estrogen has a role in breast development, bone growth, and fat deposition throughout puberty (Wen *et al.* 2010). An example of this is seen in Figure 2.5 Ovarian-Pituitary-Hippocampal axis.

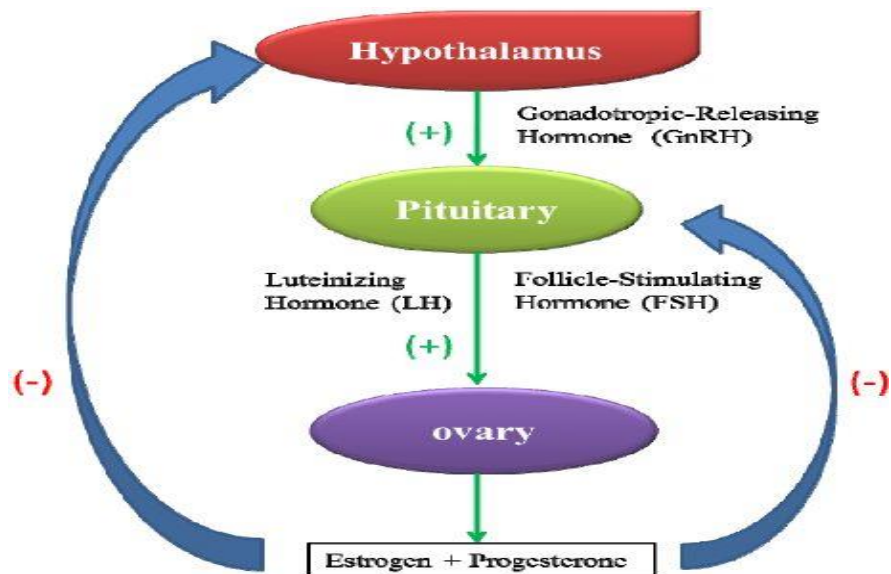


Figure 2.5 Shows Hypothalamic –Pituitary –Ovarian axis (Sun *et al.* 2013).

Core to PCOS is overactive testosterone production, which causes a rise in GnRH pulse frequency and encourages an increase in LH secretion, which, in turn, stimulates the ovarian theca cell to produce more follicle stimulating hormone (FSH). A primary fault in PCOS is androgen excess, despite the fact that the specific cause of the disorder is uncertain (Tarumi *et al.* 2014). PCOS is characterized by protracted periods of oligomenorrhea because of a reduction in FSH production, which results in reduced androgen aromatization to estradiol and decreased follicular growth (Barbieri 2014). This may be seen in (Figure 2.6).

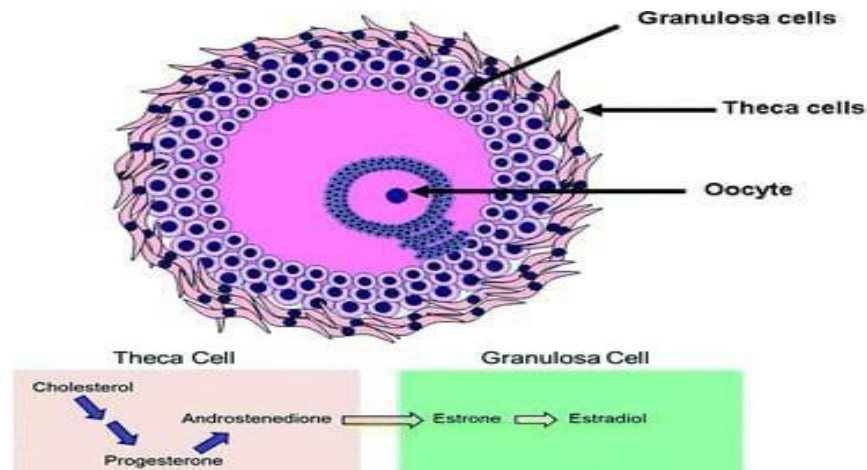


Figure 2.6 All of the cells that make up an embryo's follicle prior to fertilization may be seen. (B) Ovarian follicle synthesizing pathways for progesterone and estradiol (Barbieri 2014).

In comparison to the general teenage population, adolescents with PCOS suffer from greater levels of insulin resistance and hyperinsulinemia (Legro *et al.* 2013). Insulin resistance contributes to PCOS-associated dyslipidemia by increasing levels of free androgens in the bloodstream by promoting androgen production in ovarian theca cells and suppressing SHBG synthesis in the liver. It also decreases lipoprotein lipase activity (Baldauff and Arslanian 2015).

2.6 Polycystic Ovary Syndrome and Male Pattern Baldness

This may be caused by an excess of androgens that interfere with normal androgen production. Excess androgens promote the formation of primordial follicles and the rise in the number of antral follicles during the early gonadotropin stage (Rosenfield and Ehrmann 2016). The pituitary releases gonadotropin hormones in response to the hypothalamus secreting GnRH. Follicle-stimulating hormone (FSH) works on the FSH receptor in ovarian granular cells, which is activated by luteinizing hormone (LH) to create androgens, which are subsequently converted to estrogens by Ovarian theca cell FSH (Ashraf *et al.* 2019). If you have a problem with your neuroendocrine system, you may have an excess of gonadotropin in your body, according to the conventional wisdom. When GnRH levels are elevated, LH production is encouraged over FSH production, leading to an increase in PCOS's LH:FSH ratio (Walters *et al.* 2018, Tsutsumi and Webster 2009). as shown in (Figure 2.7) (Bakir1 *et al.* 2021).

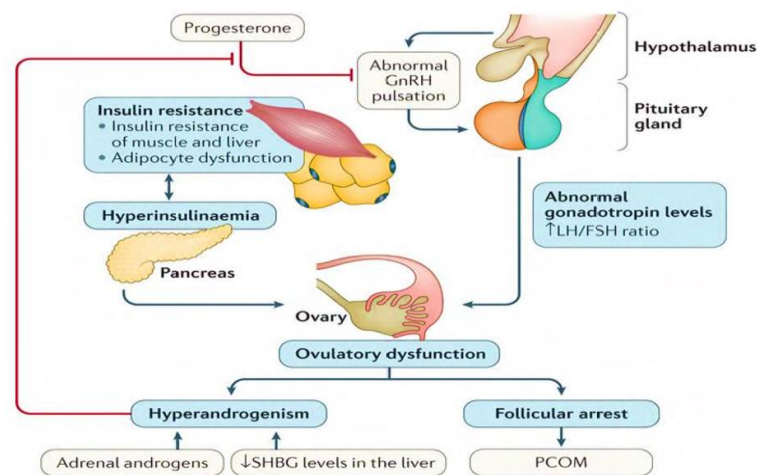


Figure 2.7 Chronic hyperandrogenicity is a hallmark of polycystic ovary syndrome (PCOS) (Bakir1 *et al.* 2021)

2.7 Treatment of PCOS

Treatment of PCOS is directed at the symptoms shown on patients, usually oral contraceptive pills that contain estrogen progesterone is the preferred medical treatment, as it reliably improves both menstrual disorders Menstruation and hyperandrogenism, and exercise is very important to reduce disease. Associated obesity and insulin resistance (Witchel 2015).

2.8 Reproductive Hormones

2.8.1 Follicle stimulating hormone

Folliculotrophs in the anterior lobe of the pituitary gland produce and release a glycoprotein containing two peptide chains and that governs sexual puberty and the reproductive processes of the organism. Because of its biological action and its ability to obstruct FSH receptors, it (Sohrabji 2019). FSH stimulates the development of germ cells in both males and females. FSH is one of the important hormones for the maturation and vitality of sperm and the maturation of the seminiferous tubules through its effect on Sertoli cells, as the hormone sticks to the surface of these cells to stimulate

the maturation of the spermatid tubes as well as stimulate the maturation of the sperm. Without this stimulation, spermatids are difficult to transform into mature spermatozoa (Oduwole, 2018). As for females, it is important for the growth and development of ovarian follicles, in completing the maturation of the oocyte and preparing the ovarian follicles for the effect of the luteinizing hormone that the follicle needs to reach the normal integrated size and the secretion of estrogen, in addition to increasing the secretory activity of the granulosa cell represented by the secretion of progesterone and estrogen in which they participate. Growth and secretion of the lining of the uterus.

2.8.2 Luteinizing hormone

The hormone LH is one of the hormones on which the reproductive process is based in both sexes. Interstitial Cell Stimulating Hormone (ICSH) is the name given to LH in men because it activates the Leydig cells found in the testicular interstitial cells, causing testosterone to be secreted (Sohrabji 2019), and the hormone couples with the receptors for the hormone located on the surface of the membranes of the target tissue to start the process of stimulating the formation of sperm (Jimoh 2012), and it has been proven that the luteinizing hormone plays an important role in controlling the production and secretion of the hormone progesterone by regulating the mRNA transporter DNA. Which encodes important enzymes in the progesterone (Bosch 2021). The effectiveness of the luteinizing hormone. The luteinizing hormone LH in both males and females is mainly responsible for reproduction, in females and during menstruation, the FSH hormone works on follicular growth, especially in the affected cells with a rise in estrogen, LH receptors also indicate the maturity of the follicles and mediated by a gland. The hypothalamus through positive feedback to work on the secretion of the hormone LH during a period of (24-48 hours) (Pagana 2017). The increased flows of LH stimulate the ovulation process as well as work to convert the remaining molecules to the corpus luteum and in return the production of progesterone to prepare the lining of the uterus for implantation of the fetus, LH is necessary in order to maintain the functions of the luteal functions for the first two weeks, and in the event of pregnancy, LH is more likely to preserve the functions of the corpus luteum through the effectiveness of human chorionic gonadotropin-HCG (a hormone very similar to LH) in

the early stages of pregnancy. pregnancy. Theca cells in the ovary also help to produce androgens and hormonal precursors to produce estradiol (Benmachiche 2019). If you are a man, LH works on Leydig cells in the testicles that produce testosterone, an anabolic hormone that enhances endocrine gland activity and sperm production in the testicles. The hypothalamus gland releases Gonadotropin releasing hormone-GnRH, which regulates LH production via the pituitary gland. genitals As an example of how these hormones are regulated, consider (Figure 2.8) (Marshall 2008).

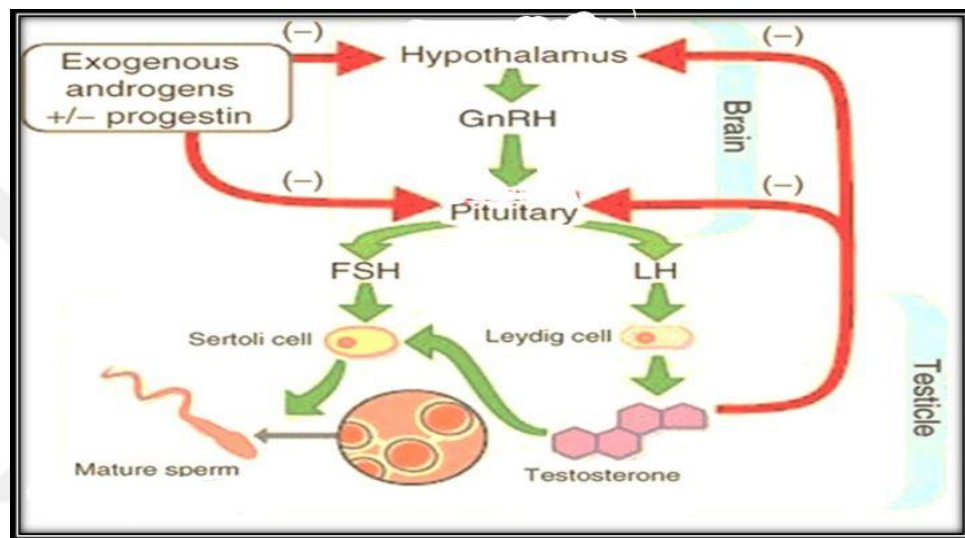


Figure 2.8 Reproductive hormone regulation in male (Sánchez-Garrido 2014)

2.8.3 Prolactin hormone (PRL)

The lute tropic hormone, prolactin, is a protein present in humans that aids in the production of milk in animals, generally females. The pituitary gland releases prolactin in reaction to eating, mating, ovulation, and lactation. It's critical for metabolism, immune system control, and pancreatic development (Bole-Feysot 1998). It is a glycoprotein that is present in different forms, single or two polypeptide chain (23 kd), and it consists of 198 amino acids. The process of secretion of this hormone is controlled by the endocrine glands, through the secretion of prolactin and the inhibitory factor (Shome 1997), and it promotes the formation of neurons in the brain of the fetus and mother (Larsen 2012).

Estrogen is the main regulating hormone for prolactin production, as it promotes the growth of prolactin-producing cells and directly stimulates its production, as well as inhibiting dopamine. The main factor for the increase in the hormone prolactin is the result of a decrease in the natural levels of the sex hormones estrogen in women, and the increase in prolactin is associated with galactorrhea and menopause. And the levels of prolactin in the blood are naturally elevated in women with polycystic ovary syndrome by about (15-20)% (Hoehn 2007).

prolactin is measured when it rises in the blood, which causes inhibition of the ovulation process, while the increased secretion of prolactin in the blood inhibits the activity of GnRH by interacting with the hormone dopamine in the hypothalamic glands by means of inhibit feedback or by direct action on the neurons of the releasing hormone of the glands (Blackwell 1998). The increased secretion of prolactin in the blood interferes with ovulation by inhibiting the secretion of gonadotropins which is necessary for ovarian activation. A problem in the anterior pituitary gland causes a rise in the blood level of prolactin, which results in an increase in prolactin production. Hyperprolactinemia. This results in a decrease in the anterior pituitary gland's ability to produce FSH and LH, which prevents ovulation (Etiology 2013).

2.9 Adipose Tissue

Adipose tissue secretes numerous proteins and bioactive peptides known as adipokines to fulfill metabolic and energy expenditure demands. These adipokines operate locally (through paracrine or autocrine action) or distally (by endocrine action) (Suriano 2020). Adipose tissue is regarded as an endocrine gland (Flier 2004).

Metabolic and cardiovascular functions as well as immunity and reproduction may be affected by several of these substances. Because of the presence of P450 aromatase in adipose tissue, androgens may be converted to estrogens, demonstrating the role of adipose tissue in reproduction. Loose connective tissue surrounds adipocytes, the primary constituents of adipose tissue, which are predominantly made up of lipid-rich

fat cells. People have mostly white adipose tissue while animals have more brown (Ahima 2000).

Adipose tissue is necessary to maintain the physiological nature (Huner 2005), and the main store of energy for the body (Kershaw 2004), and adipose tissue contains a unique type of cells called adipocytes, which are involved in the storage and mobilization of fat and when energy is needed, the triglycerides stored in adipocytes are degraded by lipases to produce free fatty acid and glycerol, which are then transferred to tissues where they are oxidized in mitochondria to produce energy (Huner 2005). Adipose tissue production of free fatty acids has been linked to obesity-related metabolic problems, according to many research (Tung 2014), and it is clear that as a result of the high flexibility that adipocytes possess, they can rapidly expand in size in people with Obesity (Mullar 2014).

2.9.1 Type of adipose tissue

Mammals, including humans, have white adipose tissue, which may account for up to 20% of a person's total body weight in males and 25% in women. The nucleus of white adipose tissue cells is pushed out by a single big drop of fat. White adipose tissue serves to save energy by storing glucose and free fatty acids produced from triglycerides when compressed to a thin edge in the cell as depicted in Figure (Banerjee 2011, Franssens 2017).

Compared to white fat cells, brown fat cells contain many tiny droplets and a considerably greater number of mitochondria. Brown adipose tissue is primarily responsible for generating heat in animals and infants. There are numerous lipid droplets and a significant number of mitochondria in brown fat cells, unlike white fat cells, which have just one droplet. Brown adipose tissue contains more capillaries than white fat because it needs more oxygen (Figure 2.9) (Enerbäck 2021)

Fig. 1

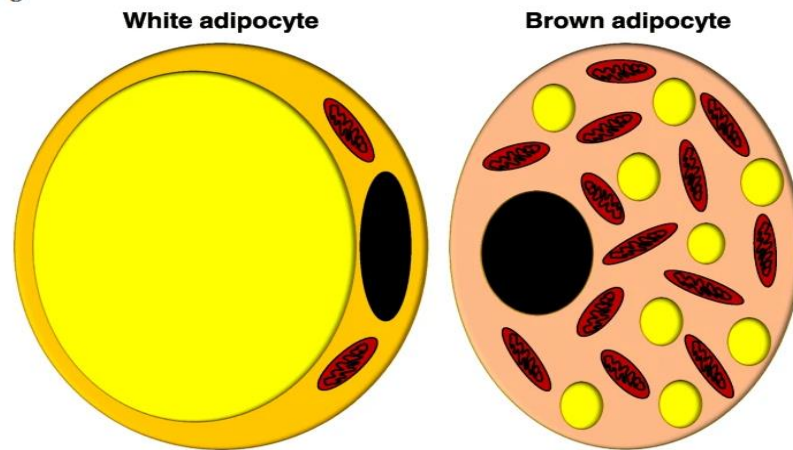


Figure 2.9 Adipose cells white and Brown (Fitzgerald 2018)

2.10 Adipokines

Some of these molecules, such as interleukin-6 and tumor necrosis factor, are solely generated by fat cells; however, other cytokines, such as interleukin-6, are also released at high amounts by tissues, such as adipose tissue (Thondam 2020, Maury 2010). Immune cells migrate into fat, and adipokines regulate adipogenesis, adipokine metabolism, and adipokine activity all because of adipokines. It is important to note that adipokines have a substantial impact on target organs such as the brain and pancreas. Adipose tissue function is reflected in the pattern of adipokine secretion, and this pattern is critical. Obesity-related metabolic and cardiovascular comorbidities may be identified for each person. There have been several new adipokines found since the discovery of leptin in 1994, when a new era in the research of white adiposity was heralded. These novel adipokines include adiponectin, chemerin, visfatin and others (apelin, and others). And adipokines are important for many bodily activities, including energy control, nutrition and glucose metabolism as well as thermogenesis (Mattu 2013, Trujillo 2006).

Appetite management, energy balance, insulin secretion and sensitivity, blood pressure regulation, endothelial function and other processes are all controlled by adipokines, which play a key role in the regulation of local metabolic processes (autocrine and

paracrine function). Endocrine features are also evident in their ability to control the body's internal activities. Because of this, adipose tissue is a crucial component of the body's hormonal system (Kumari *et al.* 2019). It is possible that obesity and insulin resistance may be linked via the role of adipokines in insulin sensitivity (Arner *et al.* 2018). here, for example (Figure 2.10) (Bluher 2014).

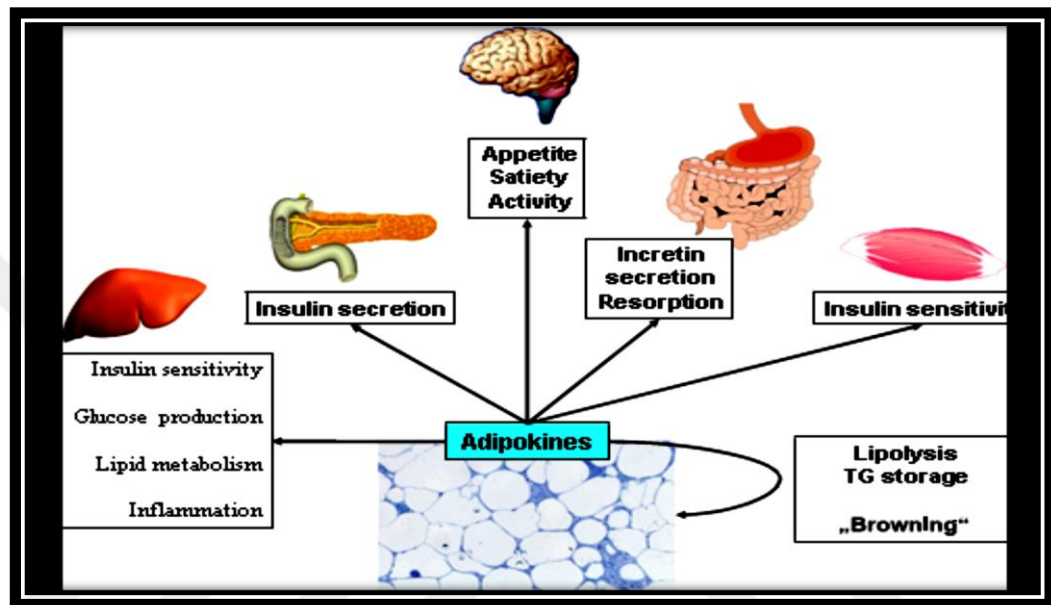


Figure 2.10 Regulation of Physiology of Adipokines (Bluher 2014)

2.10.1 Omentin-1

Accumulated visceral fat, or adipose tissue, is a glycoprotein that has 295 amino acids and is linked to oligosaccharides (Anh 2014). Among its many names are intelectin (Wiltshire), Intelectin-1, and endothelial lectin, all of which were coined by Yang back in 2003. (Lee 2001) Omentin-1 and Omentin-2 are the two types of the Intestinal Lactoferrin Receptor (Suzuki 2001, Wiltshire 2001). In the chromosomal region Iq22-Iq23, there are two kinds of Omentin-1 genes that create omentin-1 and omentin-2. While the plasma and visceral adipose tissues are the most abundant sources of omentin-1, the heart, lungs, and placenta have lower quantities of the omentin-1 form than the plasma and adipose tissues (Anh 2014).

As it is known to be expressed or generated in various physiological and pathological circumstances such as obesity, insulin resistance, and inflammatory diseases, physiological quantities of omentin-1 in humans vary from 100-800 ng/mL (Yang 2006).

Omentin-1 adipocaine is associated with obesity and plays a key role in controlling blood pressure, heart disease, vascular disease, and metabolic disorders (Kuperman 2005). Omentin works to increase insulin signaling through interactions or phosphorylation interactions through activation of a protein protein kinase activates glucose transport to fat cells and muscle tissue. When weight loss occurs as a result of insulin sensitivity, the level of omentin rises as well as decreases in the pre-diabetes steps. Obese people and women with polycystic ovarian syndrome had lower levels of omentin. Compared to healthy women, those with PCOS (Tan 2008, Moreno 2010, Tang *et al.* 2017). It has been reported that polycystic ovarian syndrome (PCOS) may be linked to an increased level of omentin-1 in women, which may play a crucial role in its development.

2.10.2 Function of omentin-1

There is recent evidence that suggests that the innate immune system's ability to recognize pathogens and bacteria is enhanced by the presence of the Ca²⁺-dependent lectin omentin/intelectin, which has been shown to bind to galacto furanosyl residues (found on bacteria and pathogens). Inflammatory conditions and obesity have been demonstrated to influence omentin gene expression (Atheer 2009).

2.10.3 Clinical relevance

Omentin mRNA was shown to be variably expressed in the omental adipose tissue of persons with Crohn's disease, which implies that omentin may be a potential candidate factor for chronic inflammatory disorders in humans, as demonstrated by this research (Arash 2012). Obesity, poor glucose control, polycystic ovarian syndrome, type 1

diabetes, and type 2 diabetes have been reported to lower circulating omentin-1 concentrations (Yan 2011). Endothelial dysfunction and cardiovascular disease (Tan 2010) and hypertension (Elik 2021) have both been linked to low levels of omentin-1 in the blood. For individuals with heart failure, it is not known if serum omentin-1 levels are correlated with clinical outcomes (Narumi 2014). With the kidney disease and renal failure, Omentin levels might fluctuate. (Alçelik 2012). Omentin-1 may improve insulin sensitivity and glucose metabolism by acting as an endocrine factor on muscles, liver, and the omental adipose depot (Yan 2011). Study by (Yilmaz *et al.* 2011) found that individuals with fatty liver disease had increased levels of omentin-1 in their blood, which is an independent predictor of hepatocyte ballooning (Yilmaz *et al.* 2011).

2.10.4 Adipsin

Adipsin, also known as complement factor D, is one of the first-described adipokines (Cook 1995) Adipsin is a serine protease discovered in 3T3 adipocytes with a 28KD protein (Zhou 2018) To keep the body's fat stores in balance, Adipsin stimulates insulin release when glucose levels rise. The alternative complement pathway is controlled by it, which increases insulin release from the pancreas and catalyzes the creation of C3a (an active version of component 3, C3) (Figure 2.11) (Lo 2014, Gómez-Banoy 2019).

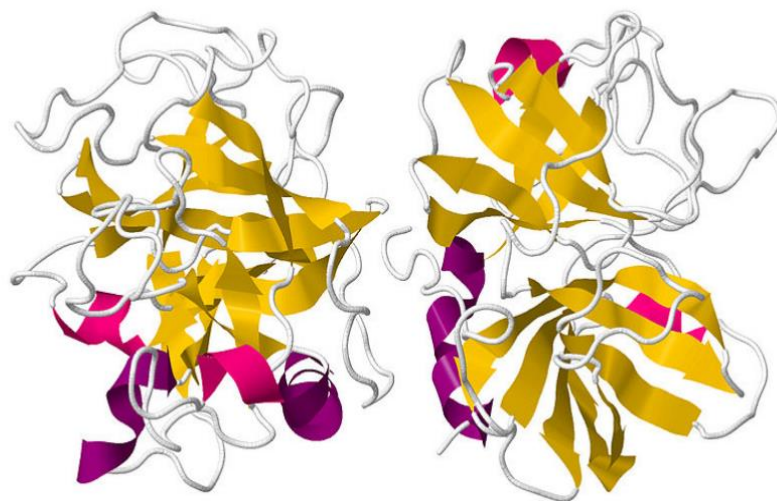


Figure 2.11 Structure of Adipsin (Narayana 1994)

As the C5–C9 membrane attack complex develops, adipsin produces signaling molecules such as anaphylatoxins, complement 3a (C3a), and C5a. According to (Lo 2014). Adipsin cleaves complement factor B in the presence of the enzyme C3 convertase, leading to the formation of numerous complement fragments such as C3a, C3b, and C5a, as well as the enzyme C5a. This is followed by the hydrolysis cascade in (Lo 2014). C3 deficiency in the family has been connected to obesity and metabolic disease; high levels of C3a have been associated to diabetes mellitus" (DM). "Song 2016" is a song written by All of Adipsin's insulin-stimulating properties have been summarized in this table (Figure 2.12).

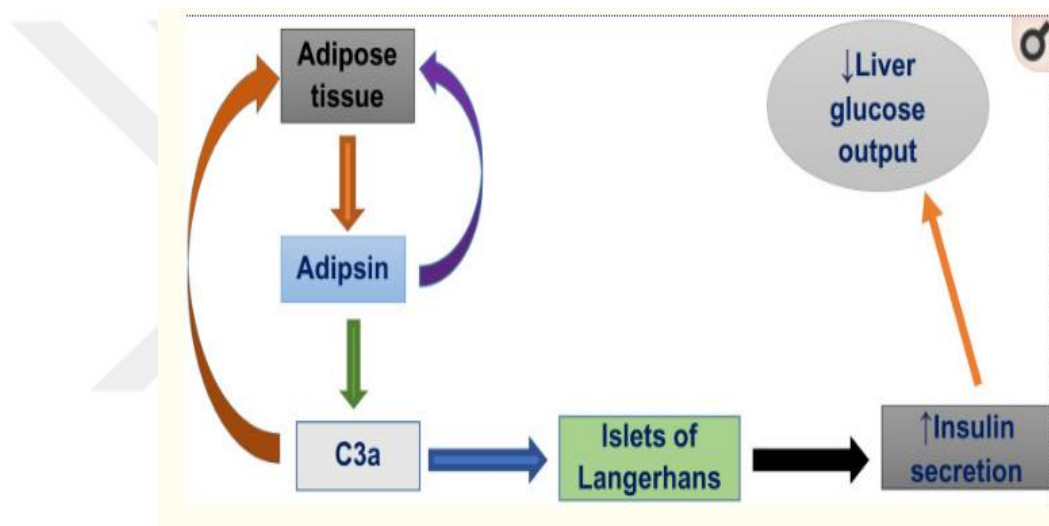


Figure 2.12 Insulin secretion and adipsin hormone interaction (Gebrehiwot 2020)

An increase in lipid accumulation and adipocyte differentiation has been linked to adipsin, which may explain the link between adipsin and metabolic diseases (Song *et al.* 2016).

Adipsin is a type of adipokine that participates in a variety of biological processes, including blood clotting and supplement activation, fertility, the immune system, tissue repair, blood pressure and ferritin degeneration, as well as cell proliferation and programmed cell death (Faina 2007, Kong 2009). Adipsin plays a prominent role in many diseases, as Legakis (Legakis 2005) indicated a decrease in the level of adipsin

in patients with type 2 diabetes, especially those who suffer from failure of beta cells - pancreatic failure. They have low levels of adiponin (Lo 2014)

2.11 Lipids Profile

Lipids are non-polar organic substances that are insoluble in water but are soluble in non-polar organic solvents (Akoh 2017). Lipids are one of the important nutritional components as it is a source of energy that provides the body with 9 kilocalories / g, as well as fatty acids such as linoleic acid, which is important in the growth of children's body through food, as well as fats are important to maintain the health of human skin and the production of some similar compounds, With hormones such as prostaglandins that are important in regulating some activities in the body (López Ibáñez 2016). Cell membranes are made of various fats in different proportions and quantities (Harayama 2018).

Lipids are made in the body by the liver and adipose tissue and also obtained through food and then transferred to different tissues and organs for use or storage. Plasma contains the main Lipids, which are ester cholesterol 36%, phospholipids 30%, triglycerides 16%, free cholesterol 14%, and fatty acids 4%.

2.11.1 Cholesterol

It spreads widely in all membranes, in the brain, spinal cord, nerves, adrenal cortex, ovaries, testicles, and in the blood in free form or in the form of esterified cholesterol, and it is accompanied in all the sources mentioned by the saturated compound called dihydrocholesterol or cholesterol $C_{27}H_{47}OH$, which makes cholesterol inside the cells of the body and from cholesterol makes bile acids. Bile acids, steroid hormones, and vitamin D3 (Figure 2.13).

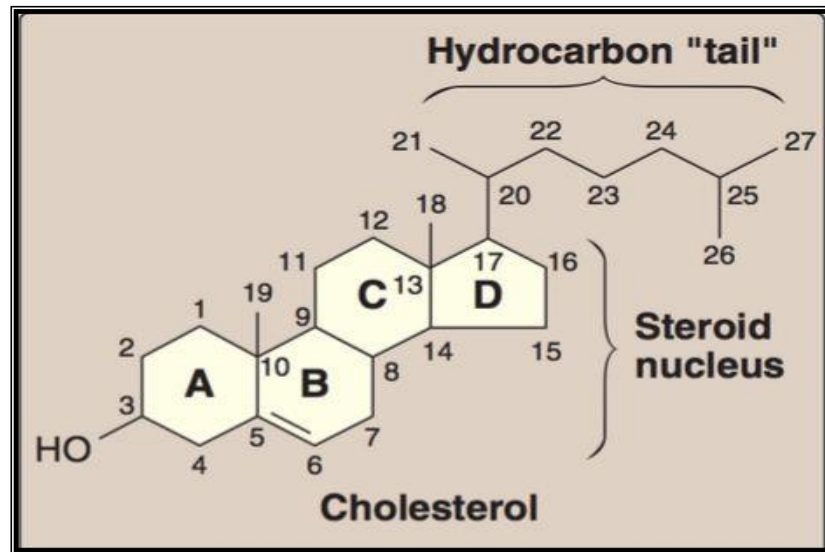


Figure 2.13 Structure of Cholesterol (Nielsen 2013)

Cholesterol is made in the body from two main sources, it may be manufactured internally in the liver and represents approximately (75- 80)% of the total cholesterol level, or it can be externally sourced, that is, it can be obtained from eating fatty foods and diets rich in cholesterol such as egg yolks, whole milk and animal fats. (Figure 2.14) shows the two sources of cholesterol.

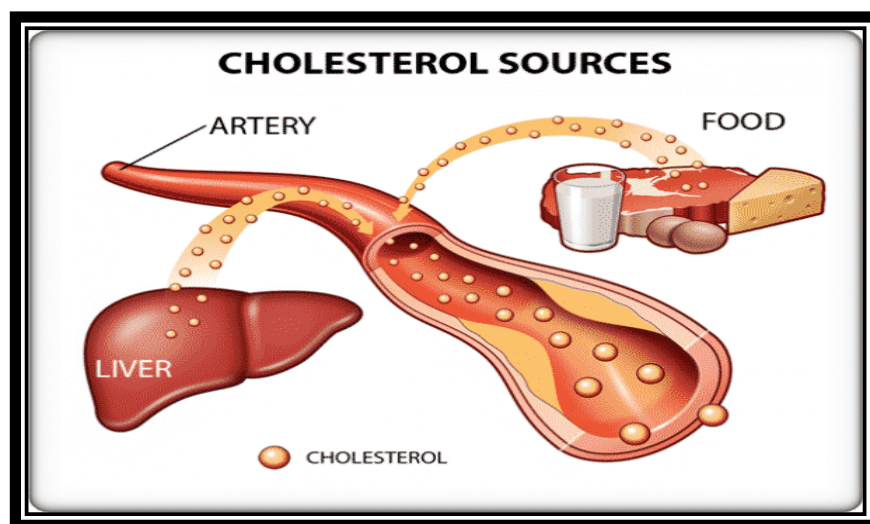


Figure 2.14 Cholesterol Source (Rader 2005)

2.11.2 Triglycerides- TG

It is a class of neutral fats that consists of esters of fatty acids with glycerol, and triacylglycerol is formed from glycerol as a basic unit in which hydroxyl groups are linked with three molecules of fatty acids. Triglycerides is the main compound in the daily food fat, and it is one of the simplest fats and the most abundant, and it is the form that is stored under the skin and inside the fatty tissues (Rice 2001).

The levels of triglycerides vary with age and sex, as it is high in people who suffer from obesity, heart disease and diabetes (Martin 2012 and Surendran 2012). High levels of triglycerides, and more especially lipoproteins rich in triglycerides, are commonly related with low levels of HDL-C and elevated levels of tiny, dense LDL-C particles, which induce atherosclerosis in a high concentration. They have been linked to an elevated risk of heart disease on their own (Reiner 2017, Arca 2018).

2.12 Lipoproteins

Lipoproteins are biochemical complexes of spherical shape and consist mainly of triglycerides and esterified cholesterol to form a nonpolar (hydrophobic) core surrounded by a single surface layer of molecules of phosphorylated degrees and free cholesterol (Al-Raaie 2016). Lipoproteins contain 59% of proteins and 41% of lipids and are composed of lipids of 18% of cholesterol and 23% of phospholipids (Figure 2.15) shows the globular structure of lipoproteins.

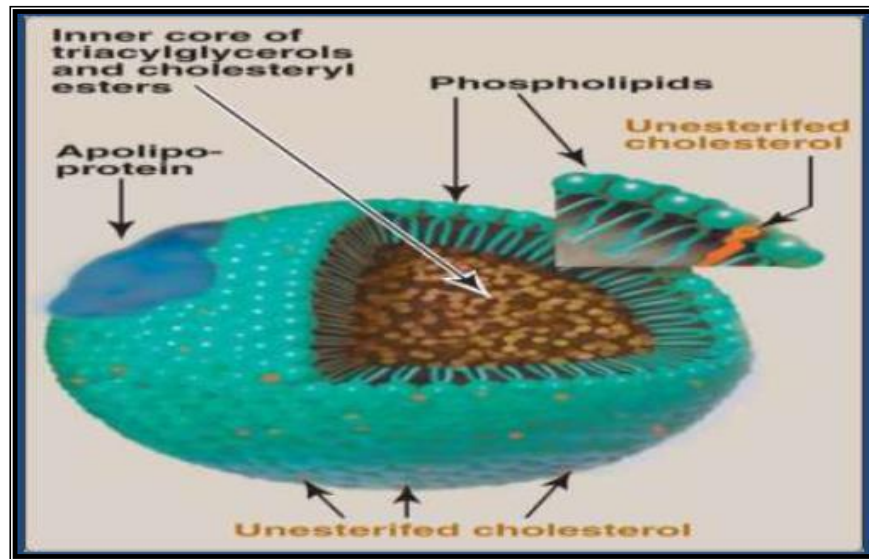


Figure 2.15 Structure of Lipoprotein (Ayyappa 2017)

2.12.1 High-density lipoproteins cholesterol (HDL-C)

This class of lipoproteins is the smallest among the classes of lipoproteins with a diameter of about 10-8 nanometers (Wasan 2002) and the percentage of protein is higher than that of fat (Khalaf *et al.* 2019). High density proteins play a major role in removing excess cholesterol. It is removed from cells and returned to the liver, where it is metabolized into bile acids and salts that are eventually eliminated through the intestines. HDL-C and LDL-C together are key factors in maintaining the balance of cholesterol in the body, due to the high correlation between cholesterol levels in the blood with HDL-C, which leads to a lower incidence of atherosclerosis in people. (Bandeali 2012). Exercise has shown to increase the level of HDL-C, which is anti-atherosclerotic, anti-inflammatory and antioxidant molecule, and has the ability to stimulate the release of nitric oxide (Franklin 2020). Also, through the effect of oxidation, high-density lipoprotein has a role in preventing heart disease and also reduces and limits the incidence of angina pectoris (Wang 2018). Shows structure of HDL-C (Figure 2.16).

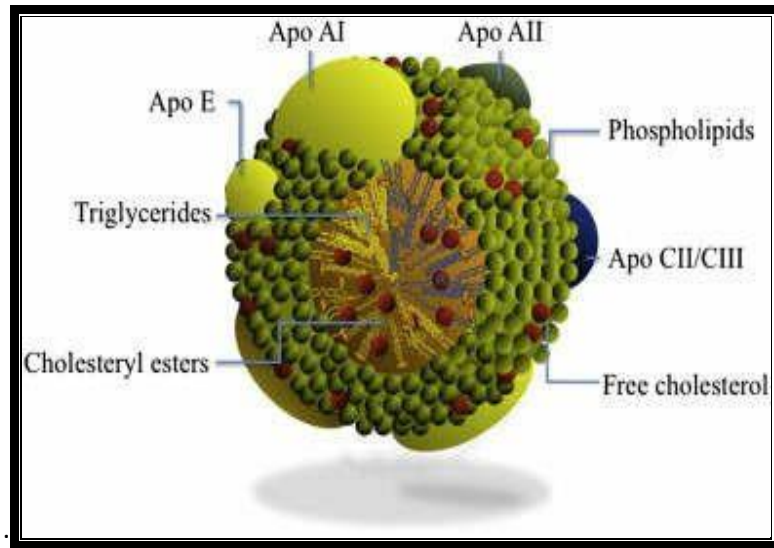


Figure 2.16 Structuer of HDL-C (Chunta.,2021)

2.12.2 Low-densit lipoproteins (LDL-C)

A small molecule with a diameter ranging between (18-25) nm (Wasan 2002) and it contains a high percentage of fat and a low percentage of protein, and cholesterol constitutes the largest part of the fat in it and it transports cholesterol And triglycerides from the liver to other tissues (Rice 2002), and it is the main carrier of cholesterol. Previous research has indicated that increased levels of LDL-C lead to atherosclerosis of the coronary arteries of the heart, and as a result of the oxidation of LDL-C by free radicals it will accumulate in the endothelium blood vessels, thus obstructing the blood flow and causing angina pectoris (Guyton 2016). Patients exposed to persistently high levels of LDL-C are at risk of developing coronary heart and vascular diseases (Penson 2020). Also, continuous exposure to high levels of LDL-C increases the risks of coronary heart disease and cardiovascular disease in the long term, as deaths have been reported in young people with an increase in the level of LDL-C more than 100 mg/cm^3 (Figure 2.17) (Abdullah 2018, Zhang 2019).

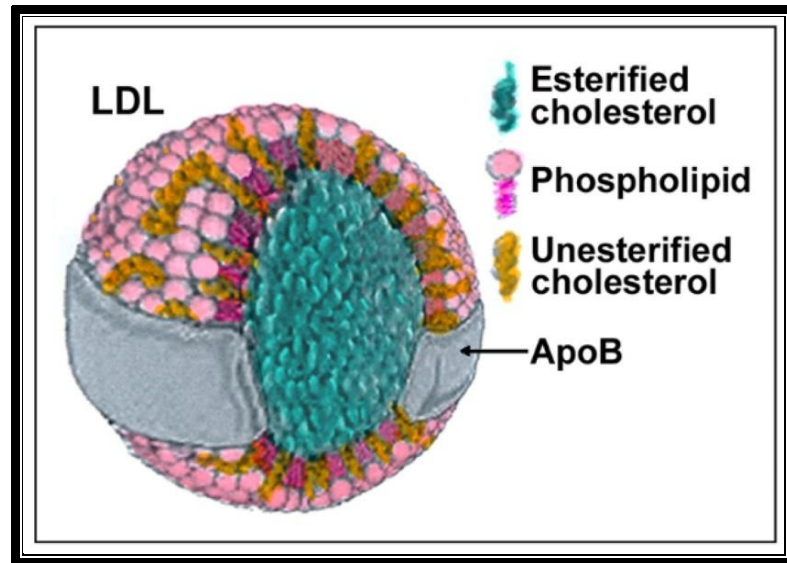


Figure 2.17 Structure of LDL-C (Jean 2021)

2.12.3 Very low density lipoproteins cholesterol-VLDL

Mainly made in the liver, and it is also made in the intestine from triglycerides and cholesterol, which is responsible for transporting phospholipids, triglycerides, and cholesterol esters of endogenous origin. Lipoproteins. The molecules of this protein are carriers of triglycerides in the circulatory system and also transport fats made in the liver to other body tissues (Lin 2016).

3. MATERIALS AND METHODS

3.1 Instruments and Materials

Table 3.1 lists the study's instruments.

Table 3.1 Instruments and their suppliers.

Instruments	Suppliers
Centrifuge	Termaks, Korea
ELISA microplate washer	BioTek ELx800 bioelisa washer / U.S.A
Incubator	LabTech, Korea
Oven	Tvmaks, Norway
Shaking water bath	Gemmyco, Taiwan
Spectrophotometer	Genesys, American

3.2 The Kits

We can see the kits used in the study

Table 3.2 Kits and their suppliers

Kits	
Omentin-1	Melsin –Chain
Adipisin	Melsin –Chain
Cholesterol kit	Randox., United Kingdom
FSH kit	Monobind Inc., USA
HDL-cholesterol kit	Randox., United Kingdom
LH kit	Monobind Inc., USA
Prolactin kit	Monobind Inc., USA
Triglycerides kit	Randox., United Kingdom

3.3 Sample Collection

One hundred of blood serum were collected from women with polycystic syndrome and healthy women from external laboratory auditors in Samarra district for the period from 1/3/2021 - 1/7/2021 after taking information from them, and their ages were (20-40) years. Into two groups samples were divided the first group is a patients with PCOS,

which includes 60 samples, and the second group is the healthy group, which also includes 40 samples considered as a control sample.

3.4 Preparation of blood samples

Blood samples for the current study were collected by withdrawing (5 mL) of venous blood using a medical syringe for one use only, and placing the blood in gel tubes with a tight-fitting cap, These tubes were placed at room temperature to coagulate and then centrifuge for 10 minutes at a 3000 rpm, the serum was withdrawn by a micropipette, then divided into several parts, distributed in eppendrof tube and kept at (-20°C) until biochemical parameters were measured.

3.5 Methods

3.5.1 Determination of serum omentin-1 concentration

The Omentin-1 concentration was measured by following the protocols included in the pre-packaged analytical kit and the instructions provided by the manufacturer.

Principle: This package includes an antibody-coated microtiter plate. Wells were incubated with HRP-conjugated d antibody and reference samples. Add Chromogen Solutions A and B after incubation and washing to remove the uncombined enzyme. The liquid's hue will shift to blue. The color changes to yellow when exposed to acid. A wavelength of 450 nanometers is used to measure the color change. After comparing the O.D. values to the standard curve, Omentin 1 concentrations in the samples may be determined.

Procedure:

- It is advised that all reagents be prepared prior to beginning the test process. The Microelisa Stripplate was loaded with two samples at a time.
- Standardized on: Preparation of standards, evaluation of sample wells
- Standard wells were augmented with 50 L standards.
- Added an example: Added a test for everything and everything possible. After that, a 40-milliliter sample diluent was put to the testing sample well; the blank well had no effect.
- An adhesive strip was placed over each well, and the HRP conjugated reagent was incubated for 60 minutes at 37°C.
- At least four times each, I aspirated the well and cleaned it. Each well should have 400 mL of Wash Solution added to it using a manifold dispenser or an autowasher. Fluids must be completely eliminated at each stage of the process in order to get the best results. Decant or aspirate any remaining Wash Solution after the last wash. Fresh paper towels were used to blot the dish once it had been turned over.
- Chromogen solution A and B, each weighing 50 liters, were distributed to the participants. A 10 L volume was incubated for 15 minutes. A diluent was added to the sample well since there was no blank well in the 37°C experiment. It's dark in here.
- Approximately 50 L of stop solution was poured into each well. Color had changed from blue to yellow in the wells. If the color in the wells is green or the change in color is not constant, softly touch the plate.
- Measure the optical density (O.D.) at 450 nm within 15 minutes using a microtiter plate reader.

Calculations: The calculation was done according to the following standard curve Figure 3.1.

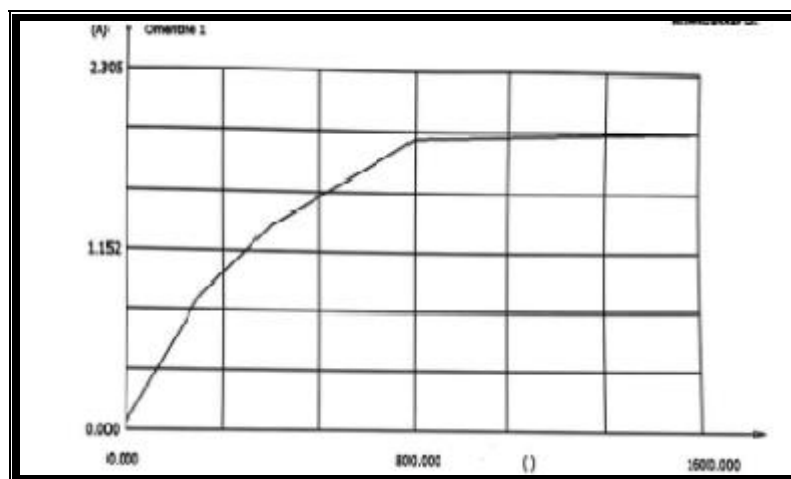


Figure 3.1 Spiraling Curve (Omentin-1)

3.5.2 Adipsin levels in the blood are measured

The adipsin concentration was calculated by following the procedures included in the pre-packaged analytical kit and by following the directions provided by the manufacturer.

Procedure:

- It is advised that all reagents be prepared prior to beginning the test process. The Microelisa Stripplate was loaded with two samples at a time.
- Standardized on: Preparation of standards, evaluation of sample wells
- Standard wells were refilled with 50 L of standards.
- Added an example: Everything has been tested. After that, a 40-milliliter sample diluent was put to the testing sample well; the blank well had no effect.
- HRP conjugated reagent was added to each well, covered with an adhesive strip, and incubated at 37°C for 60 minutes.
- A cleaned each well four more times after aspirating it, for a total of five washes. Add 400 mL of Wash Solution to each well via a squirt bottle, manifold dispenser, or autowasher. Good performance requires the complete elimination of fluids at each phase. Any residual Washed Solution was removed by aspiration or decanting

after the final wash. Clean paper towels should be used to wipe the dish once it has been inverted.

- Chromogen solution A and B, each weighing 50 liters, were distributed to the participants. incubated for 15 minutes at 37°C in a 10 liter vessel. Add 40 mL of sample diluent to the testing sample well, and then add the blank well at 37 degrees Celsius. Light is kept out.
- Each well received 50 L of Stop Solution as a precautionary measure. A change in color from blue to yellow will occur in the wells. If the wells are green or the color shift is not uniform, gently tap the plate to ensure thorough mixing.
- Measure the optical density (O.D.) at 450 nm within 15 minutes using a microtiter plate reader.

Calculations: The adipsin concentration was determined using the standard curve shown below Figure 3.2.

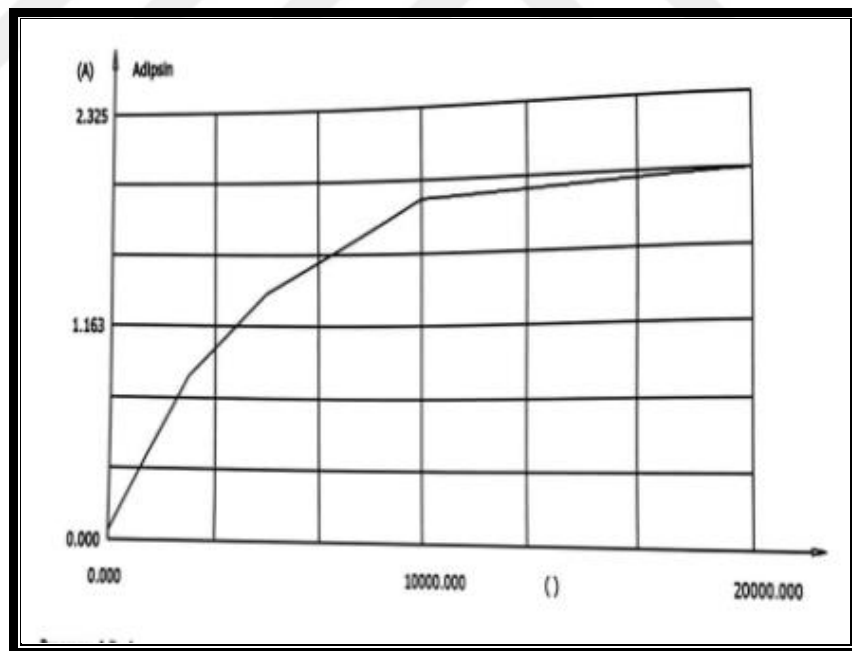


Figure 3.2 Standard Curve of (Adipsin)

3.6 Detremantion of Sex Hormones

3.6.1 Determination of serum LH concentration

An assay kit was used in accordance with the manufacturer's instructions to measure the LH hormone levels (Lenton 1982).

The solutions used:

- Cans of LH-calibrators (1) cm^3 per pack and at concentrations (100,75,50,25,10,0) milli units/ cm^3 .
- One box of LH-(3) cm^3 enzyme reagent.
- A streptavidin-coated calibration plate consisting of (96) small holes.
- One can of washing solution contains (20) cm^3 .
- One tube of substrate solution A contains (7) cm^3 .
- One tube of substrate solution B contains (7) cm^3 .
- One can of suspension solution contains (8) cm^3 .

Preparation of solutions: Add the contents of the can of (20) cm^3 to (980) cm^3 distilled water to dilute the washing solution, which was then stored at room temperature (20-27) C and utilized within (60) days after being diluted.

The designated canister A's contents were transferred to the contents of canister B, mixed well, and diluted at 8-2°C before being utilized within 24 hours (60 days).

Procedure:

- 50 microliters of each sample and control were put to the test plate's wells.
- All pits previously containing samples or controls received 100 microliters of enzyme reagent (LH - Enzyme Reagent).

- After 20-30 seconds of moving the micro plate, I had established a connection between the samples and enzyme using a special cover.
- At laboratory temperature, it was incubated for 60 minutes.
- Turn the pit contents over and dry them on moisture-absorbent paper to get rid of them.
- Repeat the cleaning procedure three times for the plate and all holes, using 300 microliters of the washing solution each time.
- All of the wells were filled with 100 microliters of working solution, then the color gradation of the samples and the control was observed.
- At room temperature, I incubated the dish for 15 minutes.
- It took around 15-20 seconds of gentle plate movement and 50 L reaction stop solution to thoroughly mix all pits.
- A wavelength of (450 nm) was used to analyze the samples after the stopping solution was added.

Calculation: The concentration of LH was carried out by use the following standard curve Figure 3.3.

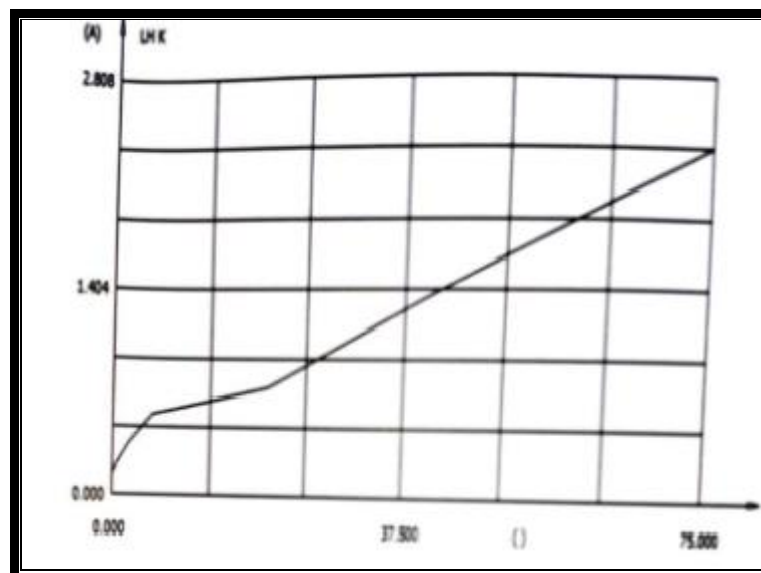


Figure 3.3 The typical LH curve

3.6.2 Detremantion of serum FSH concentration

In order to determine the concentration of FSH, the manufacturer's instructions and the protocols included in its ready-to-use assay kit were followed (Vitt 1998).

The solutions used:

- Packs of 1 FSH-Calibrators mL per pack at levels (100, 50, 25, 10, 5, 60) mL IU/cm³.
- One box of the enzyme reagent (13) FSH cm³.
- Calibration plate coated with streptavidin consisting of (96) small holes.
- One can of washing solution contains (20 cm³).
- One tube of base material A contains (7) cm³.
- One tube of substrate B contains 7) cm³.
- One package of suspension solution contains (8 cm³).

Prepared the solutions:

- This solution was prepared by diluting the can of watering solution with (980) mL of distilled water and keeping it at room temperature (20-27) C for (60) days before use.
- The contents of box A were added to box B, mixed well, and kept at (2-8) C for (60) days as a working substrate solution.

Procedure:

- 50 microliters of each sample and control were put to the test plate's wells.
- After the samples and controls had been introduced, each pit was given 100 microliters of FSH - Enzyme Reagent.
- After 20-30 seconds of moving the micro plate, I had established a connection between the samples and enzyme using a special cover.

- At laboratory temperature, it was incubated for 60 minutes.
- Turn the pit contents over and dry them on moisture-absorbent paper to get rid of them.
- Repeated the washing procedure three times, washing the plate with 300 microliters of washing solution in each hole.
- For this test, I added 100 microliters of the working solution to the working substrate in each well and observed the color gradation in the samples and controls.
- At room temperature, I incubated the dish for 15 minutes.
- After adding 50 L of reaction stop solution to each pit, the plate was gently moved for 15-20 seconds to finish the mixing procedure.
- After adding the stopping solution, the samples were read at (450 nm).

Calculation: The concentration of FSH was carried out by use the following standard curve Figure 3.4.

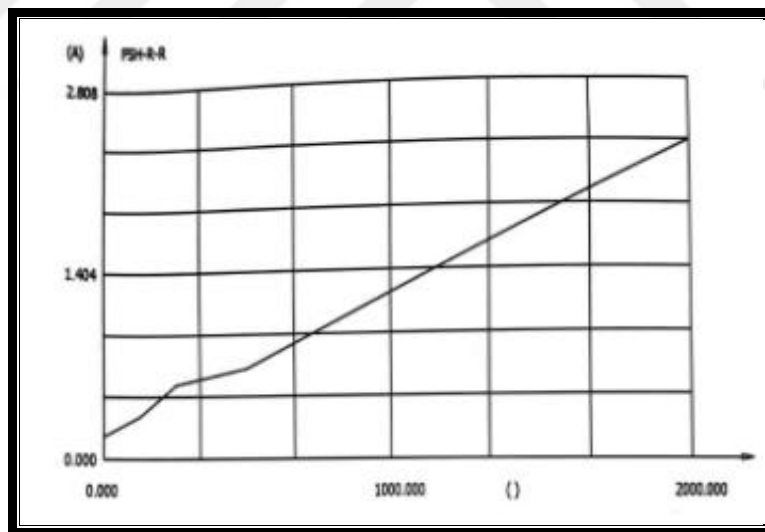


Figure 3.4 Spiraling Curve (FSH)

3.6.3 Measurement of Prolactin hormone levels

Principle: The concentration of prolactin was measured by following the ready-made steps referred to in the hormone device (minividas) exclusively in several specialized

analyzes, and they differ from one device to another and according to the manufacturer (Sapin 2001).

Procedure:

- The test strip required for the hormone in question was extracted from the refrigerator and left for at least 30 min, to take room temperature.
- The test strip and the solid phase container were used as a absorbent device (SPR) to examine each sample and it must be ensured that the storage bag is tightly closed.
- The control and calibration solutions are extracted from the test kit.
- Prolactin is selected from the screen of the device, the symbol for calibration solutions is defined by S1 and the control solutions by (C1).
- One of the important points when measuring the sample is that its serum does not contain red blood cells (R.B.Cs), and to ensure that this happens, we place the sample to be examined in a centrifuge.
- Insert the solid phase container and the test strip into the device, after making sure that the color of the mark with the test symbol on the solid phase container and the test strip are similar to each other.
- 1 μ 200 of the sample was added to the first hole in the tape and in all tests.
- The device instructed to start work, then perform the examination steps automatically by the device and within about 40 min, the result is obtained on special papers printed according to the program in the device's memory.
- When the scan is completed, both SPR and Strip are removed from the device.

Calculation: After the absorbance of the sample, standard solutions and control solutions are measured, the results are calculated automatically by the device and printed on special papers Figure 3.5.

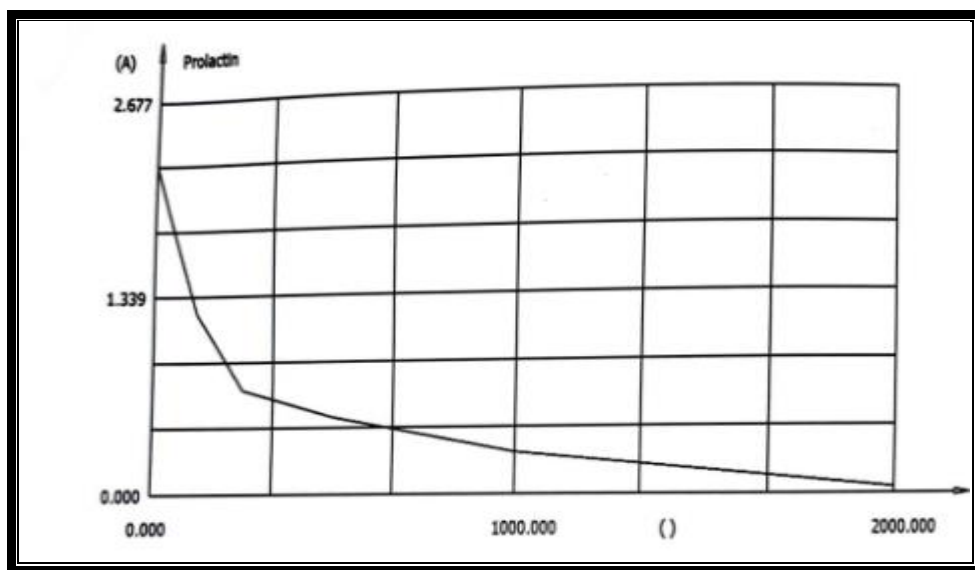


Figure 3.5 Standard Curve of (Prolactin)

3.7 Detremination of Serum Lipid Profile

3.7.1 Measurement of cholesterol levels in the blood

A kit was used to determine the level of cholesterol in the blood serum (kit) (Fossati 1982).

Principle: The cholesterol oxidase enzyme is used by the oxidation of cholesterol in the presence of the enzyme Cholesterol oxidase and in the presence of a hydrogen donor substance.

Enzyme Peroxidase spectroscopically measures the color of the colored product that results from oxidation of free fatty acids and the esterification of the cholesterol esterified by cholesterol esterase.

Reagents used:

Enzymatic solution: It consists of 0.30 mmol/L 4-Aminoantipyrine and 6 mmol/L of phenol and peroxidase enzyme at a concentration greater than or equal to 0.5 units/cm³

and 0.15 units/cm³ of Cholesterol esterase enzyme and 0.1 units/cm³ of Cholesterol esterase enzyme. Cholesterol oxidase enzyme and finally 80 mmol/L of sodium azide with pH 6.8 as a solution

Standard solution: It consists of a standard cholesterol solution with a concentration of 207 mg/100 mL.

Procedure: The method for estimating the total cholesterol concentration in the blood serum of infertile and fertile males is shown in the following Table 3.3.

Table 3.3 the total cholesterol concentration in the blood serum of infertile and fertile males

Solution	Stander	Sample	Blank
Serum	-	10 µL	-
Stander	10 µL	-	-
Working solution	1.0 mL	1.0 mL	1.0 mL

The tubes were mixed well and then Incubated for 5 minutes at a temperature of 37° C. Then the absorbance was measured at a wavelength of 500 nm versus the blank using a spectrophotometer.

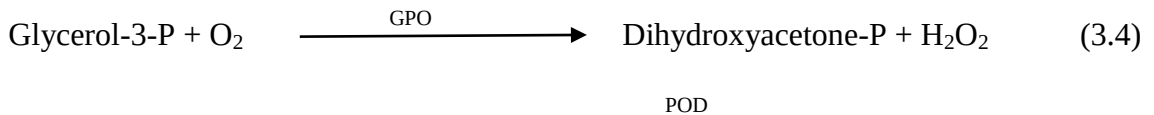
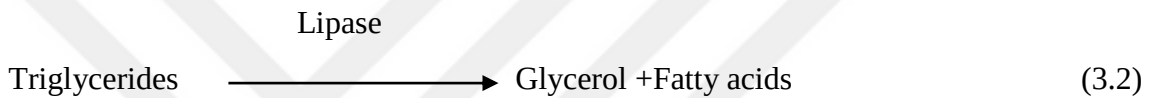
Calculation: The cholesterol concentration was calculated based on the following Equation 3.1.

$$\text{Cholesterol conc} = \frac{(A)_{\text{Sample}}}{(A)_{\text{Standard}}} \times \text{Standard Conc} \quad (3.1)$$

Standard conc.=207 mg/dL

3.7.2 Determination of Serum triglycerides Level

Principle: After lipase, an enzyme that catalyzes enzymatic hydrolysis, releases glycerol (alcohol) and free fatty acids, triglycerides are measured. Glycerol kinase converts glycerol to glycerol-3-phosphate (GK). The enzyme glycerol-3-phosphate oxidase converts glycerol-3-phosphate to dihydroxyacetone (GPO). Peroxidase (POD), an enzyme that can be detected spectrophotometrically at a wavelength of 500 nanometers, is used to produce the quinonimine-colored product (Koditssche., 1969 and Fossati., 1982) and as shown by the following Equations 3.2, Equations 3.3, Equations 3.4 and Equations 3.5



Chlorophenol:

- Enzymes reaction: Contains each of the following enzymes:
- 150,000 units/liter of lipoprotein lipase.
- 800 units/liter of glycerol kinase enzyme.
- 4000 units/liter of glycerol 3-phosphooxidase.
- 440 units/liter of peroxidase.
- 0.7 mmol/L 4-aminophenazone.
- 0.3 ATP mmol/L.

Standard solution: It consists of 200 mg/cm³ glycerol.

Working solution: It is prepared by adding 15 cm³ of buffer solution to the contents of a vial of reaction enzymes, mixed ed quietly and kept at a temperature of 8°C.

Procedure: The steps mentioned in the Table 3.4 below were used.

Table 3.4 The steps mentioned were used

Solution	Sample	Stander	Blank
Stander	-	10 µL	-
Serum	10 µL	-	-
Working solution	1 mL	1 mL	1 mL

The tubes were mixed ed well, and Incubatedd for 5 minutes at a temperature of 37 °C, then the absorbance was measured at a wavelength of 500 nanometers by means of a spectrophotometer versus an efficient one.

Calculations: The concentration of triglycerides in the blood serum is estimated based on the following Equation 3.6.

$$\text{Triglycerides concentration (mg/dL)} = \frac{A_{\text{Sample}}}{A_{\text{Standard}}} \times \text{standard conc} \quad (3.6)$$

Standrd .Conc = 200mg/dL

3.7.3 Determination of Serum HDL-C concentration

Principle: LDL-C, VLDL and chylomicrons chylimmicrons (CM) were precipitated by adding phosphotungstic acid (PTA) and magnesium chloride salt. ready-made cholesterol (Burtis 1999).

Reagents Used: The precipitation solution consists of: 13.9 mmol/liter phosphotungstic acid with 7 mmol/liter of magnesium chloride sal.

Procedure:

- Sedimentation: 0.25 cm³ of blood serum was added to a test. tube containing 0.025 cm³ of precipitation solution.
- The contents of the tube were mixed well and centrifuged for 10 minutes at 3500 rpm and at laboratory temperature.
- The process of separating the sediment from the supernatant, then measuring the concentration of HDL-C in the sediment.

The tubes were mixed well, and Incubated for 5 minutes at a temperature of 37 °C, then the absorbance was measured at a wavelength of 500 nanometers by means of a spectrophotometer versus an efficient one.

Accounts: HDL-C concentration was calculated based on the following Equation 3.7.

$$\text{HDL-C conc. (mg/dL)} = \frac{A_{\text{Sample}}}{A_{\text{Standard}}} \times \text{Standard conc.} \times 1.1 \quad (3.7)$$

*Standard conc. = 200 mg/dL

Calculation: VLDL concentration was calculated based on the following Equation 3.8 (Friedewald 1970).

$$\text{VLDL conc. (mg/dL)} = \frac{\text{TG}}{5} \quad (3.8)$$

3.7.4 Calculation of serum LDL-C concentration

The LDL-C content in blood serum was calculated (Andreoli 2001).

$\text{LDL-C conc. (mg/dL)} = \text{Total Cholesterol} - (\text{HDL-C} + \text{VLDL-C})$

$\text{VLDL} = \text{T.G} / 5$

3.8 Statistical Analysis

F. Test was employed to compare result between patients and control groups in the SPSS statistical software with a P-value of less than 0.05.

4. RESULTS AND DISCUSSION

4.1 Levels of hormones

Table 4.1 shows the levels of peptide sex hormones (Omentin-1, Adipsin, FSH, LH, and Prolactin) in the serum of women with polycystic ovarian syndrome and a control group.

Table 4.1 Mean $\pm$ SD deviation of the level of (Omentin-1, Adipsin, Prolactin, LH,FSH) for patients and healthy subjects.

Parameters	Mean $\pm$ SD		P $\leq$
	Control	Patients	
Omentine-1(ng/mL)	89.836 $\pm$ 24.054	218.291 $\pm$ 66.671	*P<0.05
Adipsin(ng/mL)	1269.376 $\pm$ 338.615	2973.955 $\pm$ 787.817	*P<0.05
Prolactin(ng/mL)	108.007 $\pm$ 57.610	583.234 $\pm$ 236.605	*P<0.05
LH(ng/mL)	48.039 $\pm$ 12.618	7.751 $\pm$ 7.147	*P<0.05
FSH(ng/mL)	1263.841 $\pm$ 341.861	363.618 $\pm$ 184.227	*P<0.05

*P<0.05

4.1.1 Level of serum omentin-1

Females with polycystic ovarian syndrome had an Omentin-1 concentration of (218.291 66.671) ng/mL, whereas the control group had an Omentin-1 concentration of (89.836 24.054) ng/mL. (the healthy ones).

As shown in Figure 4.1, women with polycystic ovarian syndrome exhibited considerably greater levels of Omentin-1 than did the control group (P0.05).

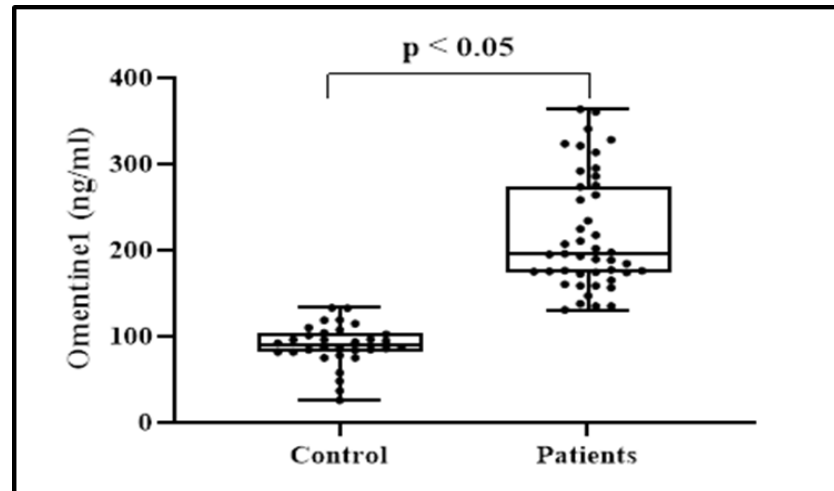


Figure 4.1 Omentin-1 levels in the sera of women with PCOS and the control group were compared

Omentin-1 levels were found to be higher in obese women on metformin than in obese women not taking the medicine, according to the present research's findings, which are in line with those found in a previous study by Kareem and his team (Kareem 2017). There were no variations in Omentin-1 levels between women with polycystic ovaries and healthy women in another investigation (Akbarzadeh 2012). When it comes to obese women with PCOS and hyperandrogenism, (ozgen *et al.* 2019) observed that omentin-1 levels were significantly lower in such individuals. In addition, PCOS is more common in women who are thin. In 2020, (Franik 2020) omentin-1 was found to be much lower in the sera of pcos patients than has been previously reported by other investigations. Women with PCOS who use oral contraceptives had greater levels of omentin-1 in their sera, according to a recent research (Anithasri *et al.* 2019). Anitha, Anitha.

Obese tissue (not subcutaneous) produces omentin, which is an aglycoprotein that plays a key function in an insLin sensitizer by enhancing the signal from the insLin receptor by activating the protein kinase pathway, thereby increasing insLin stimulation.

Human adipocytes, as well as the ovary, the placenta, the lung, and the heart, exhibit delayed glucose absorption (Yang 2006). In other words, the rise in serum omentin-1 levels might be attributable to the disease's reaction to therapy, while the medicine

combats insulin resistance by blocking hepatic glucose synthesis (Johnson 1993). As a result, hyperglycemia may influence the concentration of omentin-1 (Tan 2010). Otherwise, the reduction in inflammation after therapy with a medicine like metformin, which is widely used to treat PCOS, might be explained in part by an increase in serum omentin-1 levels.

4.1.2 Level of serum adipon hormone in the blood serum

Females with polycystic ovarian syndrome had an adipon concentration of (2973.955-787.817) ng/mL, whereas the control group's concentration was only (1269.376-338.615) ng/mL. (the healthy ones).

Figure 4.2 shows that adipon levels are considerably higher in women with polycystic ovarian syndrome ($P < 0.05$) than in the control group.

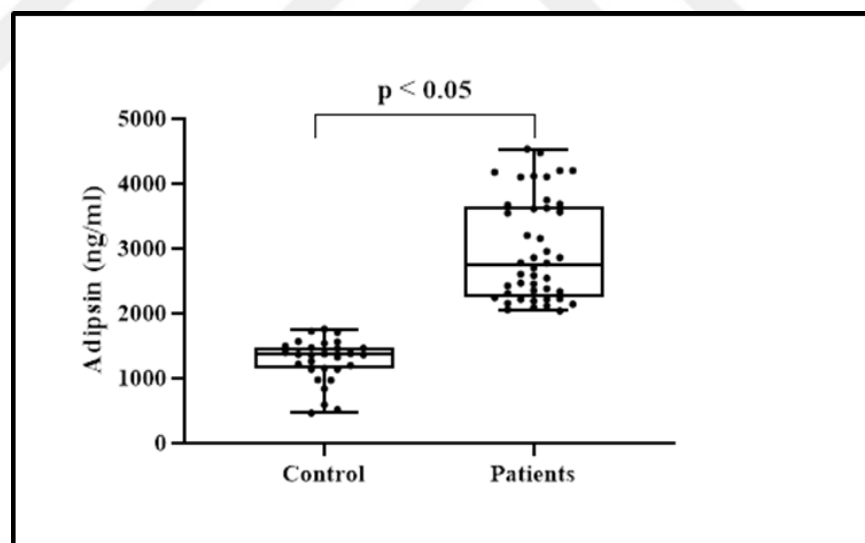


Figure 4.2 Serum adipon concentrations in women with pcos as well as those in the control group were measured

The findings of this study are in agreement with the findings of other studies, which found that the level of adipon in the sera of women with PCOS was significantly elevated and closely associated with increased risks of cardiovascular and metabolic disorders, while (Hashemi *et al.* 2012) showed that there was no significant correlation

between serum adipsin and PCOS disease (Hashemi 2012), Another study indicated that the adipsin level rose considerably with an increase in (IR) (MLinar *et al.* 2007). (MLinar 2007). Abdominal and subcutaneous fat have differing levels of angiopoietin (Adipisin). Because of this, the risk of adipisin is increased in those with a high BMI (Xia 2003).

When it comes to the body's many functions, adipcin is an important adipokine. It is involved in everything from blood coagulation to supplement activation to reproductive health to the immune system to tissue repair to high blood pressure and reproductive organ degeneration. According to (Kong 2009, Frühbeck 2001, Legakis 2016) observed that individuals with type 2 diabetes have lower levels of adipsin in the beta cells of the pancreas, adiabacin seems to play a significant role in numerous disorders (Lo 2014).

There was a 3.25-fold increase in the incidence of polycystic ovarian syndrome in women with greater levels of adipsin than in those with lower levels, suggesting that the anomalies in adipose tissue identified in PCOS patients may contribute to the development of reproductive and metabolic dysfunction.

4.1.3 Level of serum prolactin

As shown in Table 4.1, women with PCOS had a prolactin content in their blood serum that was significantly lower than that of women without PCOS. Figure 4.3 shows that the amount of prolactin in the sera of women with PCOS is considerably increased ($p < 0.05$) compared to the control group.

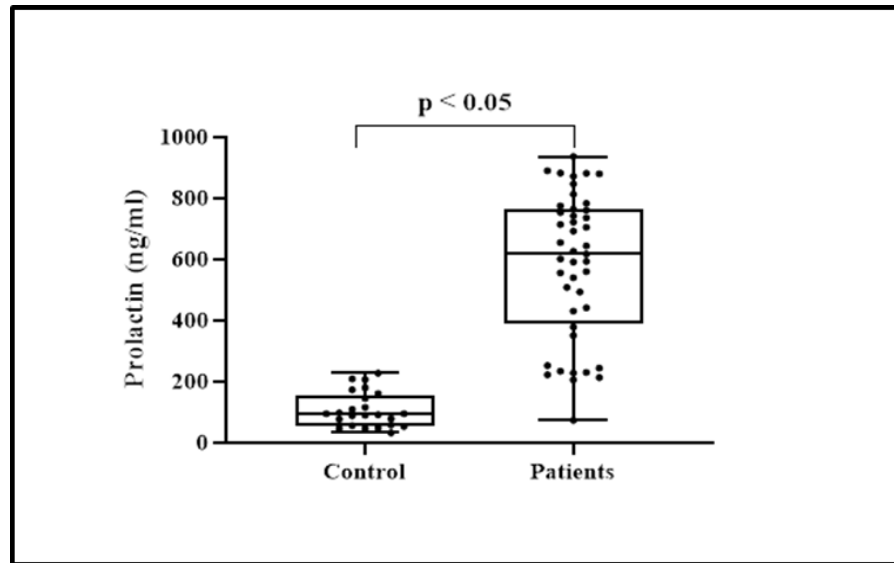


Figure 4.3 Prolactin levels in the sera of PCOS patients and healthy women

The elevation of Serum prolactin in Sera of women with PCOS may be still unclear, Although studies were conducted this elevation (Kyritsi 2018, Mahdi 2021, Davoudi 2021). In Connection with the main causes of high prolactin in sea of women with pcos, the data obtained from the recent literature are in agreement and appear to weaken the hypothesis that high prolactin is a part of the polycystic ovary Syndrome disease (Delcour 2019), In which (Hayashida *et al.* 2014) indicate that 6 % of PCOS women had high level of prolactin (Hayashida *et al.* 2014).

So the results of studies do not seem many to establish the presence of elevated prolactin in sera of PCOS women. However, the studies are still scanty results of many on this subject, and to be definite a strict study on big Cohort women with PCOS is needed to set these results.

Thus the most popular hypothesis to demonstrate the correlation between high level of prolactin and PCOS is may be popular hypothalamic - pituitary anomaly that can elucidate both hyper prolactin (Delcour 2019).

4.1.4 Level of serum LH

Table 4.4 shows that luteinizing hormone (LH) was (7.751 ± 7.147) ng/mL in the blood serum of females with polycystic ovary syndrome compared with (48.039 ± 12.618) ng/mL of the control group (the healthy ones).

Women with polycystic ovarian syndrome had considerably lower amounts of luteinizing hormone in their blood serum compared to the control group ($P < 0.05$) Figure 4.4.

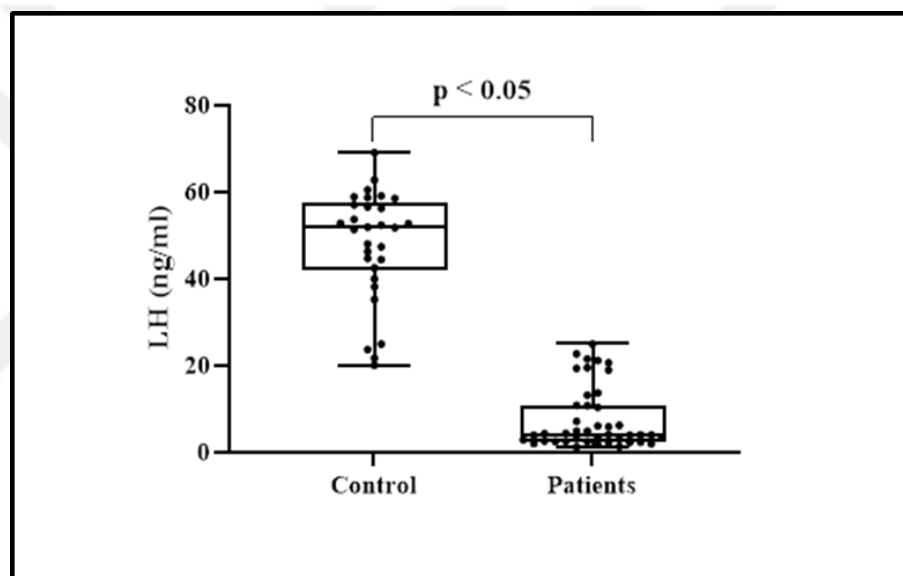


Figure 4.4 Sera from women with PCOS and a control group were tested for LH concentrations

Several studies (Christodouloupoulou 2016, Abdulwahid 2019) found that individuals with PCOS had significantly higher levels of luteinizing hormone than those who participated in the present investigation.

The reduction in the level of prolactin may be associated with low Concentration of may be Circulating insulin, which induced either dietary restriction Or by drugs (Butzow 2000, Pasquali 2000, Dunaif 1996). This study indicates that the rate of ovulation affected treatment, so it was through ambiguous if their reduction in LH level

in sera of women with PCOS was correlated with the of TON blood insuLin concentration or to the feedback an effect of reduced e While impact of high ovarian estrogen production . Several studies have been unable to indicate . concentration on LH lowering or circpLating insuLin it's responses to Gonadotropin releasing hormone after treatment with drag sor after Loss of body weight in women with PCOS (Pasquali 2000, Azziz 2001, Holte 1995).

4.1.5 Level of serum FSH

Table 4.1 shows that FSH was (363.618 ± 184.227) ng/mL in the blood serum of females with polycystic ovary syndrome compared with (1263.841 ± 341.861) ng/mL of the control group (the healthy).

Figure 4.5 shows that the amount of FSH in the blood serum of women with polycystic ovarian syndrome decreased significantly ($P < 0.05$) compared to the control group.

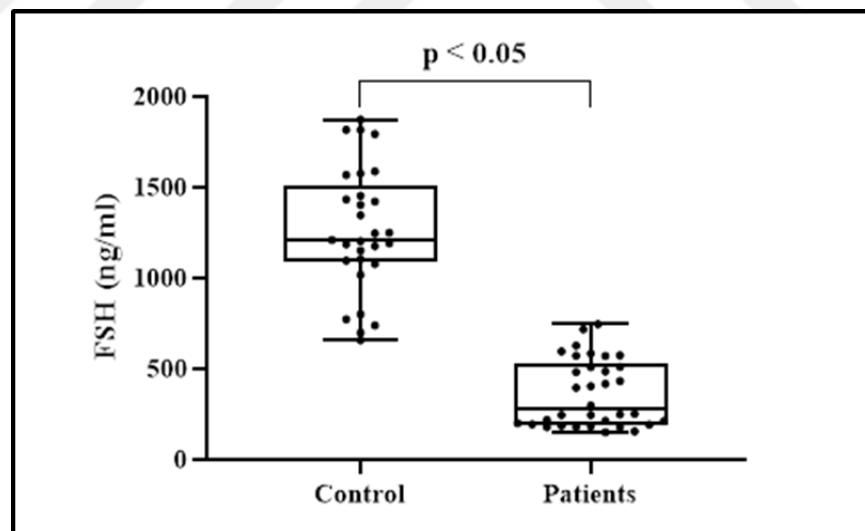


Figure 4.5 FSH levels in the sera of women with PCOS and the control group were compared

The new study's findings are in line with previous research showing that the concentration of luteinizing hormone in women with PCOS is significantly reduced, as well as the findings of the Farhod study (Farhood 2017). Which indicated that the level

of the hormone FSH did not show significant differences between the two groups of patients with PCOS and the healthy ones as a control group, and the slight decrease in the level of the hormone, which did not show a significant difference, may be due to the fact that the high concentration of prolactin may inhibit the secretion of FSH (Eldar-Geva 2001) When the adrenal gland produces adrenaline, it has an effect on the concentration of FSH, which in turn influences the concentration of the hormone via the release of androgens.

Other studies also confirmed that the level of FSH hormone did not have a significant change between the two groups of patients and control, as FSH stimulates the growth of cyst when its level increases above the normal level and thus causes failure in ovarian function through its relationship with estrogen through the negative feedback mechanism as The pituitary gland secretes high amounts of FSH in women with polycystic ovary syndrome.

4.2 Serum Lipid Profile

The level of lipids and lipoprotein (TC, TG, HDL-C, LDL-C, VLDL) was measured in the sera women with PCOS me compared with the control group, The result obtained are summarized in Table 4.2, which shows the mean $\pm$ standard deviation of the level of lipids and lipid profile in sera of patients and healthy women.

Table 4.2 Mean $\pm$ standard deviation of lipids level in the blood serum with polycystic ovary syndrome compared with the control group

Parameters	Mean $\pm$ S.D		$p \leq 0.05$
	Control	Patients	
T.C (mg/dL)	160.58 $\pm$ 30.489	171.40 $\pm$ 42.028	N.S
T.G (mg/dL)	124.35 $\pm$ 37.197	139.34 $\pm$ 55.814	N.S
HDL-C (mg/dL)	40.361 $\pm$ 6.734	31.960 $\pm$ 10.038	0.0001*
LDL-C (mg/dL)	96.377 $\pm$ 26.836	111.021 $\pm$ 30.296	0.04*
VLDL (mg/dL)	24.342 $\pm$ 7.218	30.241 $\pm$ 12.403	0.036*

4.2.1 Level of serum cholesterol

Table 4.2 shows that the level of cholesterol was (171.40 ± 42.028) mg/100 mL in sera of women with PCOS as compared with (160.58 ± 30.489) mg/100 mL as control group.

Women with PCOS exhibit no significant variations in blood cholesterol levels ($P > 0.05$) compared to their healthy counterparts, according to the results Figure 4.6.

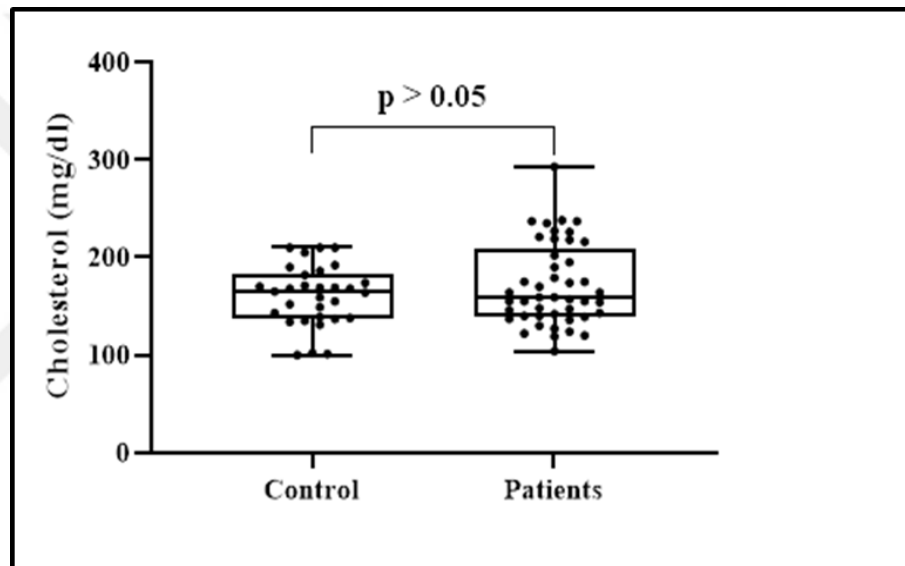


Figure 4.6 Serum cholesterol levels in PCOS women and a non-PCOS control group

The results of the current study agree with the finding study of (Luque-Ramírez 2007) and disagreement with the study of, which demonstrated that the change in lipid levels of patients with polycystic ovary syndrome is caused by a lipid disorder in women with hyperinsulinemia and androgenism. And also genetic differences in these variables that occur as a result of the environment factor (Diamanti-Kandarakis 1998).

But this is inconsistent with studies that suggest PCOS appears to increase the risk of cardiovascular disease due in part to a dyslipidemia characterized by increased plasma triglycerides and low levels of high-density lipoprotein (HDL) cholesterol (Rajkhowa).

The unchanged in the cholesterol levels between patient and control .group may be due to the lifestyle of women with PCOS in their diet ,Which depend on the diet with Low carb and fat to reduce the BMI in avoiding the over weight or obesity as one way to reduce the insuLin resistance and thus improved the owlation.

4.2.2 Level of serum triglycerides

Females with polycystic ovarian syndrome had a triglyceride level of (139.34 55.814) mg/100 mL of blood serum compared to (124.35 37.197) mg/100 mL in the control group, according to Table 4.2. (the healthy ones).

Women with polycystic ovarian syndrome and the control group had similar levels of blood serum, as shown in Figure 4.7, and there were no significant differences in the results.

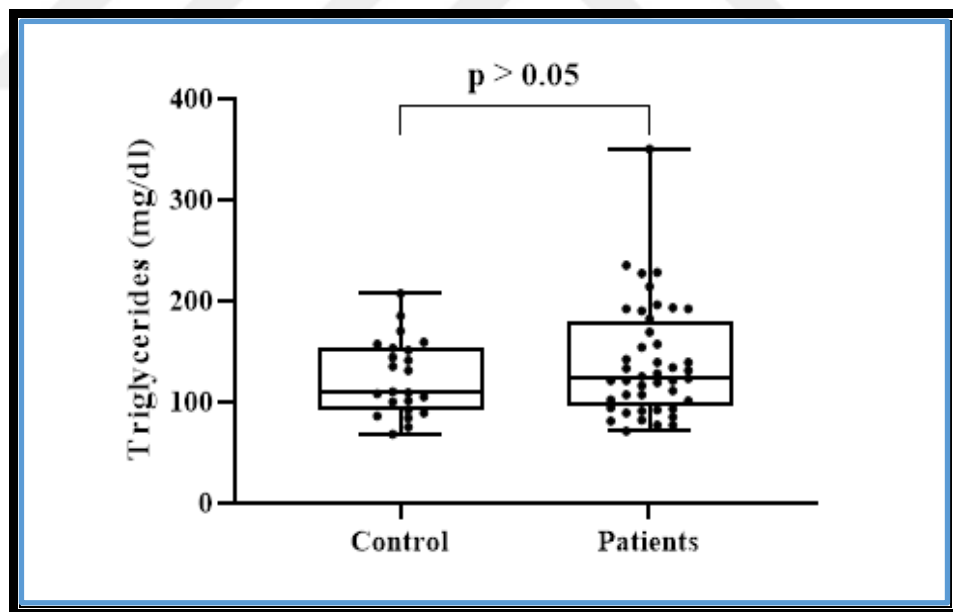


Figure 4.7 T.G. levels in the sera of women with PCOS and a control group were compared

There is agreement between the present study's findings and those found in (Luque-Ramírez 2007). Studies confirmed that one of the causes of PCOS is insuLin resistance, and that individuals who suffer from insuLin resistance, have increased Levels of (TG)

while levels of (HDL) decreased, this means that there is a relationship between (TG and HDL) and insulin resistance (Laughlin 2003). Triglycerides are the most dangerous form of fat found in the blood plasma and constitute a high percentage in most of the fat stored in adipose tissue (Noor 2017). Triglycerides can be reduced by exerting muscular and physical effort, as well as leading to the consumption of greater amounts of calories that result from the metabolism of glycerides. And fats and proteins, which leads to a decrease in their level (Bronk 1999).

4.2.3 Level of serum HDL-C

Table 4.2 reveals that among women with polycystic ovary syndrome, high-density lipoproteins - cholesterol were (31.960 10.038) mg/100 mL as opposed to (40.361 6.734) mg/100 mL in the control group (the healthy ones).

Those with PCOS who donated blood had significantly lower serum levels than women without PCOS, as seen in Figure 4.8.

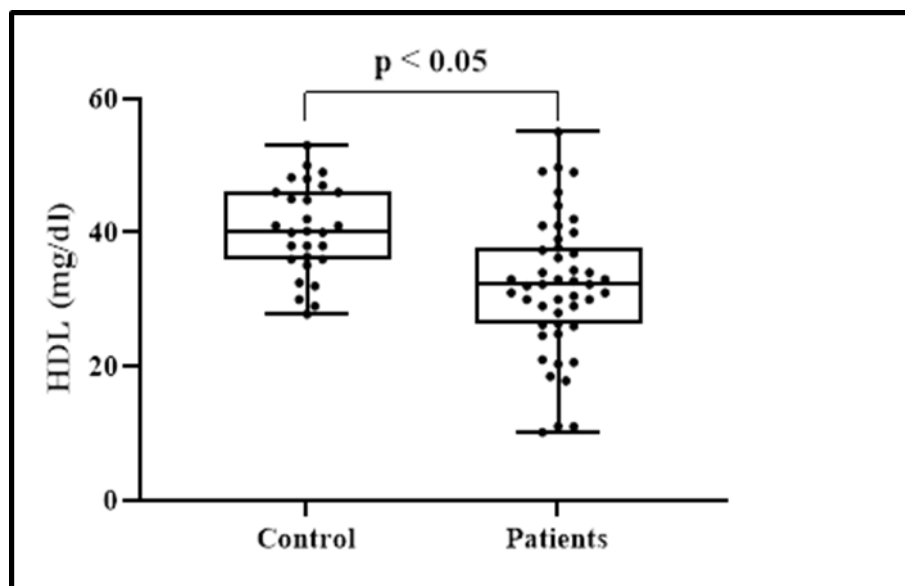


Figure 4.8 HDL-C levels in PCOS patients' blood compared to those in the control group

According to previous studies, women with PCOS are at greater risk of cardiovascular disease because of dyslipidemia, which is why the results of this research are consistent with those of Durmus U and his group (Durmus 2017) and Pinola P and his group (Pinola 2017). Additionally, obesity, insulin resistance, and hyperandrogenism are hallmarks of PCOS and may influence lipid metabolism (Rajkhowa 1997).

4.2.4 Level of serum LDL

Table 4.2 demonstrates that the LDL-C concentration in the sera of women with PCOS was (111.021 30.296) mg/100 mL, compared to (96.377 26.836) mg/100 mL in the control group.

Figure 4.9 shows a substantial rise in resLts between the PCOS sera group and the control group, with a p-value of (0.05).

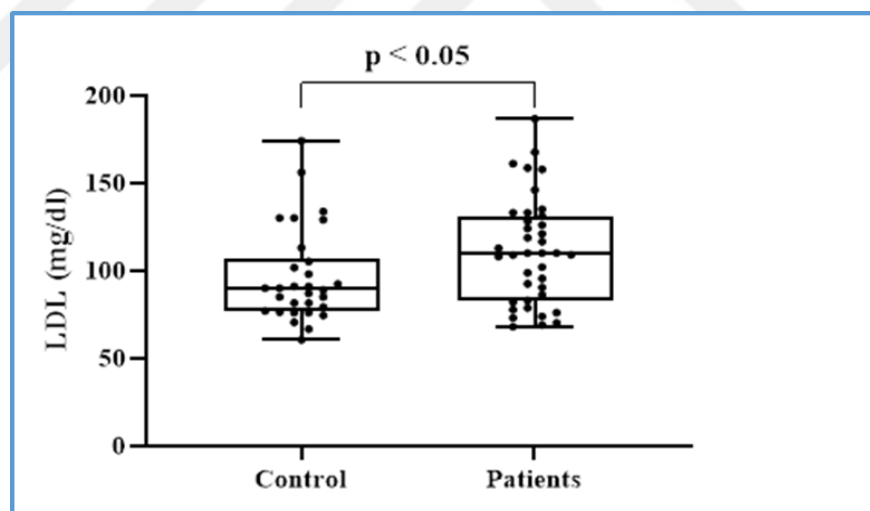


Figure 4.9 Serum LDL-C concentrations in PCOS patients and a non-PCOS control group

Robinson S. (Robinson 1996), Jayrasree R. and his colleagues found similar findings in their research (Robinson 1996, Jayrasree 2010). A substantial rise in LDL-C was seen in the sera of women with PCOS, according to the researchers. As the most prevalent endocrine condition in women of reproductive age, PCOS has been linked to

dyslipidemia, hyperandrogenism, hyperinsufficiency of fatty acids, oxidative stress, and infertility in certain studies. Dyslipidemia has been linked to the development of polycystic ovary syndrome (PCOS). Women with PCOS often have abnormalities in their lipids, and these abnormalities play a significant role in illness (Liu 2019).

As a result of their polycystic ovarian syndrome, polycystic ovary syndrome women are more likely to develop cardiovascular disease (Kumar 2017),

As a result, abnormalities in the lipid profile may have an impact on the progression of PCOS (Diamanti-ndarakis 2007).

4.2.5 Level of serum VLDL

Table 4.2 demonstrates that among women with PCOS, VLDL levels were higher (30.241 12.403) mg/100 mL than in the control group (24.342 7.218) mg/100 mL.

Serum from women with PCOS showed a substantial rise, as indicated in Figure 4.10, with a p-value of less than 0.05.

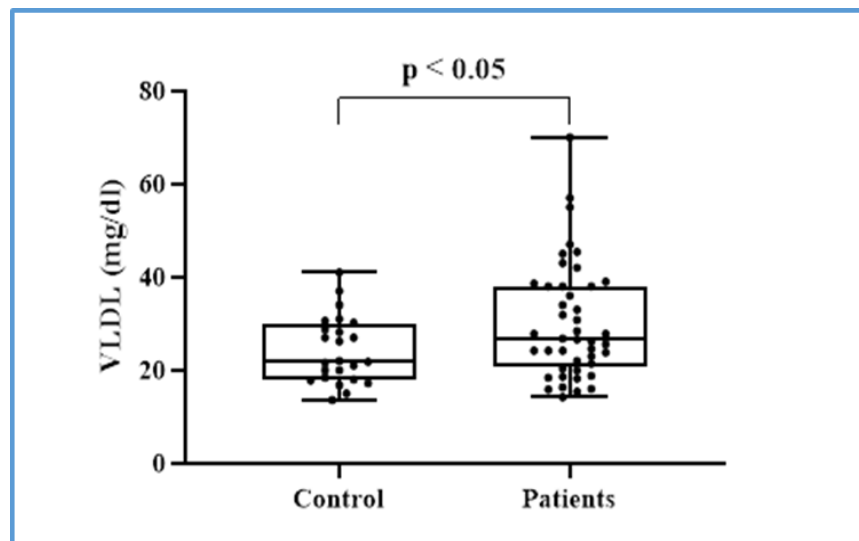


Figure 4.10 VLDL levels in the sera of women with and without pcos

The results of the present study are in agreement with those of Manjunatha (Manjunatha,2014). In PCOS, there were statistically significant increases in serum LDL, TG and VLDL while serum HDL was lower in PCOS compared to the PCOS group.

There are a lot of TGs in the VLDL molecule and apoB-100, as well as phospholipids, cholesterol, and replaceable micro-lipids, so the levels of their respect may generally be correlated with a reference TG level. Increases in VLDL should be expected as triglyceride levels rise.

As these proteins are built in the liver, especially after eating a meal rich in fat, and they participate with LDL-C in causing coronary atherosclerosis, and their secretion is inhibited by the presence of insulin in a high concentration because it reduces the processing of the proteins needed for its manufacture (Smith 2005) , This very low-density lipoprotein is also produced by eating a diet rich in fat, especially in the liver, and may also play an important role in the development of cardiovascular diseases (Rathinasabapathi 1997).

4.3 Correlation Between Omentin-1 and Biochemical Level

The relationship between the level of Omentine-1 and the level of data under consideration in the table has been studied Table 4.3.

Table 4.3 It shows the correlation between the level of Omentine-1 and the level of the studied data

Parameters	Omentine-1
	R
	Patients
Adipsin	0.673**
Prolactin	0.096
LH	0.418**
FSH	0.011
Cholesterol	0.332*
Triglycerides	0.112
HDL	0.027
LDL	0.174
VLDL	0.353*

4.3.1 The relationship between the level of Omentine-1 and the level of Adipsin in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of Adipsin in the group of patients was positive and significant, as the value of the correlation coefficient was ($r = 0.673$).

4.3.2 The relationship between the level of Omentine-1 and the level of Prolactin in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of Prolactin in the group of patients was positive, as the value of the correlation coefficient was ($r = 0.096$).

4.3.3 The relationship between the level of Omentine-1 and the level of LH in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of LH in the group of patients was positive and significant, as the value of the correlation coefficient was ($0.418 = r$).

4.3.4 The relationship between the level of Omentine-1 and the level of FSH in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of FSH in the group of patients was positive, as the value of the correlation coefficient was ($r = 0.011$).

4.3.5 The relationship between the level of Omentine-1 and the level of Cholesterol in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of cholesterol in the group of patients was a positive significant relationship, as the value of the correlation coefficient was ($r = 0.332$).

4.3.6 The relationship between the level of Omentine-1 and the level of T.G in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of triglycerides in the group of patients was positive, as the value of the correlation coefficient was ($r = 0.112$).

4.3.7 The relationship between the level of Omentine-1 and the level of HDL-C in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of HDL in the group of patients was a positive relationship, as the value of the correlation coefficient was ($r = 0.027$).

4.3.8 The relationship between the level of Omentine-1 and the level of LDL-C in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of LDL in the group of patients was positive, as the value of the correlation coefficient was ($r = 0.174$).

4.3.9 The relationship between the level of Omentine-1 and the level of VLDL in the blood serum of the samples under study

The results showed that the correlation of Omentine-1 with the level of VLDL in the group of patients was a significant positive relationship, as the value of the correlation coefficient was ($r = 0.353$).

5. CONCLUSIONS AND RECOMMENDATION

5.1 Conclusion

- In women with polycystic ovarian syndrome, the hormones omentin-1 and adiponin are found to be considerably elevated in the bloodstream.
- In women with polycystic ovarian syndrome, prolactin levels climbed dramatically, although the levels of LH and FSH in their blood serum reduced significantly.
- In terms of cholesterol and triglyceride levels, there were no significant alterations, however HDL-C levels decreased and LDL-C levels increased.
- Omentin-1 and (Adiponin, LH, cholesterol, VLDL) had a strong positive connection in the PCOS group, the data revealed.

5.2 Recommendations

- Blood serum levels of tumor necrosis factor and interleukin-6 in women with polycystic ovarian syndrome are being studied for their possible function in inducing inflammation in the body.
- Assessment of Chemerin and Visfatin concentrations in polycystic ovary syndrome patients' blood serum.

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