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EVALUATION OF LEVELS OF FOLLICLE GROWTH-STIMULATING HORMONE (FSH), LUTEINIZING HORMONE (LH) AND TESTICULAR LUTEINIZING (T) IN INFERTILE MEN

Wijdan Kamal Noor[1] and Dr. Bushra Abbas ALzubaidi[2]

[1]Ph.D-Student and [2]Advisor, Part of Thesis, Department of Biology, College of Education for Girls, University of Kufa, Iraq

ABSTRACT

The aim of the study was to determine the changes in reproductive hormones and their relationship in each of the infertility subgroups (oligospermia, asthenozoospermia, azoospermia and unexplained infertility) compared with the control group. Methods: This study included examining 200 samples after a period of abstinence of 3-5 days, where samples were collected for oligospermia (40 samples), asthenozoospermia (40 samples), azoospermia (40 samples), and unexplained infertility (40 samples).) and the control group (40 samples) whose ages ranged between (21 - 45) years in the laboratories of the Fertility Center / Al-Sadr Medical City / Najaf Governorate and the Al-Buraq and Al-Sadiq laboratories. All men included in the current study (healthy and sick) were subjected to special tests for blood samples. Semen samples, blood sample tests included measuring biochemical parameters, which included measuring reproductive hormones (follicle growth stimulating hormone (FSH), luteinizing hormone (LH), and testicular lipid hormone (T) using the FAIDS device. Conclusion: A decrease or increase in the levels of reproductive hormones may be a cause of the problem of infertility.

Keywords: FSH، LH ، T

INTRODUCTION

Infertility is the failure of a couple to conceive even after one year, which is caused by many complications and variables from one region to another, depending on a number of biological and social factors (Agarwal et al., 2021). A person's weight can have a profound impact on fertility. Men who suffer from infertility Or an increase in the ideal weight, they have a greater risk of infertility. When the body mass index is less or greater than the desired value, this may lead to fertility problems in males and may disturb the hormonal balance necessary for the natural production of sperm (Al-ubaidy, 2014), and is known as infertility. In general, it is the inability of couples to have a child after one year of regular, unprotected intercourse, which affects (10-15%) of couples (Vander-Borght and Wyns, 2018), and according to the latest World Health Organization statistics, about (50-80) suffer from it. Million) people worldwide are infertile (Cui, 2010). This problem affects 10-15% of couples in the United States. Male infertility is partly or completely responsible for about 30-55% of infertility cases. There are approximately 15% of couples suffering from infertility, including approximately 30-50%. It is linked to male infertility (Thoma et al., 2013), and the male infertility factor constitutes 30% of infertile couples, while the female infertility factor constitutes about 35%. Moreover, 20% of infertility cases have both male and female factors, while cases Unexplained or idiopathic is about 15% and male infertility factors are broadly classified as primary testicular failure (sperm dysfunction), secondary testicular failure (endocrine dysfunction) and posttesticular obstruction (Liu et al., 2005; De Rose et al., 2018). Infertility can have a female or male origin, with the male factor only being present in a third of cases. The diagnosis of infertility in men depends mainly on semen analysis in terms of semen parameters (sperm concentration, appearance, and motility) (Jamsai, et al., 2008; Punjani et al. al., 2021) Infertility in men can be the result of a variety of causes. However, there are approximately 40% of infertile men who do not have a clear cause for their disease.

There are various causes of male infertility, the most important of which are: hormonal deficiency, physical causes, sexually transmitted problems, environment and lifestyle, genetic factors, genetic mutations, aneuploidy, infectious diseases, obstruction of the ejaculatory duct, varicocele, radiation, oxidative stress, chemotherapy and inability to on erection (Krausz and Riera - Escamilla, 2018; Mari'c et al, 2021). A review of 28 previously conducted population-based surveys from various countries found that infertility prevalence rates range from 3.5% to 16.7% in more developed countries, with an average overall prevalence estimated at 9%. Based on

Australian data, infertility affects approximately 1 in 6 people. Of reproductive age couples, 42% will have male factors (which are the subject of this study) contributing to their infertility. Surrogate markers of male fertility have declined over the past four decades (Tsevat, et al., 2017) and in up to 50 % of cases the cause remains unexplained while putative mechanisms including environmental factors have been linked to idiopathic male infertility (Hart and Tadros, 2019). The role of trace elements in human semen quality and their mechanism of action has been the focus of study by many researchers (O'Connor, 2001; Shinohara et al., 2005). Many recent studies have indicated an increased prevalence of various abnormalities in the male reproductive system as possible factors that explain This phenomenon of stress, lifestyle and a variety of endocrine-altering chemicals in the environment can be linked to decreased male reproductive capacity as demonstrated primarily by the results of experimental studies on animals (Massanyi et al., 2003; 2004; 2005). Male fertility depends on the compatibility between nervous, hormonal, and psychological mechanisms, or as a result of the close relationship between the reproductive system and these mechanisms. Any obstruction of one or more of these mechanisms results in infertility. Evaluating infertility in men requires complete knowledge of the physiology of the male reproductive system and conducting studies and laboratory tests. Necessary tests, such as hormonal tests, genetic investigations, semen analysis, and performing some immunological tests, such as checking for sperm antibodies, and even urine analysis in some cases (Kollettis, 2003; Shibahara et al., 2003). Although hormonal imbalance constitutes a significant percentage of the causes of infertility in men, investigating this imbalance is essential in many cases, especially in patients with oligospermia, asthenozoospermia, and necrozoospermia, which reflects the functional state of the endocrine glands, and that hormones are the main regulator. For male reproductive function (Cannarella et al., 2020; Jin et al., 2021), hormonal evaluation of sex hormones is a vital component in the evaluation of male fertility as endocrine disorders are among the causes of male infertility and are significantly reversible (Sengupta and Dutta (2019), the hypothalamus gland produces gonadotropin-releasing hormones (GnRH), which affect the anterior or adenohypophysis lobe of the pituitary gland to encourage it to produce hormones that nourish the gonads, represented by the luteinizing hormone (LH). The Follicle Growth Stimulating Hormone (FSH) affects the testicles and works to regulate the testicular production of male hormones, such as Testosterone Hormone and reproductive gametes (Dutta et al., 2010). Other hormones affect the milk hormone Prolactin (Dutta et al., 2010). PRL, estrogen (E2), progesterone, insulin, and thyroid stimulating hormone (TSH) affect testicular function.

METHODOLOGY

This study was conducted in the laboratories of the Fertility Center of Sadr City in Al-Najaf Governorate - Najaf Health Directorate/Ministry of Health/Iraq and in Al-Buraq and Al-Sadiq National Laboratory. The study extended from 1/1/2022 to 1/1/2023, and semen samples and semen samples were collected. Serum separately from infertile patients with oligospermia, asthenospermia, azoospermia and unexplained infertility, in addition to a control group of fertile men who had at least one child within a year. Samples were taken randomly from various regions of Najaf Governorate and their ages ranged. Between (21-45) years old and their number was (200) men divided into (40) samples for each group (men who smoked and those with chronic diseases were excluded from the study). The levels of reproductive hormones (FSH), (LH) and (T) were measured in the serum. Blood using the Vidas device manufactured by Vidas-France with a ready-made test kit, following the instructions included in the test kit for FSH, LH and T hormones.

RESULTS AND DISCUSSION

Figure (1) shows a significant increase ($P<0.05$) in the FSH level of infertile patients compared to the control group, as the FSH level of the patients reached (5.086 mlU/mL) compared to the control group, which reached (2.215 mlU/mL). The results of Table (1) showed a significant increase ($P<0.05$) in the FSH level of the group of patients with infertility (oligospermia, asthenozoospermia, and azoospermia) compared to the control group, as the FSH level of the patients reached (6.770 mlU/mL), (6.253 mIU/mL) and (6.214 mIU/mL), respectively, compared to the control group, which reached (4.215 mlU/mL). The results of Table (1) also showed no significant differences ($P>0.05$) in the FSH level for the group of patients with unexplained infertility compared

with the control group, as the FSH level for patients with unexplained infertility reached (4.105 mlU/mL) compared with the control group. Which amounted to (4.215 mlU/mL). When comparing groups of patients with infertility (oligospermia, asthenospermia, azoospermia and unexplained infertility), it was noted that there was no significant difference (P>0.05) in the level of FSH between the group of patients (oligospermia, asthenozoospermia and azoospermia and azoospermia), which amounted to (6.770). mIU/mL), (6.253 mIU/mL) and (6.214 mIU/mL), respectively. It was also noted that there was a significant increase (P<0.05) in the level of FSH for the group of patients with infertility (oligospermia, asthenozoospermia, and azoospermia) compared to The group of patients with unexplained infertility, as the FSH hormone level for patients (oligospermia, asthenozoospermia and azoospermia) reached (6.770 mIU/mL), (6.253 mIU/mL) and (6.214 mIU/mL) respectively compared to patients with unexplained infertility, which It reached (4.105 mIU/mL). The results of Figure (2) indicated a significant increase (P<0.05) in the level of the LH hormone for infertile patients compared to the control group, as the level of the LH hormone for the patients reached (6.081 mIU/mL) compared with the control group, which reached (5.453 mIU/mL). . The results shown in Table (2) showed a significant increase (P<0.05) in the level of the LH hormone for groups of patients suffering from infertility (oligospermia, asthenozoospermia and azoospermia) compared to the control group.

The level of LH hormone for patients reached (6.315 mIU/mL), (6.080 mIU/mL) and (6.108 mIU/mL), respectively, compared with the control group, which reached (5.453 mIU/mL). As Table 2 shows, there is no Significant differences (P>0.05) in the level of the LH hormone for the group of patients with unexplained infertility compared with the control group, as the level of the LH hormone for patients with unexplained infertility reached (5.443 mIU/mL) compared with the control group, which reached (5.453 mIU/mL). When comparing groups of patients with infertility (oligospermia, asthenozoospermia, azoospermia and unexplained infertility), no significant difference (P>0.05) was observed in the level of the LH hormone between the group of patients (oligospermia, asthenozoospermia and azoospermia and azoospermia) which amounted to (6.315). mIU/mL), (6.080 mIU/mL) and (6.108 mIU/mL) respectively, while a significant increase (P<0.05) was observed in the level of the LH hormone for the group of patients with infertility (oligospermia, asthenozoospermia and azoospermia) compared to With the group of patients with unexplained infertility, the level of LH hormone for patients with (oligospermia, asthenozoospermia and azoospermia) reached (6.315 mIU/mL), (6.080 mIU/mL) and (6.108 mIU/mL), respectively, compared with the group of unexplained infertility patients. Interpreted, which amounted to (5.443 mIU/mL). The results in Figure (3) indicated a significant decrease (P<0.05) in the level of the T hormone for infertile patients, which reached (3.662 ng/mL) compared to the control group, which reached (6.400 ng/mL). Table (3) shows that there is a significant decrease (P<0.05) in the level of the T hormone for groups of patients with infertility (oligospermia, asthenozoospermia, and azoospermia) compared to the control group, as the level of the T hormone for the patients reached (2.888 ng/mL), (3.200 ng/mL) and (2.495 ng/mL) respectively compared to the control group which amounted to (6.400 ng/mL), while it was noted that there were no significant differences (P>0.05) in the T- hormone level for patients with unexplained infertility, as it was (6.065 ng/mL) compared to the control group, which amounted to (6.400 ng/mL). When comparing groups of patients with infertility (oligospermia, asthenozoospermia, azoospermia and unexplained infertility), it was noted that there was no significant difference (P>0.05) in the level of the T hormone between the groups of patients (oligospermia, asthenozoospermia and azoospermia and azoospermia), which amounted to (2.888). ng/mL), (3.200 ng/mL) and (2.495 ng/mL) respectively, while a significant increase (P<0.05) was observed in the level of the T -hormone in the group of unexplained infertility patients, reaching (6.065 ng/mL) compared to With totals of patients (oligospermia, asthenozoospermia and azoospermia) which amounted to (2.888 ng/mL), (3.200 ng/mL) and (2.495 ng/mL), respectively.

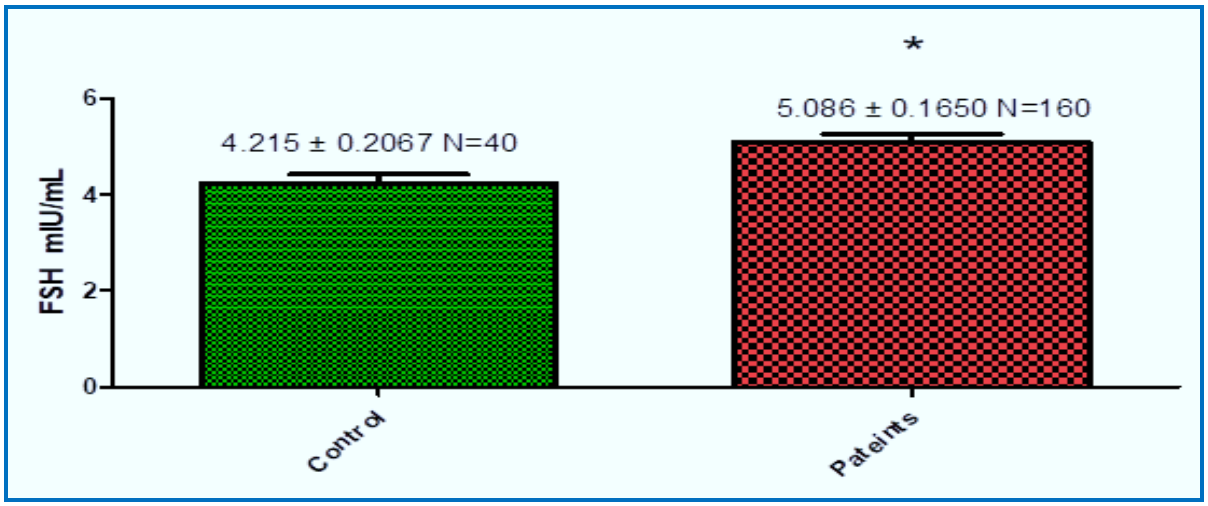

Fig.1: Level of follicle growth stimulating hormone (FSH) in infertile patients and the control group

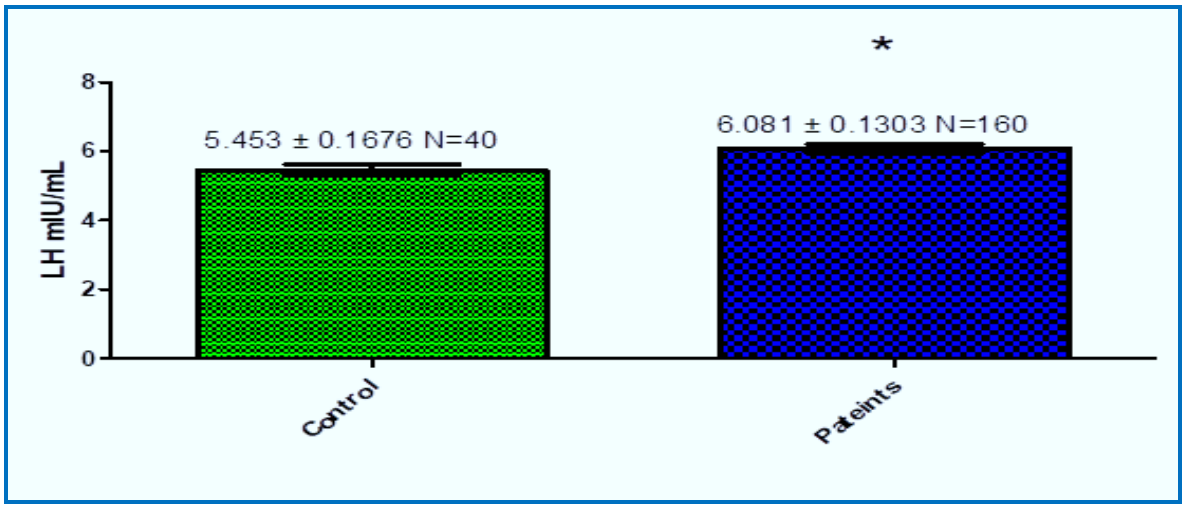

Fig.2: LH- level in infertile patients and control groups

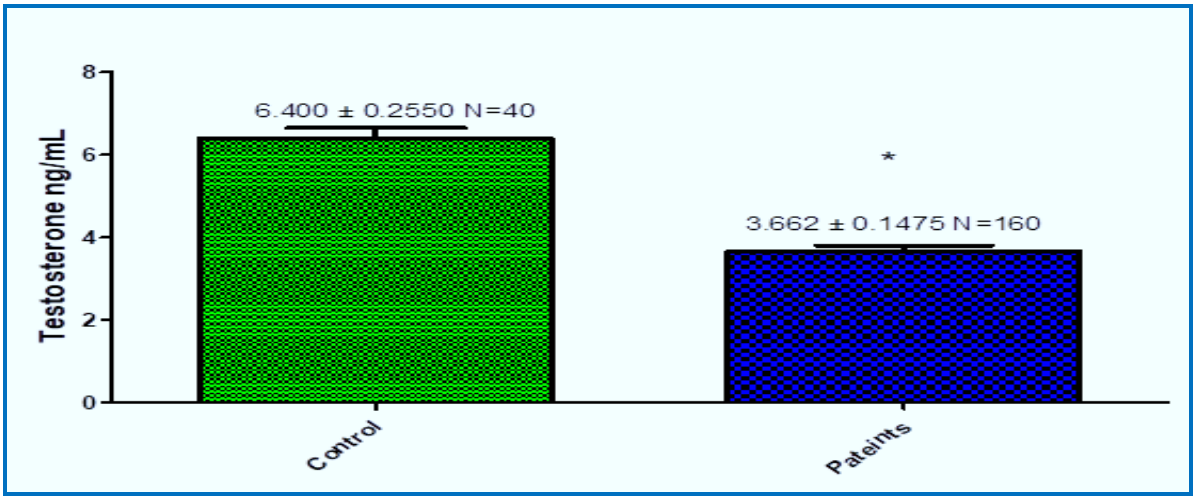

Fig. 3: The level of testicular lipid hormone Testo in infertile patients and the control group

T: represents the standard error

***:** represents a significant difference at the probability level (P<0.05) between the group of infertile patients and the control group.

N: Number of samples (40 for the control group) and (160 for the infertile patient group)

Table. 1: The level of follicle growth stimulating hormone (FSH) in groups of infertile patients and the control group

Groups		Level of follicle growth stimulating hormone (TSH) mIU/mL
Control		4.215 ± 0.2067[a]
patients	Oligospermia	6.770 ± 0.3043[b]
	Sperm weakness	6.253 ± 0.2911[b]
	Sperm death	6.214 ± 0.2786[b]
	Infertility without causes	4.105 ± 0.1816[a]

Table. 2: Luteinizing hormone (LH) level in infertile patients and the control group

Groups		Luteinizing hormone level (LH) mIU/mL
Control		5.453 ± 0.1676[a]
patients	Oligospermia	6.315 ± 0.1627[b]
	Sperm weakness	6.080 ± 0.1385[b]
	Sperm death	6.108 ± 0.1411[b]
	Infertility without causes	5.443 ± 0.1671[a]

Table.3: The level of testicular lipid hormone T in groups of infertile patients and the control group

Groups		Testicular lipid hormone level (Testo) ng/mL
Control		6.400 ± 0.2550[a]
patients	Oligospermia	2.888 ± 0.2086[b]
	Sperm weakness	3.200 ± 0.1846[b]
	Sperm death	2.495 ± 0.1415[b]
	Infertility without causes	6.065 ± 0.2308[a]

Values Represent: standard error ± arithmetic mean

Number of Samples: 40 per group

Different Letters: represent the presence of significant differences at the probability level (P<0.05) between the groups of infertile patients and the control group.

Similar Letters: represent the absence of significant differences at the probability level (P<0.05) between the groups of infertile patients and the control group.

The results of the current study indicated that there was a significant increase in the level of the hormone (FSH) for infertility patients compared with the control group, as well as when comparing groups of males with infertility (oligospermia, asthenozoospermia, and azoospermia) and the healthy group. It was also noted that there were no significant differences in the group of males with unexplained infertility. Compared to fertile males, consistent with a study (Ismael et al., 2017), the reason for this is that reproductive hormones such as follicle growth stimulating hormone (FSH) act as major regulators of germ cell development, as abnormal sperm formation is often linked to changes in gonadal hormones. In the blood (Ramesh Babu et al., 2004), the successful and complete development of male germ cells depends on the balanced interaction between the endocrine glands in the hypothalamus, pituitary gland, and testicles, and gonadotropin-releasing hormone (GnRH), which is secreted from the hypothalamus. To the release of gonadotropin hormones, namely follicle growth-stimulating

hormone (FSH) and luteinizing hormone (LH) from the pituitary gland (De Krester, 1979; Anderson et al., 1997), as follicle growth-stimulating hormone (FSH) binds to receptors in Sertoli cells and stimulates The formation of sperm and the failure of the pituitary gland to secrete it leads to a disturbance in testicular function, leading to infertility. The causes of low male fertility can be linked to an imbalance in the levels of reproductive hormones, a decrease in sexual activity, and changes in sperm motility and DNA integrity (Sartorius and Nieschlag, 2010). It was reported that Previous studies have shown that circulating levels of specific reproductive hormones in males are associated with semen quality parameters. In particular, follicle-stimulating hormone (FSH) is thought to be a marker of spermatogenesis and Sertoli cell function. It has been suggested that measuring this hormone in serum could serve as a surrogate. To measure semen quality or ability to fertilize (Meeker et al., 2007). It has been proven that reproductive hormones, including FSH, play a vital role in sperm formation and maturation, and that circulating levels of reproductive hormones were linked to sperm concentration, movement, and shape (Uhler et al., 2003; Keskin et al., 2015; Patel et al., 2016). The results of our current study are consistent with many studies (Kumanov et al., 2006; Myers et al., 2009; Jorgensen et al., 2010; Grunewald et al., 2013), which showed that there is a relationship and correlation between FSH levels. and sperm count, which indicates that this hormone was not primarily a marker of fertility, but rather a marker of semen quality and thus fertility (Barbotin et al., 2015).

She confirmed that higher levels of FSH are inversely related to the number, movement, and shape of sperm (Kumanov et al., 2006), as well as to sperm morphology, which indicates the importance of this hormone for maintaining normal morphology (Bhongade et al., 2015). Failure of the pituitary gland to secrete FSH and LH causes disruption of testicular function, leading to infertility. Evaluating FSH, LH, and T is useful in managing male infertility. FSH is necessary to begin the formation process. Sperm and its maturation (Bennet et al., 1991; Subhan et al., 1995). An increase in the level of FSH in males who suffer from oligospermia and asthenozoospermia is an indicator of the destruction and damage of seminiferous tubules (Bergmann et al., 1994). FSH is also important for predicting the histological condition of the testicle and the presence of sperm in the tissues (Barbotin et al. al., 2015) and in line with previous studies that agreed with our results (Atig et al., 2017; Makarim et al., 2017; Subramanian et al., 2018), which found that infertile males with a majority age ranges of 30- 45 years of age. It was observed that there was a significant increase in the level of the hormone (FSH) over the years. This is because there is a positive and important relationship between age, the duration of infertility, the number of children, and marital status. This can be explained by the fact that as males age, testicular function and metabolism deteriorate when the testicle undergoes changes. Age-related morphology such as a decrease in the number of germ cells, Leydeck cells, and Sertoli cells, in addition to structural changes including shrinkage of seminiferous tubules (Gunes et al., 2016; Durairajanayagam, 2018). The hormone (FSH) controls spermatogenesis and spermatogenesis by affecting both the germinal epithelium and Sertoli cells (Beastall et al., 1987). In sterile males, a high level of FSH is considered a reliable indicator of cell damage and destruction. germinal epithelium (Sheikh et al., 2005). High levels of this hormone are associated with increased severity of seminiferous epithelial destruction, which was consistent with our current study and a group of studies (Merino et al., 1980; Smith et al., 1985), and that males Infertile people and those who suffer from germinal epithelial injury, stimulation of spermatogenesis does not occur due to a decrease or deficiency in the production of proteins associated with androgens. In addition, the production of inhibin, the hormone (T), dihydrotestosterone, and estradiol is affected, which causes a disturbance in the negative feedback mechanism, leading to an increase in levels of the hormone (FSH). (Gnessi et al., 1997), high levels of FSH represent a decrease in the biological activity of the hormone, an imbalance in the responses of the pituitary gland, and the presence of damage to the germinal epithelium (Saeed et al., 1994). Moreover, the hormone stimulating follicle growth ((FSH) secretion is regulated by negative feedback from the hormone (T), inhibin, and a substance of the type protein A that is produced in Sertoli cells, and the actual production of inhibin reflects the extent of the change in sperm formation, as the increase in oligospermia as a result of the decrease in inhibin synthesis leads to an increase in the production of the hormone (FSH). FSH), so inhibin can be used as a marker for FSH, which is of great importance in examining the epithelial function of seminiferous tubules (Zhao et al., 2020). It has also been observed that

there is a negative relationship between FSH and inhibin B levels in infertile males with high levels of FSH (Subhan et al., 1995), which may be due to pituitary tumors, testicular failure, or hyperactivity of the C-axis. FSH) or it may be due to mutations in the FSH receptor (Hopkinson et al., 1997). FSH plays a key role in stimulating meiosis and DNA division in sperm (Nistal et al., 1990). Therefore, Increasing its levels may disrupt the process of spermatogenesis, which leads to a decrease in sperm count and thus infertility (Turek et al., 1995).

After statistical analysis of the results of this study, it became clear that there was a significant increase in the level of the hormone (LH) for infertile males compared to the group of fertile males, as well as when comparing groups of infertile males (oligospermia, asthenospermia, and azoospermia) to the control group, while it was noted that there were no differences. Significant for males with unexplained infertility compared to healthy males. It is very important in evaluating male infertility to take into account the levels of reproductive hormones, which have a major role in sperm formation in males (Al-Faisal, 2010), as the relationship between hormone concentration and testicular function parameters is variable. Abnormal spermatogenesis sometimes occurs in conjunction with endocrine abnormalities (Blanchard et al., 1991). The hormones (FSH), (LH), and (T) are major regulators of germ cell development. Quantitative sperm production generally requires the presence of (FSH), (LH), and (T). FSH acts directly on the seminiferous tubules while stimulating LH stimulates spermatogenesis indirectly via testicular luteinizing hormone (Anderson et al., 1997). The increase in the level of LH in the blood serum of the oligospermia and asthenospermia group compared to the control group was in agreement with (Turek et al., 1995; Reyes - Fuentes et al., 1997), who found that the increase in serum levels of gonadotropin hormone (LH may have disrupted the process of sperm formation, leading to a decrease in sperm count and lack of sperm motility and thus infertility. The primary role of LH in the male is to stimulate the production of T hormone by Leydeck cells (Weinbauer and Nieschlag, 1995), and it has been suggested that the relationship between LH and semen parameters (decreased sperm concentration, motility, and morphology) is related to disorders and compensatory mechanisms. in the hypothalamus, pituitary, and gonad (Meeker et al., 2007). The results of our current study agreed with studies (Zabul et al., 1994; Weinbauer and Nieschlag, 1995), which showed elevated levels of the hormone (LH) in men with infertility if the cause was varicocele or they had abnormal testicular tissue, as were our results. It is in agreement with (Micic, 1983), who also showed high levels of LH in sterile males, and the results were explained by the fact that they suffer from Sertoli cell syndrome only. Our study also agreed with the findings of (Sheikh et al., 2005). There are many reasons that prevent the correct functioning of the luteinizing hormone, such as mutations that affect the hormone's structure, hindering its binding to its receptors on Leydeck cells. Studies have indicated that these mutations cause disability. It is significant in sexual development and puberty by obstructing the process of manufacturing testicular steroids and thus causing infertility (Schubert et al., 2003). This is what was observed in the current study, as the levels of the hormone (T) were low, or destruction of the germinal epithelial cells of the Leydeck cells may occur. However, they remain intact (Shoab et al., 2005), and mutations may occur to hormonal receptors, causing complete or partial inhibition of the work of these receptors and an obstruction of the process of hormone binding to them, leading to a failure in the hormone process (Martens et al., 2002).

It is possible that the functioning of the luteinizing hormone is normal, but the infertility is due to reasons other than the hormone (LH). This study showed that concentrations of the hormone (LH) in the serum were inversely related to the number and movement of sperm and their natural shape as well after controlling for various lifestyle factors, which confirms the importance of the role of the hormone (LH) in the movement, number and shape of sperm, which supports the benefit of circulating levels of the hormone (LH). As a biomarker to evaluate sperm quality (Kumanov et al., 2006), and likewise another study conducted by (Meeker et al., 2007), which was consistent with our results, as significant negative correlations were reported between (LH) and sperm concentration, movement, and morphology, which Supports the central role of LH in sperm quality. This is due to the primary role of LH in males, which is stimulating the production of the hormone (T) by Leydeck cells, which then, along with the hormone (FSH), controls the formation of sperm cells and the formation of sperm in Sertoli cells (Jarow, 2003). Other studies have indicated that Gonadal failure is one of the causes of infertility and is

characterized by increased levels of (LH) and (FSH) (Beastall et al., 1987). The study of Bennet et al in 1991 was consistent with our study, which found that infertile males who suffer from oligospermia, asthenozoospermia, and azoospermia They have higher (LH) concentrations than controls, and this increase reflects the inability of the testes to form sperm normally. On the other hand, (LH) may affect the use of fructose, glucose oxidation, and adenylyl cyclase activity in sperm, which are important means through which sperm derive the necessary energy. for movement (Sheth et al., 1976). The acquisition of sperm motility occurs during sperm maturation in the epididymis, and LH receptors have been discovered in the epididymis epithelium (Sun et al., 2012; Sun et al., 2015). Moreover, in the absence of LH, the addition of (Although T) and FSH can only partially rescue the abnormal sperm phenotype (Pakarainen et al., 2005), this evidence supports our findings, suggesting a crucial role for LH in sperm motility. Recent studies support that LH may also be involved in sperm morphology (Kovac et al., 2017; Liu et al., 2017). On the other hand, two studies reported that luteinizing hormone (LH) levels do not have any relationship with the number, movement, and morphology of sperm (Subhan et al., 1995; Uhler et al., 2003), and these results did not agree with the results of our current study.

The results of the current study confirmed the presence of a significant decrease in the levels of the hormone (T) in male infertile patients compared to the control group, as well as when comparing the groups of infertile patients (oligospermia, asthenozoospermia and azoospermia) and the control group, while no significant changes were observed when compared to the control group. Comparing the group of sterile males who suffer from unexplained infertility and the fertile males, this may be due mainly to testicular dysfunction in the Leydeck cells responsible for producing this hormone, ruling out the presence of a dysfunction in the pituitary gland or hypothalamus (HoldGraft and Braun, 2004). It is possible that low levels of the hormone (T) are due to the presence of varicocele, because varicocele causes an impairment of the effectiveness of the enzyme 17B-Hydroxysteroid Dehydrogenase, which is important in facilitating the manufacture of hormone (T) from one of its sources, which is Anderstendione, and thus a decrease in the levels of this hormone occurs ((Ismail) and Barth, 2001. The testicular adipose hormone is of great importance in preserving the development and continuation of sperm, so any decrease in its levels reflects negatively on this process, and this is what actually happened and to it may be attributed oligospermia, asthenozoospermia, and sperm death, and the decrease may be due to a deficiency in sperm synthesis. The hormone itself or a defect in the enzyme 5-α Reductase, which metabolizes the hormone (T) into Dihydrotestosterone (DHT), which is the active form of the hormone (T), as it has a high affinity for binding to male hormone receptors equivalent to double the affinity possessed by the hormone (T) (McLachlan et al. al., 1996). The study we found agreed with the study of (Mohamad et al., 2016), who found a significant decrease in the levels of the hormone (T) in males who suffer from oligospermia and asthenozoospermia compared to fertile controls. The decrease in hormone levels Testicular fat may have disrupted the process of sperm formation, leading to a decrease in sperm count and infertility. The hormone (T) also showed a significant positive correlation and relationship with sperm movement, as it was suggested that the relationship of this hormone with semen factors (sperm movement) is linked to disorders. And compensatory mechanisms in the hypothalamus, pituitary gland, and reproductive glands (Meeker et al., 2007), and low levels of the hormone (T) in the group of sterile males (oligospermia, asthenozoospermia, and azoospermia) indicate abnormal formation of sperm, and this study was supported by some Previous studies (Andersson et al., 2004; Najar, 2010), and (Masud et al., 2007; Gangwar et al., 2020), who agreed with the results of our study, explained that one of the reasons for the decrease in the hormone (T) may be the increase in the prolactin hormone (Pro), which prevents the pulsatile secretion of hCG. Gonadotropin (GnRH), which causes a decrease in the pulsatile release of the hormone (T), which in turn leads to the cessation of sperm formation, impeding its movement and changing its quality, which subsequently results in secondary hypogonadism and infertility (Arowojolu et al., 2004). Also, the hormone (T) is directly and positively linked to all semen parameters. Therefore, a low level of this hormone affects the number, movement, and formation of sperm, in agreement with (Ibrahim and Ramzi, 2021). In addition, the levels of the hormone (LH) and the hormone (FSH) lead to (Andersson et al., 2004; Ulloa-Aguirre and Lira-Albarran, 2016).

Low levels of the hormone (T) are attributed to psychological stress, which is an important factor in the development of reproductive dysfunction in males (Nargund, 2015), as stressful events increase the psychological burden and accelerate disease, including infertility in males (Cachofeiro et al., 2008). Psychological stress leads to depression in addition to neuroendocrine and reproductive hormone disorders. The high level of corticosterone in the blood leads to the activation of the signals of the glucocorticoid receptors in the testicles, stops the cell cycle in sperm and destroys the reproductive function in males. Stress leads to a significant decrease in hormone levels. (T) and an increase in programmed germ cell death, which in turn leads to a gradual decrease in proteins associated with sperm development and ultimately leads to reproductive damage in males (Toufexis et al., 2014; Bhongade et al., 2015; Li et al., 2020) Studies conducted on animals also showed that chronic stress affects sexual drive and destroys testicular cells in male rats (Hou et al., 2014). Previous studies have shown that oxidative stress stimulates programmed cell death in the mitochondria and affects the levels of the hormone (T) (0An et al., 202). They confirmed damage to the mitochondria under stress and chronic psychological stress, which prevents the potential mechanism for generating the hormone (T). As is known, cholesterol participates. Blood and acetate in the synthesis of the hormone (T) through the smooth endoplasmic reticulum, mitochondria, and microbodies. Mitochondria are the most important organelles in cell energy metabolism and one of the main organelles involved in the biosynthesis of the hormone (T). Leydic cells contain a series of metabolic enzymes such as StAR and CYP11A. They are important mitochondrial proteins that regulate the transport and metabolism of cholesterol precursors (T) in mitochondria (Samie et al., 2018; Aaa and Ge, 2020). While our results did not agree with the results of the study (Eniola et al., 2012), which confirmed that there was no significant difference in the levels of the hormone (T) in the serum when comparing sterile cases and fertile controls, our study also contradicted the study of (Kawakami et al., 2000).

CONCLUSIONS

The results from our study showed that there were no significant differences between the sterile males and the control group. It also indicated that the hormone (T) had no effect on the concentration, movement and formation of sperm.

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AUTHOR CONTRIBUTIONS

The authors carried out : Conceptualization, Methodology, Investigation, Software.

DECLARATION OF COMPETING INTEREST

The authors state that they have no conflicts of interest related to the contents of this work, either financial or otherwise.

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