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Characteristics Of Hormonal Disorders In Infertility In Men

Kurbonova Zumrad Ch., Saifutdinova Zukhra A., Yarmukhamedov Alisher S., Dulanova Dilobar., Sadikova Nigora M., Islamova Zulfiya S.
Tashkent State Medical University
Institute of Human Immunology and Genomics, Academy of Sciences of the Republic of Uzbekistan

Abstract. This study is devoted to the study of the main types of hormonal disorders associated with infertility in men and the mechanisms of their development. The study analyzes the effect of changes in testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin levels on spermatogenesis. The clinical significance of hormonal imbalances, diagnostic methods, and their role in improving male reproductive health are highlighted.
Keywords. male infertility, hormonal disorders, testosterone, FSH, LH, prolactin, spermatogenesis, reproductive health.

Background

Male infertility remains one of the most pressing issues in modern medicine, affecting millions of couples worldwide and causing significant psychological, emotional, and social difficulties [1]. According to WHO data, approximately 15–20% of married couples experience difficulties in conceiving a child, and in 30–40% of cases the cause is related specifically to the male partner's health. Among the many factors influencing reproductive function, hormonal dysfunctions occupy a special place as one of the most common and complex etiological categories of male infertility [2].

Three main hormones play a key role in the regulation of spermatogenesis and sexual activity: follicle-stimulating hormone (FSH), luteinizing hormone (LH), and total testosterone (TT). These hormones are produced by the anterior pituitary gland and control processes occurring in the seminiferous tubules and interstitial tissue of the testes. Disruption of any link in this hormonal chain leads to decreased semen quality, oligo- or asthenozoospermia, and even complete absence of spermatozoa—azoospermia [3].

An increase in FSH levels indicates depletion of the seminiferous tubules' ability to produce spermatozoa. This may be associated with primary damage to the seminiferous epithelium, chronic inflammatory processes, trauma, or exposure to toxins or radiation. Elevated FSH levels are often accompanied by reduced inhibin B levels, a protein that negatively regulates FSH secretion [4]. Thus, high FSH combined with low inhibin B indicates degenerative damage to the seminiferous tubules, which is characteristic of conditions such as seminiferous tubule atrophy, androgen deficiency, and postgonadal testicular atrophy.

As for LH, it directly stimulates Leydig cells in the testes to produce testosterone. Increased LH levels may indicate secondary testosterone deficiency, when the testes fail to adequately respond to pituitary signals, or disorders affecting the hypothalamic-pituitary axis [5]. In men with pituitary hypoplasia, Cushing's disease, chronic stress, or those taking certain medications (corticosteroids, anti-hormonal drugs), elevated LH is observed as a compensatory response to reduced testosterone levels. However, in some cases, especially in chronic developmental delay or genetic mutations, excessive LH may be observed even with normal or elevated testosterone levels, indicating impaired receptor sensitivity to hormones [6].

Total testosterone (TT) is one of the most important indicators affecting not only libido and erectile function but also spermatogenesis quality. Normal TT levels range from 11 to 35 nmol/L in adult men; however, the "normal range" may vary depending on time of day, age, body weight, and other factors. In young men, levels are higher than in older individuals, while in those with obesity, metabolic syndrome, or hypogonadism, TT is often below the normal threshold. A particularly concerning condition is low testosterone combined with elevated LH, which is a classic picture of secondary (hypogonadotropic) hypogonadism, in which the pituitary gland fails to adequately stimulate the testes due to disrupted hypothalamic signaling [7].

Of particular importance is not only the absolute hormone levels but also their relationships and ratios, such as FSH/LH, LH/TT, and FSH/TT. A high FSH/LH ratio may indicate predominant seminiferous tubule

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June 2026

damage, whereas a high LH/TT ratio suggests impaired testosterone production by the testes. In addition, analysis of these ratios helps differentiate forms of hypogonadism: primary (testicular failure), secondary (hypothalamic-pituitary dysfunction), and tertiary (central disorders related to psychological stress, drugs, or neurological conditions) [8].

From a diagnostic perspective, a comprehensive hormonal assessment includes not only morning blood sampling (since testosterone levels fluctuate throughout the day) but also additional tests such as stimulation tests (using hCG, HCG, GnRH), inhibin B analysis, free testosterone measurement, and sex hormone-binding globulin (SHBG) evaluation. Clinical history is also essential, including trauma, surgeries, chronic diseases, medication use, and lifestyle factors (smoking, alcohol consumption, overwork, and obesity), all of which significantly affect hormonal status [9].

Thus, a deep understanding of hormonal changes in men with infertility is not only of scientific interest but also a clinical necessity. It enables accurate identification of reproductive dysfunction causes and the selection of appropriate treatment strategies, including hormone replacement therapy (for hypogonadism), drugs stimulating LH/FSH production (e.g., human chorionic gonadotropin), or specialized support in cases of testicular hypertrophy. Moreover, knowledge of hormonal status helps predict the likelihood of successful conception, including through assisted reproductive technologies such as in vitro fertilization (IVF) with intracytoplasmic sperm injection (ICSI), which is particularly relevant in severe spermatogenic disorders.

Therefore, comprehensive hormonal diagnostics is an essential component of reproductive specialists', endocrinologists', and urologists' work. Only an objective assessment of all parameters, their dynamics, age-related features, and individual characteristics allows not only accurate diagnosis but also the development of a personalized, targeted, and effective reproductive rehabilitation plan.

Aim of the Study

The aim of this study is to analyze the hormonal profile of 380 infertile men divided into five groups according to FSH, LH, and TT levels, and to identify the relationship between different types of hormonal disorders and patients' clinical status.

Materials and Methods

The study included 380 men suffering from infertility. For each group, FSH, LH, and TT levels were determined. Patients were divided into the following groups:

1. Hypergonadotropic hypogonadism (HyperG) – men with primary testicular failure, in whom FSH and/or LH are compensatorily elevated, and TT is decreased.
2. Normogonadotropic hypogonadism (NormoG) – men with normal FSH and LH levels but reduced TT levels.
3. Normogonadotropic eugonadism (NormoE) – men with normal levels of all hormones.
4. Hypergonadotropic eugonadism (HyperE) – men with compensated primary hypogonadism, in whom FSH and/or LH are elevated but TT remains normal.
5. Hypogonadotropic hypogonadism (HypoG) – men with low FSH and LH levels, leading to reduced TT.

Hormone levels (FSH, LH, TT) were determined using a standard chemiluminescent immunoassay method. Reference values were taken from the literature:

- FSH: 1.5–12.4 IU/L
- LH: 1.42–7.6 IU/L
- TT: 9.72–31.6 nmol/L

Results of the Original Research

The presented results demonstrate testosterone (TT) levels in patients divided into five groups, each characterized by a specific state of hypogonadism or eugonadism. These groups differ both in mean TT levels and in their ranges, allowing analysis of various disorders of the hypothalamic–pituitary–gonadal axis and their relationship with androgen levels.

Hypergonadotropic hypogonadism (HyperG) includes patients with a pronounced testosterone deficiency accompanied by elevated gonadotropin levels (LH and FSH), indicating primary testicular failure. In this group, the mean TT level is 4.20 nmol/L, reflecting a significant reduction in testosterone secretion. The TT

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June 2026

range varies from 0.47 to 9.72 nmol/L, corresponding to different degrees of androgen deficiency depending on disease severity.

In normogonadotropic hypogonadism (NormoG), testosterone levels are reduced despite normal LH and FSH levels. The mean TT level in this group is 6.66 nmol/L, with a range from 1.46 to 12.10 nmol/L. This condition is most often associated with hypothalamic–pituitary dysfunction or external factors such as chronic diseases, obesity, or prolonged stress. Unlike hypergonadotropic hypogonadism, gonadotropin levels remain within the normal range, indicating impaired androgen regulation at a higher level.

In normogonadotropic eugonadism (NormoE), the mean testosterone level is significantly higher, at 20.81 nmol/L, with a range from 12.10 to 31.60 nmol/L. This range is characteristic of healthy men with high physical activity, low fat mass, and good adaptive capacity, which corresponds to normal LH and FSH levels. Such a hormonal profile may also be observed in men following diets low in carbohydrates and rich in proteins and minerals.

In contrast, hypergonadotropic eugonadism (HyperE) is characterized by elevated testosterone levels alongside increased LH and/or FSH concentrations. The mean TT level in this group is 16.26 nmol/L, with a range from 12.10 to 26.80 nmol/L, which may indicate compensatory mechanisms aimed at correcting androgen imbalance. This condition may also be associated with long-term use of steroid drugs or agents stimulating gonadotropin secretion.

The last group, hypogonadotropic hypogonadism (HypoG), shows reduced levels of both testosterone and gonadotropins. The mean TT level in this group is 4.67 nmol/L, ranging from 1.68 to 9.45 nmol/L. This condition is often associated with central nervous system damage, hypothalamic tumors, as well as the consequences of radiotherapy or brain trauma. Disruption of hypothalamic–pituitary regulation leads to decreased secretion of both gonadotropins and testosterone, resulting in secondary androgen deficiency.

FSH and LH Analysis

Analysis of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels in infertile men revealed significant differences between the groups, allowing identification of characteristic features of each condition.

In the hypergonadotropic hypogonadism (HyperG) group, FSH and LH levels were significantly elevated. The mean FSH level was 21.47 IU/L, ranging from 12.60 IU/L to 44.80 IU/L, while the mean LH level was 17.13 IU/L, ranging from 8.70 IU/L to 33.90 IU/L. These findings indicate impaired testicular function with preserved compensatory pituitary activity.

In the normogonadotropic hypogonadism (NormoG) group, FSH and LH levels remained within normal ranges. The mean FSH level was 7.36 IU/L (range: 4.00–10.00 IU/L), while LH averaged 7.09 IU/L (range: 4.00–10.00 IU/L). This condition suggests normal pituitary function despite reduced testosterone levels.

In the normogonadotropic eugonadism (NormoE) group, FSH levels averaged 9.99 IU/L, ranging from 5.15 to 12.40 IU/L, while LH levels averaged 8.42 IU/L, ranging from 2.36 to 10.40 IU/L. These data reflect normal or slightly increased pituitary activity alongside high testosterone levels.

In the hypergonadotropic eugonadism (HyperE) group, FSH and LH levels were also elevated. The mean FSH level was 15.05 IU/L (range: 12.50–29.00 IU/L), while LH averaged 11.61 IU/L (range: 4.20–20.40 IU/L). These values may reflect a compensatory pituitary response to androgen imbalance despite normal or elevated testosterone levels.

Finally, in the hypogonadotropic hypogonadism (HypoG) group, FSH levels were markedly reduced, with a mean of 1.20 IU/L (range: 0.90–1.45 IU/L). Similarly, LH levels were low, with a mean of 1.36 IU/L (range: 0.70–1.65 IU/L), indicating hypothalamic–pituitary dysfunction leading to combined FSH and LH deficiency.

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June 2026

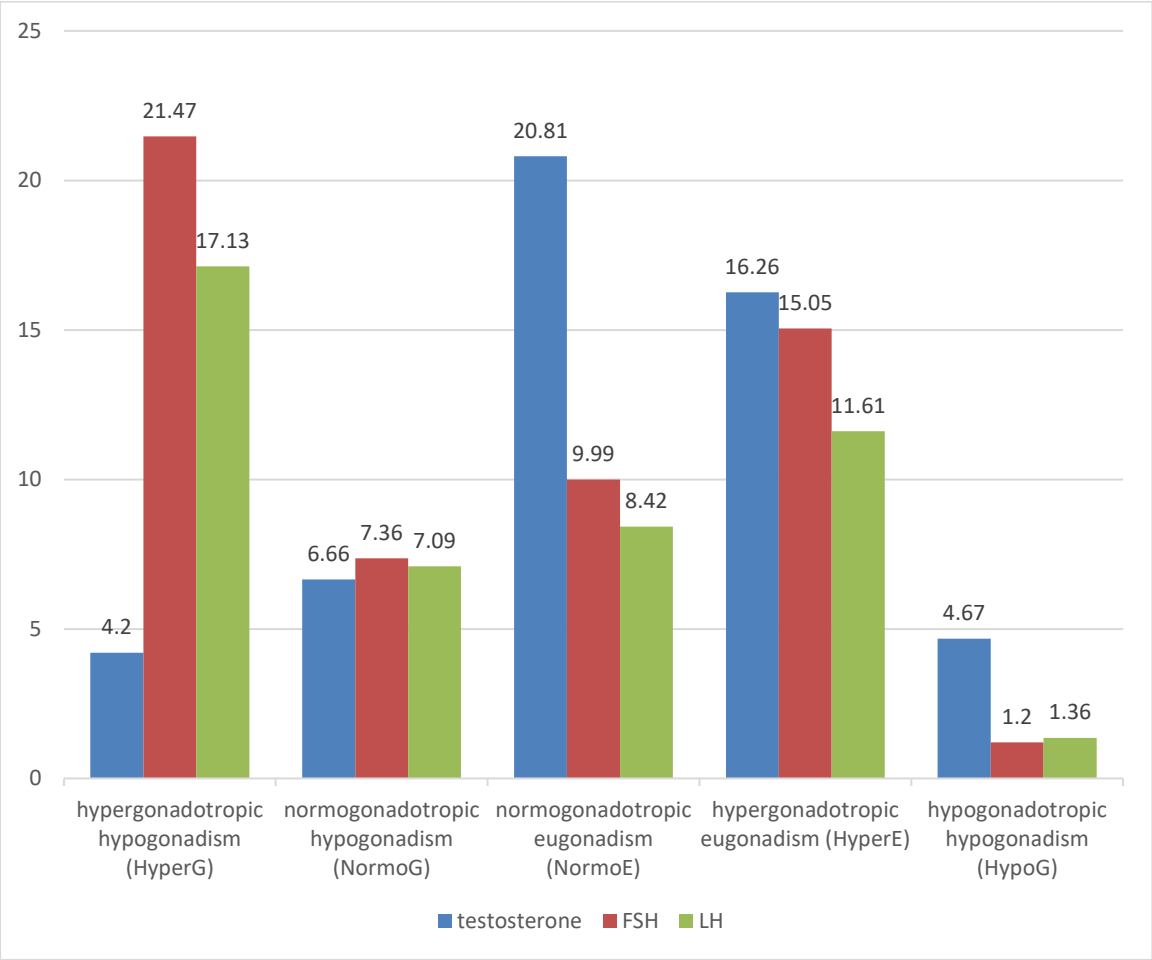

Figure. Mean hormone levels across different groups of hypogonadism and eugonadism

Thus, the analysis of mean testosterone levels and their ranges across different groups provides a deeper understanding of the mechanisms underlying dysfunction of the hypothalamic–pituitary–gonadal axis. These data play an important role in the differential diagnosis of various types of hypogonadism and eugonadism, as well as in selecting the optimal treatment strategy for each patient.

Discussion

Analysis of hormonal profile parameters showed that the group with hypergonadotropic hypogonadism (HyperG) exhibited the lowest levels of total and free testosterone, ranging from 0.5 to 1.2 nmol/L, which is a typical marker of primary testicular failure. These values reflect reduced androgen-producing capacity of the testes, which is further supported by their atrophic size and decreased scrotal pigmentation observed in most patients. At the same time, a marked compensatory increase in follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels was observed, reaching 40–60 IU/L and 35–50 IU/L, respectively. This indicates an active pituitary response to insufficient testicular stimulation: the organism attempts to restore hormonal balance within the hypothalamic–pituitary–gonadal axis through increased gonadotropin secretion; however, due to irreversible testicular damage, this compensation remains ineffective.

Patients with normogonadotropic eugonadism (NormoE) demonstrated high testosterone levels, averaging 18–22 nmol/L (normal >12 nmol/L), indicating fully preserved testicular function and intact hypothalamic–pituitary axis regulation. FSH and LH levels were within the physiological range (FSH: 3–12 IU/L; LH: 3–12 IU/L), excluding both hormonal excess and deficiency. This group was characterized by stable psycho-emotional status, well-developed muscular structure, normal libido, and absence of hypogonadal symptoms, confirming adequate endocrine regulation.

In the group with hypogonadotropic hypogonadism (HypoG), simultaneously low levels of testosterone (7–9 nmol/L on average) and gonadotropins (FSH and LH below 2–3 IU/L) were observed. This pattern is characteristic of secondary (central) hypogonadism caused by hypothalamic or pituitary dysfunction. The most common causes include head trauma, tumors of the hypothalamic–pituitary region (craniopharyngioma,

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June 2026

pituitary adenoma), genetic mutations (associated with the KAL1 gene), and prolonged stress leading to temporary suppression of GnRH secretion. A particularly severe clinical presentation is observed in Kallmann syndrome, where both FSH and LH secretion is absent due to impaired neuronal migration of gonadotropin-releasing hormone (GnRH) neurons. Despite the absence of overt clinical signs, such patients often present with reduced libido, depression, weight loss, delayed sexual maturation, and absence of spermatogenesis. The key diagnostic distinction between groups is evident: HyperG reflects pituitary overactivity with primary testicular failure, NormoE represents physiological harmony of all system components, while HypoG reflects a "silent" system due to lack of central stimulation.

The results demonstrated significant differences in hormonal levels among patient groups, indicating distinct mechanisms of male infertility. In particular, elevated FSH and LH levels in the HyperG group indicate primary testicular insufficiency. In this case, the pituitary increases gonadotropin secretion in an attempt to stimulate the testes, which are unable to produce sufficient testosterone. This is further supported by elevated LH levels, which are a characteristic marker of impaired testicular function.

In the NormoG group, normal FSH and LH levels were observed, indicating preserved pituitary function. However, testosterone levels remained low, suggesting testicular dysfunction, possibly due to structural or functional abnormalities such as underdevelopment or damage limiting testosterone production.

Patients with normogonadotropic eugonadism (NormoE) showed normal levels of all hormones, indicating proper functioning of the hypothalamic–pituitary axis. In these cases, infertility may be related to non-hormonal factors such as mechanical obstruction or genetic causes.

The group with hypergonadotropic eugonadism (HyperE) demonstrated elevated gonadotropin levels with normal testosterone levels, which may indicate a compensated form of primary hypogonadism. This condition is often observed in early stages when the testes are still able to maintain normal testosterone production despite reduced function.

In the hypogonadotropic hypogonadism group (HypoG), reduced levels of both FSH and LH were observed, indicating secondary dysfunction related to pituitary or hypothalamic impairment. This leads to insufficient testicular stimulation and, consequently, decreased testosterone production. This condition requires further evaluation of hypothalamic and pituitary function.

Thus, variations in FSH and LH levels among infertile men reflect the heterogeneity of hormonal disorders and underlying causes of infertility. Accurate hormonal assessment is essential for effective diagnosis and individualized treatment planning.

Conclusions

1. Infertile men can be classified into several hormonal types, allowing differentiation of disease mechanisms and causes.
2. Hormonal assessment, including FSH, LH, and testosterone levels, plays a key role in diagnosing male infertility and guiding optimal treatment strategies.
3. Hormonal disorders must be considered in infertility management, especially in hypo- and hypergonadotropic conditions.
4. Variations in FSH and LH levels confirm the diversity of hormonal causes of infertility, ranging from primary testicular failure to hypothalamic–pituitary dysfunction.
5. Elevated FSH and LH with low testosterone (HyperG) indicate primary testicular failure, whereas normal gonadotropins with low testosterone (NormoG) suggest testicular dysfunction.
6. Comprehensive evaluation of hormonal parameters is essential for accurate diagnosis and appropriate treatment selection.
7. In patients with hypogonadotropic hypogonadism (HypoG), hypothalamic and pituitary functions should be carefully evaluated, as specialized diagnostic and therapeutic approaches may be required.
8. This study confirms the importance of a comprehensive approach to the diagnosis and treatment of male infertility, considering individual hormonal profiles.

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June 2026

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