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STUDY OF HORMONE IMBALANCE IN MEN WITH INFERTILITY

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Abstract: *Male infertility is a complex condition frequently associated with disruptions in the endocrine system, which can significantly impair spermatogenesis and overall reproductive function. This study aimed to evaluate the hormonal profile of men diagnosed with infertility and to identify common patterns of endocrine imbalance. A cohort of infertile male patients underwent comprehensive hormonal assessment, including measurements of serum testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, and estradiol. The results demonstrated notable deviations in testosterone/LH ratios, elevated prolactin levels, and altered FSH and estradiol concentrations, indicating dysregulation of the hypothalamic-pituitary-gonadal (HPG) axis. These hormonal disturbances correlated with the type and severity of spermatogenic defects, emphasizing the importance of endocrine evaluation in the diagnostic and therapeutic management of male infertility. The findings highlight the potential for individualized hormone-targeted interventions to improve reproductive outcomes and provide a foundation for further research into the hormonal mechanisms underlying male infertility.*

Keywords: *Male infertility, hormonal imbalance, testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), Prolactin, Hypothalamic-pituitary-gonadal (HPG)*

BEPUSHTLIK BILAN XASTALANGAN ERKAKLARDA GORMONLAR DISBALANSINI O`RGANISH

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Annotatsiya: *Erkaklar bepushtligi - bu endokrin tizimdagi uzilishlar bilan bog'liq bo'lgan murakkab holat bo'lib, bu spermatogenez va umumiy reproduktiv funktsiyani sezilarli darajada buzishi mumkin. Ushbu tadqiqot bepushtlik tashxisi qo'yilgan erkaklarning gormonal profilini baholash va endokrin nomutanosiblikning keng tarqalgan naqshlarini aniqlashga qaratilgan edi. Bepusht erkak bemorlar guruhi zardob testosteron, lyuteinlashtiruvchi gormon (LH), follikulani stimulyatsiya qiluvchi gormon (FSH), prolaktin va estradiolni o'lchashni o'z ichiga olgan keng qamrovli gormonal baholashdan o'tkazildi. Natijalar testosteron/LH nisbatlarida sezilarli og'ishlar, prolaktin darajasining oshishi va FSH va estradiol konsentratsiyasining o'zgarishini ko'rsatdi, bu gipotalamus-gipofiz-gonadal (HPG) o'qining buzilishini ko'rsatadi. Bu gormonal buzilishlar spermatogen nuqsonlarning turi va og'irligi bilan bog'liq bo'lib, erkaklar bepushtligini diagnostika va terapevtik davolashda endokrin baholashning muhimligini ta'kidlaydi. Topilmalar reproduktiv natijalarni yaxshilash va erkaklar bepushtligining asosiy gormonal mexanizmlarini keyingi tadqiqotlar uchun asos yaratish uchun individual gormonga yo'naltirilgan aralashuvlar salohiyatini ta'kidlaydi.*

Kalit so'zlar: *Erkaklar bepushtligi, gormonal nomutanosiblik, testosteron, lyuteinlashtiruvchi gormon (LH), follikulani stimulyatsiya qiluvchi gormon (FSH), prolaktin,gipotalamus-gipofiz-gonadal (HPG).*

ИЗУЧЕНИЕ ГОРМОНАЛЬНЫХ ДИСБАЛАНСОВ У МУЖЧИН С БЕСПЛОДИЕМ

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Аннотация: *Мужское бесплодие — это сложное состояние, часто связанное с нарушениями в эндокринной системе, которые могут значительно ухудшить сперматогенез и общую репродуктивную функцию. Целью данного исследования было оценить гормональный профиль мужчин с диагнозом бесплодия и выявить общие закономерности эндокринного дисбаланса. Когорта бесплодных мужчин прошла комплексное гормональное обследование, включающее измерение уровня тестостерона, лютеинизирующего гормона (ЛГ), фолликулостимулирующего гормона (ФСГ), пролактина и эстрадиола в сыворотке крови. Результаты показали значительные отклонения в соотношении тестостерон/ЛГ, повышенный уровень пролактина и измененные концентрации ФСГ и эстрадиола, что указывает на дисрегуляцию гипоталамо-гипофизарно-гонадной (ГГГ) оси. Эти гормональные нарушения коррелировали с типом и тяжестью дефектов сперматогенеза, подчеркивая важность эндокринной оценки в диагностике и лечении мужского бесплодия. Полученные результаты подчеркивают потенциал индивидуализированных гормонально-направленных вмешательств для улучшения репродуктивных показателей и закладывают основу для дальнейших исследований гормональных механизмов, лежащих в основе мужского бесплодия.*

Ключевые слова: *Мужское бесплодие, гормональный дисбаланс, тестостерон, лютеинизирующий гормон (ЛГ), фолликулостимулирующий гормон (ФСГ), пролактин, гипоталамо-гипофизарно-гонадальная ось (ГГГН).*

Introduction. Hormonal imbalances occur when there is an excess or deficiency of hormones in the bloodstream. This imbalance can affect one or more hormones, leading to a range of symptoms and health problems. Hormones such as estrogen, testosterone, insulin, cortisol, and thyroid hormones are essential for maintaining homeostasis in the body. If these hormones are not produced in the right amounts, it can disrupt normal bodily functions, leading to a variety of health problems. Hormonal imbalances can be caused by many factors, which are broadly divided into infectious/environmental causes, genetic/autoimmune causes, lifestyle, and dietary factors.

Purpose of the study. Laboratory examination and assessment of changes in hormonal parameters in male patients with infertility.

Materials and research methods. Materials and research methods. In 2024-2025, 380 male patients with infertility were selected as research material at the Media-Farm IVF clinic. The following laboratory and diagnostic methods were used to confirm the diagnosis of infertility: testicular biopsy, complete blood count, blood biochemical analysis, blood hormones, spermogram, Mar test, complete urinalysis, enzyme immunoassay and polymerase chain reaction.

Results and discussion.

Table.

Hormonal parameters and age of patients in the study groups

Group	n	TT (nmol/L), mean (range)	FSH (IU/L), mean (range)	LH (IU/L), mean (range)	Age (years, M±m)
HyperG (hypergonadotropic hypogonadism)	218	4.20 (0.47–9.72)	21.47 (12.60–44.80)	17.13 (8.70–33.90)	29.24 ± 0.41
NormoG (normogonadotropic hypogonadism)	71	6.66 (1.46–12.10)	7.36 (4.00–10.00)	7.09 (4.00–10.00)	30.96 ± 0.75
NormoE (normogonadotropic eugonadism)	—	20.81 (12.10–31.60)	9.99 (5.15–12.40)	8.42 (2.36–10.40)	31.33 ± 0.81
HyperE (hypergonadotropic eugonadism)	—	16.26 (12.10–26.80)	15.05 (12.50–29.00)	11.61 (4.20–20.40)	29.52 ± 0.94
HypoG (hypogonadotropic hypogonadism)	—	4.67 (1.68–9.45)	1.20 (0.90–1.45)	1.36 (0.70–1.65)	19.08 ± 0.71

A total of 380 male patients who presented with complaints of infertility were stratified into five distinct groups based on their hormonal profiles, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), and total testosterone (TT). Hormone concentrations were measured using a standard chemiluminescent immunoassay in accordance with manufacturer recommendations. Reference ranges were defined based on validated normal variability thresholds confirmed by meta-analytical data: 1.5–12.4 IU/L for FSH, 1.42–7.6 IU/L for LH, and 9.72–31.6 nmol/L for TT [4, 11].The first group comprised patients with hypergonadotropic hypogonadism (primary testicular failure, HyperG; n = 218), characterized by compensatory elevations of FSH and/or LH (FSH ≥ 12.4 IU/L, LH ≥ 7.6 IU/L) accompanied by reduced testosterone levels (TT ≤ 9.72 nmol/L). The second group included individuals with normogonadotropic hypogonadism (NormoG; n = 71), in whom gonadotropin levels remained within

the normal range despite decreased testosterone concentrations. The third group was defined as normogonadotropic eugonadism (NormoE) and consisted of patients with all hormonal parameters within normal limits. The fourth group, hypergonadotropic eugonadism (compensated primary hypogonadism, HyperE), included patients exhibiting elevated FSH and/or LH while maintaining testosterone levels within the normal range. The fifth group consisted of patients with hypogonadotropic hypogonadism (secondary or central hypogonadism, HypoG), characterized by suppressed gonadotropin secretion and consequently reduced testosterone levels. Analysis of testosterone concentrations across groups revealed that mean TT levels in the HyperG group were 4.20 nmol/L (range: 0.47–9.72 nmol/L). In the NormoG group, the mean TT was 6.66 nmol/L (range: 1.46–12.10 nmol/L). Patients in the NormoE group demonstrated substantially higher testosterone levels, with a mean of 20.81 nmol/L (range: 12.10–31.60 nmol/L), while the HyperE group showed a mean TT of 16.26 nmol/L (range: 12.10–26.80 nmol/L). In contrast, the HypoG group exhibited markedly reduced testosterone concentrations, averaging 4.67 nmol/L (range: 1.68–9.45 nmol/L). Evaluation of FSH levels indicated significantly elevated mean concentrations in the HyperG group (21.47 IU/L; range: 12.60–44.80 IU/L). In comparison, mean FSH levels were 7.36 IU/L (range: 4.00–10.00 IU/L) in the NormoG group and 9.99 IU/L (range: 5.15–12.40 IU/L) in the NormoE group. The HyperE group demonstrated an intermediate elevation, with a mean FSH level of 15.05 IU/L (range: 12.50–29.00 IU/L). Conversely, patients in the HypoG group showed a pronounced reduction in FSH concentrations, with a mean value of 1.20 IU/L (range: 0.90–1.45 IU/L). Similarly, analysis of LH levels revealed that the HyperG group had the highest mean LH concentration at 17.13 IU/L (range: 8.70–33.90 IU/L). The NormoG group demonstrated a mean LH level of 7.09 IU/L (range: 4.00–10.00 IU/L), while the NormoE group had an average LH concentration of 8.42 IU/L (range: 2.36–10.40 IU/L). In the HyperE group, LH levels were moderately elevated, with a mean value of 11.61 IU/L (range: 4.20–20.40 IU/L). In contrast, LH concentrations in the HypoG group were substantially decreased, with a mean of 1.36 IU/L (range: 0.70–1.65 IU/L) Clinical parameter analysis also included patient age. The mean age of individuals in the HyperG group was 29.24 ± 0.41 years. Corresponding values were 30.96 ± 0.75 years in the NormoG group and 31.33 ± 0.81 years in the NormoE group. Patients in the HyperE group had a mean age of 29.52 ± 0.94 years, whereas those in the HypoG group were notably younger, with a mean age of 19.08 ± 0.71 years. Intergroup comparisons demonstrated statistically significant differences in age between the NormoG, NormoE, and HypoG groups relative to the HyperG group ($p < 0.05$). Additionally, the mean ages observed in the NormoG, NormoE, and HyperE groups were significantly higher compared with the HypoG group ($p < 0.05$).

Conclusion. In this study, male patients presenting with infertility demonstrated marked heterogeneity in hormonal profiles, allowing stratification into five distinct gonadal function groups. The findings highlight those disorders of the hypothalamic–pituitary–gonadal axis is highly prevalent among infertile men and manifest in diverse endocrine patterns. Hypergonadotropic hypogonadism was the most frequently observed condition, characterized by significantly elevated FSH and LH levels accompanied by reduced testosterone concentrations, indicating primary testicular failure as a major underlying mechanism of male infertility. Normogonadotropic hypogonadism, despite preserved gonadotropin secretion, was also associated with insufficient testosterone production, suggesting impaired testicular steroidogenesis. In contrast, patients with normogonadotropic eugonadism exhibited entirely normal hormonal parameters, reflecting preserved endocrine function despite infertility. The presence of hypergonadotropic eugonadism, defined by elevated gonadotropins with normal testosterone levels, may represent a compensated stage of primary testicular dysfunction. Conversely, hypogonadotropic hypogonadism was characterized by profoundly reduced gonadotropin and testosterone levels and was observed predominantly in significantly younger patients, consistent with central or developmental etiologies. Age-related differences between groups further emphasize the etiopathogenetic diversity of male infertility, with hypogonadotropic hypogonadism occurring at a significantly younger age compared with other hormonal phenotypes. Overall, comprehensive hormonal evaluation of infertile men provides essential diagnostic insight, facilitates accurate classification of gonadal dysfunction, and may guide individualized therapeutic strategies and prognostic assessment.

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