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Hormonal and Semen Profiles in Infertile Men from Maysan Province, Iraq

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ABSTRACT

Background & Objective: Male factors account for approximately 50% of global infertility cases. This study aimed to compare the relationship between key serum reproductive hormones Luteinizing Hormone (LH), Follicle-Stimulating Hormone (FSH), and testosterone and semen parameters in infertile men versus healthy, fertile controls across various clinically defined infertility causes.

Materials & Methods: A comprehensive descriptive cross-sectional study was conducted in Maysan Province, Iraq. A total of 477 participants were enrolled, including 393 infertile men and 84 age-matched healthy controls. The infertile group was divided into six categories: Obstructive Azoospermia, Non-obstructive Azoospermia, Primary Hypogonadism, Secondary Hypogonadism, Asthenozoospermia, and Teratozoospermia. All participants underwent serum hormone analysis using validated ELISA techniques and detailed semen analysis. Female-factor infertility was carefully excluded to focus solely on male-specific causes.

Results: Significant differences in hormonal profiles were observed among the groups. FSH levels were markedly elevated in Non-obstructive Azoospermia and Primary Hypogonadism compared to healthy controls and the Obstructive Azoospermia group. LH concentrations were highest in Primary Hypogonadism and Non-obstructive Azoospermia, while suppressed in Secondary Hypogonadism. Testosterone levels were significantly lower in both Primary and Secondary Hypogonadism compared to healthy controls. Semen analysis showed distinct impairments across the infertile groups consistent with their underlying causes, while healthy controls exhibited optimal values.

Conclusion: This study characterizes the distinct hormonal and semen parameter profiles associated with various male infertility etiologies in an Iraqi population. The findings highlight that FSH is a beneficial biomarker for differentiating between testicular failure and post-testicular causes, and confirm that integrating multiple parameters is a more effective approach than relying on a single marker for improved diagnosis and prognostic assessment in male infertility management.

Keywords: Asthenozoospermia, Azoospermia, Hypogonadism, Iraq, Male Infertility, Teratozoospermia

1. Introduction

Male infertility is now a highly alarming health concern across the globe, with an average of 17.5% of adults affected all over the world. The male factor is the only cause of infertility in 20-30% of cases and is associated with approximately 50% of all infertility cases (1). The most alarming trend is that prevalence has risen in male infertility over the last four decades, and meta-regression analyses have shown a decline in sperm concentration and total sperm count that has not been explained, all signifying an urgent demand for more research and clinical explanation in andrology (2).

Male infertility pathophysiology is primarily regulated by the hypothalamic-pituitary-gonadal (HPG) axis, a coordinately integrated endocrine system that controls spermatogenesis and male reproductive function (3). Luteinizing hormone (LH) acts as a central regulator of this axis, stimulating Leydig cells to produce testosterone, which is crucial for the initiation and maintenance of spermatogenesis (4). Follicle-stimulating hormone (FSH)

similarly acts on Sertoli cells to promote germ cell proliferation, maturation, and survival, in addition to the fact that both hormones are indispensable for optimal sperm production (5). Testosterone is the main androgenic hormone; it optimally sustains spermatogenesis, maintains secondary sexual characteristics and libido in males, and serves as a critical indicator of reproductive health (6).

The advances in the techniques of diagnosis and assisted reproductive technologies have been so enormous that the underlying cause of male infertility remains unclear in almost 30% of the cases that fall in the category of idiopathic infertility (7). In addition, this area becomes even more complicated by the traditional seminal analysis which focuses solely on the assessment of the male parameter of fertility while providing a partial view of his reproductive potential (8). More recently, research has been leaning toward endocrine biomarkers as complementary diagnostic tools, where hormonal profiling is poised to provide valuable insights into what actually occurs in abnormal spermatogenic conditions (9). The updated reference values for semen parameters are provided by the World Health Organization (WHO) 2021 guidelines and establish the standard procedure for assessing male fertility (10). However, in actual clinical practice, the integration of comprehensive hormonal assessment and typical semen analysis has not been widely used, particularly in developing regions where these specialized andrology services may not be available (8, 11).

Another hurdle in male infertility research is the striking regional imbalance. There are very broad studies on Western and Asian populations, but little to no knowledge exists on hormonal profiles and forms of reproduction in the Iraqi population. Such geographical bias in research limits the generalizability of existing findings and speaks to the need for studies specific to populations that can informevidence-based clinical practice. Difficulties that challenge male infertility go beyond simple hormonal measurements and into various clinical phenotypes like obstructive and non-obstructive azoospermia, primary and secondary hypogonadism, Asthenozoospermia, and Teratozoospermia (7). Each condition was associated with, distinct hormonal signatures and diagnostic challenges that made it necessary to develop comprehensive evaluation approaches integrating multiple biomarkers rather than single-parameter assessments (12). Recent technological advances, including artificial intelligence applications and novel molecular biomarkers, have opened new avenues for the diagnosis and management of male infertility (13). However, rigorous validation studies across various populations and health care settings would be needed to translate these innovations into everyday clinical practice (2).

By conducting a thorough examination of serum hormonal parameters (FSH, LH, and testosterone) and basic sperm parameters (volume, pH, vitality, and concentration) across seven clinically defined male categories in the Iraqi population, this study aims to fill knowledge gaps. To create population-specific reference frameworks for evaluating male fertility, systematic comparisons were conducted between healthy controls and various infertility phenotypes, including obstructive azoospermia, nonobstructive azoospermia, primary and secondary hypogonadism, asthenozoospermia, and teratozoospermia. This study aims to advance our knowledge of some male reproductive endocrinology to derive clinical relevance with respect to diagnostic accuracy and therapeutic decision-making involved in managing male infertility. It will do this by combining well-characterized cross-sectional studies with thorough hormonal profiling and more thorough semen analysis.

2. Materials and Methods

2.1 Aim of Study

The study aims to compare serum LH, FSH, and testosterone levels, as well as analyze semen parameters, among the descriptive cross-sectional study of infertile men (n=393) and healthy male controls (n=84). Using a descriptive cross-sectional design and working closely with gynecologists to more effectively exclude cases due to female-factor infertility, the study systematically identifies the underlying causes of male infertility.

2.2 Study Design

The study was carried out at several hospitals in Maysan Province, Iraq, including the Specialized Surgery Hospital (Al-Mustasfi Al-Jarahi Al-Thaksasou) in Al-Amara, Al-Sader Hospital (Maysan Province), Hakim Hospital (in Al-Majar Al-Kabir), Al-Kahla Hospital (in Al-Majar Al-Kabir), and Al-Majar Al-Kabir Hospital. From a pool of 400 infertile males (cases) and 100 healthy, age-matched men (controls), participants were chosen. To ensure thestudy only looked at male factors, selection procedures included a thorough medical history, physical examination, laboratory testing, consultation with gynecologists, and exclusion of female causes of infertility. Males between the ages of 20 and 45 who had at least one document attesting to a year of infertility, no significant illnesses that could impair reproductive health, and no chromosomal or genetic abnormalities were eligible participants. The controls had confirmed fertility, were age-matched, and were in good health. Every participant underwent thorough clinical and laboratory evaluations, including a thorough semen analysis performed in accordance with WHO criteria and the determination of serum LH, FSH, and testosterone levels using proven immunoassay techniques. It was created with the goals of maximizing diagnostic accuracy, fostering research group comparability, and accurately describing male infertility, hormonal, and seminal characteristics in this area (14, 15).

2.3 Serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone analysis

ELISA kits for each hormone were used in accordance with the manufacturer's standard protocols for the

quantitative measurement of serum levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone (Human FSH ELISA Kit, EH202RB, Invitrogen; Human LH ELISA Kit, EHLH, Invitrogen; Human Testosterone Rapid ELISA Kit, EIATES, Invitrogen). Serum was obtained by centrifuging intravenous blood samples, letting them clot for a short time at room temperature, and then storing them at -20°C until analysis. For each experiment, pre-coated wells of a microtiter plate containing monoclonal antibodies specific for FSH, LH, or testosterone were supplemented with known quantities of hormone calibrators, quality controls, and patient serum samples in duplicate.

After an incubation period, biotinylated detection antibodies and enzyme conjugates (horseradish peroxidase) were added sequentially, following the specified incubation and washing steps to remove unbound reagents and ensure specificity. The chromogenic substrate (TMB) was added, and the reaction was stopped after sufficient color development with the stop solution, producing an optical density proportional to the hormone concentration in each sample. Absorbance was measured at 450 nm using a calibrated microplate spectrophotometer. Standard curves for FSH and LH were generated using a four-parameter logistic (4PL) model with absorbance values from kit standards, ensuring a good fits (R^2>0.99). Hormone concentrations in the study samples were then interpolated from these curves. Negative and positive controls were included in each run, and all samples were analyzed in duplicate to ensure reliability and reproducibility. Inter- and intra-assay coefficients of variation were maintained below 10% **(16)**.

2.4 Semen Parameters Evaluation

Semen Collection and Analysis Participants were advised to withhold ejaculation for 2–5 days before the collection of semen. Samples of semen were procured by masturbation in sterile cups and transported to the laboratory within 60 minutes of collection at body temperature. The semen analysis was carried out using standardized protocols, following the latest recommendations of the WHO, principally. The parameters included semen volume, pH, sperm motility, and sperm concentration, with validated methods selected to ensure accuracy and reproducibility **(17)**.

2.5 Semen Volume

The semen volume was measured by accepting the whole ejaculate into a calibrated graduated container. Measurements were made by direct pipetting, and results were expressed in milliliters (mL) as the WHO recommended.

2.6 Semen pH

Semen sample pH was assessed immediately upon liquefaction using pH indicator strips of appropriate sensitivity or a calibrated pH meter. It gives some critical insight into the acid-base state of the ejaculate, which in turn can have an impact on sperm function or further fertility potential assessment.

2.7 Sperm Vitality

Sperm vitality was assessed through eosin-nigrosin staining. Briefly, a small aliquot of semen is mixed with eosin-nigrosin solution, and then a smear is prepared on a glass slide, air-dried, and examined under a light microscope. Vital (live) spermatozoa exclude or resist dye entry and therefore appear white or only faintly colored, indicating intact plasma membranes. In contrast, non-viable (dead) sperm that have compromised membrane integrity absorb the eosin dye and therefore appear pink or red.

2.8 Sperm Concentration

The sperm concentration was determined using a well-mixed aliquot of semen, appropriately diluted, and loaded into a hemocytometer (counting chamber). Spermatozoa were counted under a light microscope within designated grid areas, and the concentration was calculated according to the WHO protocol formula:

$$\text{Concentration}\left(\frac{\text{millions}}{\text{mL}}\right) = \frac{\text{Average count per square} \times \text{dilution factor} \times 10^6}{Valum\ of\ counted\ square\ (mL)}$$

2.9 Quality Control and Data Analysis

All assays included internal quality controls, and the manufacturer's guidance was followed. Hormonal and semen analysis data for the infertile and control groups were compared statistically, using the appropriate statistical tools, with $p < 0.05$ set as the significance threshold. The methodology is intended to study the hormonal and seminal profiles of infertile men exhaustively to generate strong data for assessing the male-specific causes of infertility **(18)**.

3. Result

3.1 FSH Levels as Key Diagnostic Tool for Male Infertility

In the present study, the FSH values observed in healthy controls (180±35 pg/mL) align closely with the international reference range, supporting their use as a physiological baseline and confirming the appropriateness of this control group (Table 1 and Fig. 1A). Obstructive azoospermia (110.81±25.4 pg/mL) typically presented with normal FSH secretion, showing no significant difference compared to controls (p>0.05), because spermatogenesis remains intact in this condition. Conversely, non-obstructive azoospermia showed a marked increase in FSH levels (754±120 pg/mL) compared to both controls and the obstructive group (p<0.001). Similarly, in primary hypogonadism (926±86 pg/mL), FSH remained significantly elevated (p<0.001) due to loss of negative feedback. In contrast, FSH remained low or inappropriately normal in secondary hypogonadism (60.34±17.3 pg/mL), which was significantly lower than all other infertile groups (p<0.01).

A Mild to moderate FSH elevation was observed in cases of Asthenozoospermia (192±43 pg/mL; reduced motility) and Teratozoospermia (853±131 pg/mL; morphological defects), with the latter showing a significant difference compared to controls ($p<0.05$).

3.2 LH Hormone Testing as a Powerful Tool for Male Fertility Diagnosis and Treatment Planning

In healthy controls (46.20±8.70 pg/mL), average LH levels fell within the typical physiological range. Men with obstructive azoospermia (40.54±7.21 pg/mL) showed normal serum LH levels that were similar to those of healthy men and significantly lower ($p<0.05$) than levels found in men with non-obstructive azoospermia (107.3±42.28 pg/mL). Primary hypogonadism (122.8±70.40 pg/mL) demonstrated a marked and significant increase in serum LH levels ($p<0.05$) compared to all fertile and non-obstructive groups. In contrast, secondary hypogonadism (15.7±4.6 pg/mL) was characterized by significantly suppressed ($p<0.05$) or inappropriately normal LH concentrations. Asthenozoospermia (41.76±10.21 pg/mL) showed mildly elevated LH levels not significantly different from controls ($p>0.05$). Similarly, Isolated Teratozoospermia (31.47±8.52 pg/mL) showed no significant deviations in LH levels compared to fertile controls ($p>0.05$) (Table 1 and Fig. 1B).

3.3 Testosterone Profiling as a Critical Tool for Diagnosing and Treating Male Infertility

Total testosterone levels in healthy controls were 5.60±1.20 ng/mL, falling at the higher end of the normal reference range (3.5 to 7.0 ng/mL) (Fig. 1C). Testosterone levels in men with obstructive azoospermia (5.26±1.1 ng/mL) were within the normal range, showing no significant difference compared to healthy controls ($p>0.05$). In contrast, testosterone levels were significantly lower in the non-obstructive azoospermia group (3.65±1.07 ng/mL) ($p<0.05$), frequently indicating low-androgen states. Primary hypogonadism (2.7±0.80 ng/mL) and secondary hypogonadism (2.26±0.74 ng/mL) both registered significantly low values for serum testosterone ($p<0.01$ for both *vs.* controls). Finally, men suffering from isolated Asthenozoospermia (3.92±1.42 ng/mL) and Teratozoospermia (4.71±1.53 ng/mL) displayed serum testosterone levels that were only slightly, but not significantly, lower than those found in healthy controls ($p>0.05$) (Table 1).

3.4 Comprehensive Sperm Analysis as a Powerful Diagnostic Tool for Male Infertility Subtypes

The analysis comprised the systematic evaluation of four fundamental sperm parameters: semen volume (Fig. 2A), semen pH (Fig. 2B), sperm vitality (Fig. 2C), and sperm concentration (Fig. 2D) across seven clinically defined patient groups. These case-control included healthy controls and six subfertile/infertile groups: obstructive azoospermia, non-obstructive azoospermia, primary hypogonadism, secondary hypogonadism, Asthenozoospermia, and Teratozoospermia.

Semen parameters obtained from healthy controls consistently met or exceeded reference values, establishing a robust baseline against which the subfertile and infertile groups were measured (Table 2). Patients diagnosed with obstructive azoospermia, despite being azoospermic, presented with semen volumes and pH values that were not significantly different from the healthy control group ($p>0.05$ for both parameters). Conversely, the NOA group was characterized by normal to mildly diminished semen volume and a significantly alkaline pH ($p<0.05$ *vs.* controls), coupled with absent sperm and significantly reduced vitality ($p<0.0001$ where sperm were rarely detected), which is indicative of severe spermatogenic dysfunction.

Concentrations of sperm in primary hypogonadism were recorded as low, below the clinical criteria for severe oligospermia, and with severely depressed vitality, that is, the findings of volume were less than normal and pH was alkaline. In secondary hypogonadism semen volume and sperm concentration were generally low with mild reduction in vitality and pH. Asthenozoospermia was characterized by severely slow sperm motility along with sperm concentrations, normal close to vitality reduced moderately and normal semen volume and pH. In Teratozoospermia, spermatozoa possess a decreased percentage of morphologically normal forms with sperm concentration, semen volume, and pH remaining normal along with preservation of vitality.

Table 1. Comparative analysis of key endocrine parameters-FSH, LH, and testosterone (expressed as mean ± SD)-across seven clinically defined male groups.

	Healthy (n=84)	Azoospermia (n=131)		Hypogonadism (n=132)		Semen abnormalities (n=130)	
		Obstructive (n=42)	Non-obstructive (n=89)	Primary (n=76)	Secondary (n=56)	Asthenozoo spermia (n=83)	Teratozoo spermia (n=47)
FSH (ρg/mL) mean±SD	180±35	110.81±25.4	754±120	926±86	60.43±17.3	192±40	853±131
LH (ρg/mL) mean±SD	46.20±8.70	40.54±7.21	107.3±42.28	122.8±70.40	15.7±4.6	41.76±10.21	46.20±8.70
Testosterone (ng/mL) mean±SD	5.60±1.20	5.26±1.1	3.65±1.07	2.7±0.80	2.26±0.74	3.92±1.42	4.71±1.53

Table 2. Comparative analysis of fundamental sperm parameters-semen volume, semen pH, sperm vitality, and sperm concentration (expressed as mean ± SD)-across seven clinically defined male groups.

	Healthy (n=84)	Azoospermia (n=131)		Hypogonadism (n=132)		Semen abnormalities (n=130)	
		Obstructive (n=42)	Non-obstructive (n=89)	Primary (n=76)	Secondary (n=56)	Asthenozoo spermia (n=83)	Teratozoos permia (n=47)
Semen Volume (mL) mean±SD	3.25±0.86	1.58±0.62	2.95±0.74	1.42±0.71	1.60±0.53	2.59±0.86	1.92±1.40
Semen pH mean±SD	7.65±0.33	7.21±0.41	7.35±0.38	7.2±0.20	7.3±0.20	7.47±0.46	7.71±0.14
Sperm Vitality (%) mean±SD	77.50±10.20	53.50±18.4	47.57±9.46	52.27±12.80	46.27±12.54	44.68±13.20	53.50±14.20
Sperm Concentration (millions/mL) mean±SD	60±22	Not semen	Not semen	5.25±3.5	2.78±1.40	29.50±9.48	48.15±15.43

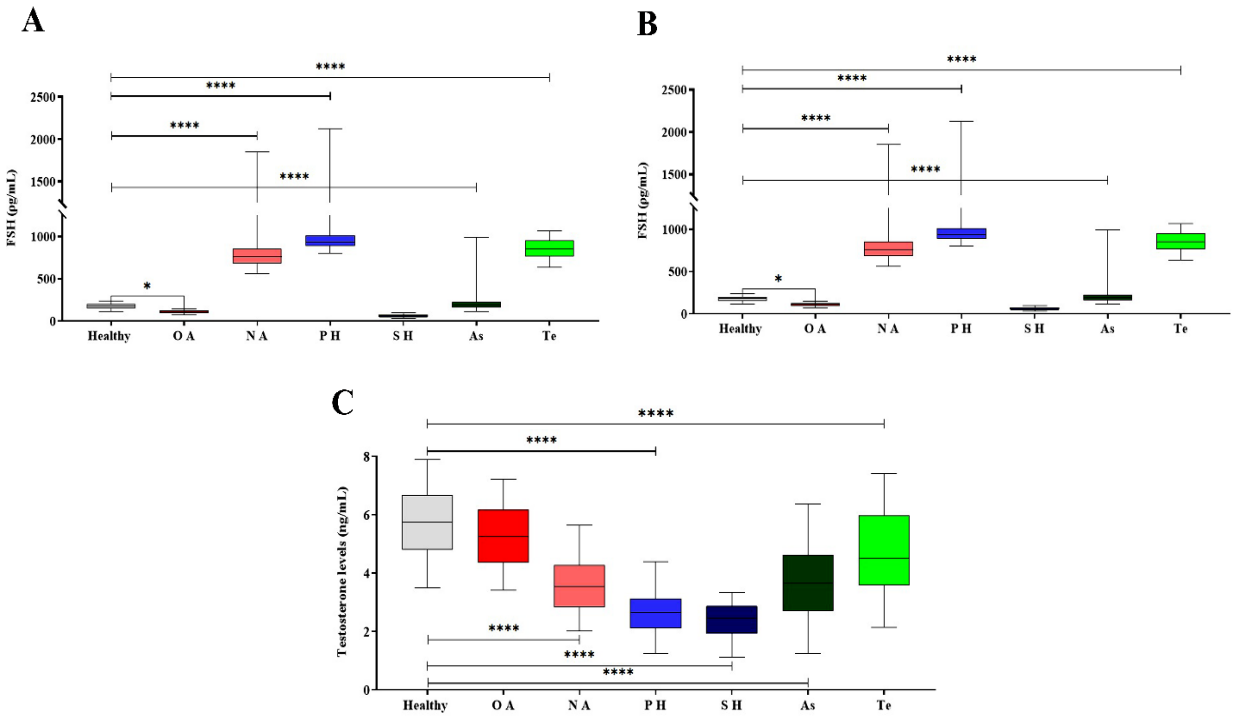

Figure 1. (A) Box plot analysis of serum FSH levels (pg/mL), **(B)** serum LH levels (pg/mL), and **(C)** testosterone levels (ng/mL) across seven clinically defined male groups. Comparison of FSH concentrations among Healthy controls, Obstructive Azoospermia (O A), Non-obstructive Azoospermia (N A), Primary Hypogonadism (P H), Secondary Hypogonadism (S H), Asthenozoospermia (As), and Teratozoospermia (Te) groups. Statistical significance indicators: **** p<0.0001, *** p<0.001, * p<0.05 (**Prepared by Authors, 2025**).

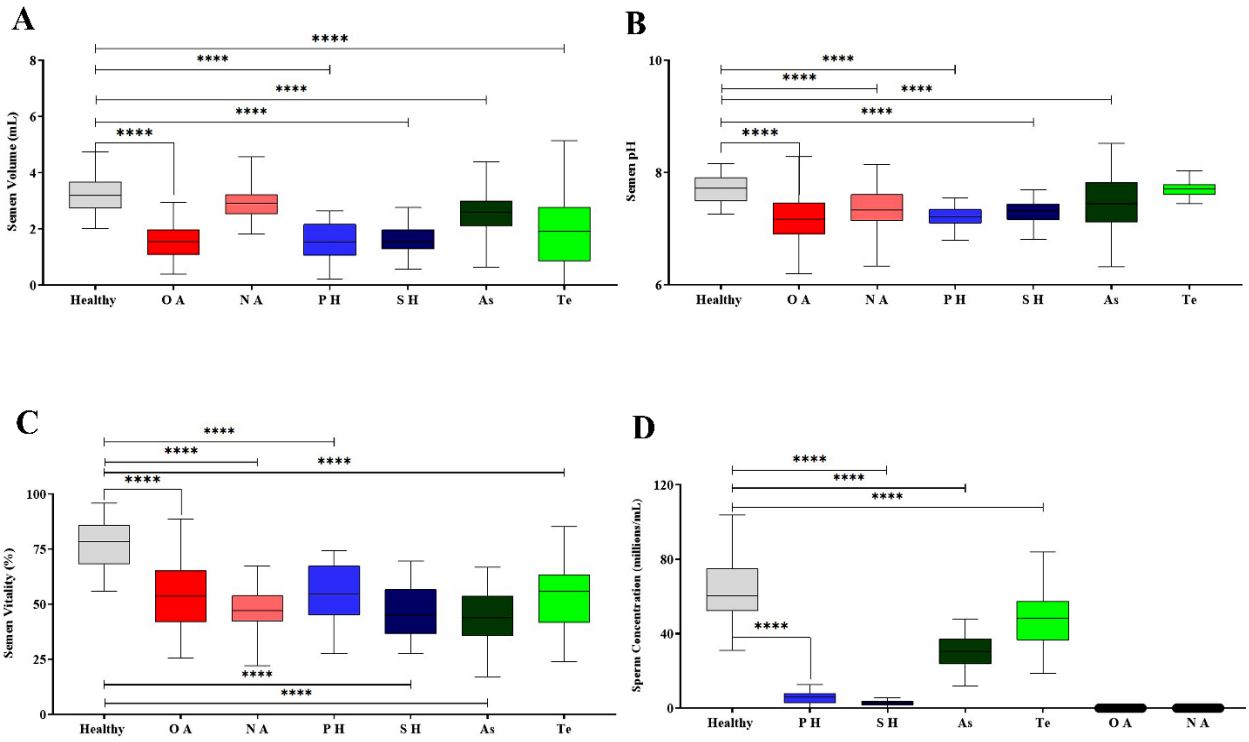

Figure 2. (A) Box plot analysis of serum semen volume (mL), **(B)** serum semen pH, **(C)** serum semen vitality (%), and **(D)** serum sperm concentration (millions/mL) across seven clinically defined male groups. Comparison of semen volume among Healthy controls, Obstructive Azoospermia (OA), Non-obstructive Azoospermia (NA), Primary Hypogonadism (PH), Secondary Hypogonadism (SH), Asthenozoospermia (As), and Teratozoospermia (Te) groups. Statistical significance indicators: **** p<0.000 (**Prepared by Authors, 2025**).

4. Discussions

By simultaneously collecting and analyzing a comprehensive dataset (FSH, LH, testosterone, semen volume, pH, vitality, and concentration) from well-defined case-control groups of Iraqi men, this study aims to establish new clinical benchmarks and reference standards that will improve differential diagnosis, prognosis, and treatment guidance in the region and internationally. This aims to close a critical gap left by previous studies that analyzed hormonal and seminal parameters separately, which makes this research extremely significant.

FSH is a critical regulator of spermatogenesis and serves as the most effective single endocrine marker for distinguishing between different types of male infertility. Elevated FSH levels, such as those observed in Non-Obstructive Azoospermia (754±120 pg/mL) and Primary Hypogonadism (926 ±86 pg/mL) compared to the normal reference (180±35 pg/mL), directly reflect intrinsic testicular damage (i.e., Sertoli cell dysfunction and germ cell loss), which leads to reduced Inhibin B production and subsequent hypergonadotropic signaling **(19, 20)**. In direct contrast, FSH levels in the Obstructive Azoospermia group remain normal or slightly lower (110.81±25.4 pg/mL), strongly confirming that spermatogenesis remains intact and the azoospermia is caused by a post-testicular blockage **(21)**. Our findings successfully validate FSH's diagnostic power, confirming its utility in clarifying hypogonadism diagnoses and identifying poor testicular tissue health associated with lower sperm concentration, motility, and morphology in other subfertile groups **(22)**. Crucially, the significant differences in FSH cutoff values derived from this study provide a robust clinical benchmark for stratifying risk and better informing patients about their prognosis, particularly regarding the lower success rates expected for sperm retrieval in cases with markedly elevated FSH levels **(23)**.

Luteinizing Hormone (LH) is the primary gonadotropin acting on Leydig cells to stimulate testosterone (T) synthesis, making its serum concentration a critical indicator of hypothalamic-pituitary-gonadal (HPG) axis function **(24)**. This hormonal pattern is key to the differential diagnosis of male infertility. Specifically, LH levels are significantly elevated in Non-obstructive Azoospermia and Primary Hypogonadism because the pituitary attempts to compensate for severe primary testicular failure by over-secreting LH **(25, 26)**. Conversely, LH levels remain normal or comparable to those of healthy controls in Obstructive Azoospermia, since Leydig cell function and T production are preserved **(27)**. Furthermore, Secondary Hypogonadism is defined by low or inappropriately normal LH levels alongside low T, reflecting a central pituitary defect **(28)**. Lastly, for isolated sperm defects such as Asthenozoospermia and Teratozoospermia, LH levels are generally not significantly altered, as T production is maintained, suggesting the etiology involves post-testicular factors or localized spermiogenesis defects rather than severe endocrine failure **(29, 30)**.

Testosterone, synthesized by Leydig cells under LH stimulation, is fundamental for spermatogenesis and serves as a crucial diagnostic marker when interpreted alongside gonadotropin levels **(31)**. Testosterone levels generally remain within the normal range in Obstructive Azoospermia, reflecting conserved Leydig cell function and an intact hypothalamic-pituitary-gonadal (HPG) axis **(32, 33)**. Conversely, testosterone is often significantly low in primary gonadal failure states, such as Primary Hypogonadism (invariably low testosterone with compensatory high gonadotropins) **(34)** and a substantial portion of Non-obstructive Azoospermia cases (low testosterone associated with high FSH/LH) **(35)**. The opposite pattern, Secondary Hypogonadism, is defined by low testosterone coupled with inappropriately low or normal LH/FSH, signaling a central pituitary or hypothalamic defect **(36)**. Importantly, in isolated sperm motility (Asthenozoospermia) or morphology defects (Teratozoospermia), testosterone levels are typically maintained and statistically comparable to normozoospermic men **(37, 38)**. This preservation suggests that the primary etiology is often post-testicular or a subtle defect in spermiogenesis, rather than a primary Leydig cell dysfunction. Therefore, testosterone analysis is indispensable for distinguishing primary from secondary hypogonadism and for characterizing azoospermic and isolated sperm defects **(39, 40)**.

The systematic evaluation of fundamental semen parameters is crucial for resolving diagnostic ambiguity between different causes of male infertility. Specifically, Obstructive Azoospermia (OA) is characterized by an absence of sperm while semen volume and pH remain normal, indicating preserved accessory gland function and confirming a post-testicular obstruction **(41, 42)**. In contrast, Non-obstructive Azoospermia (NA) presents with absent sperm, often with a normal or mildly diminished volume, and a potentially alkaline pH, and laboratory analysis is essential to distinguish it from obstructive causes. Semen analysis combined with hormone profiling is also vital for classifying hypogonadism: Primary Hypogonadism often shows a decreased seminal volume due to secondary androgen deficiency, while Secondary Hypogonadism is distinguished by its inappropriately low gonadotropin levels, which directs appropriate hormonal therapy **(43)**. For isolated sperm defects, such as Asthenozoospermia and Teratozoospermia, preservation of semen volume, pH, and sperm concentration suggests that the primary issue is motility or morphology, respectively, with only minor reductions in vitality in severe cases **(44)**. Ultimately, consensus guidelines (WHO/EAU) support the use of these macroscopic and microscopic parameters to differentiate between OA and NOA, and to subclassify the source of hypogonadism, thereby improving clinical management **(45, 46)**.

5. Conclusion

This work offers a comprehensive, evidence-based framework for hormonal evaluation of male infertility, significantly advancing understanding of reproductive endocrinology worldwide. An integrated approach to hormonal and seminal parameter assessment gives clinicians effective tools for accurate diagnosis, prognosis, and treatment planning. These findings will lead the way toward personalized, hormone-guided therapies that can ultimately improve fertility outcomes for couples seeking reproductive assistance. This work makes significant progress in andrology and establishes a new approach to a comprehensive male infertility workup in Middle Eastern populations, while providing insights that medical practitioners and researchers worldwide in reproductive medicine may find valuable.

6. Declarations

6.1 Acknowledgments

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6.2 Ethical Considerations

Consent for publication was obtained from all participants included in this study. The research protocol, including consent procedures, was reviewed and approved by the Ethical Approval Committee of the College of Pharmacy, University of Misan (Approval No. EA_318). All participants were informed about the purpose of the study and agreed that their anonymized data could be published in scientific journals.

6.3 Authors' Contributions

Publisher's Note: In accordance with the journal's publication policies, authors are required to provide information for this section. As no statement was provided, no information on the authors' contributions is available for this article.

6.4 Conflict of Interest

The authors report no conflict of interest.

6.5 Fund or Financial Support

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

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