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Comparing Blood Serum and Seminal Plasma Stem Cell Factor Concentrations in Non-obstructive Azoospermic and Oligospermic Individuals

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Abstract

Objectives: Stem cell factor (SCF) is a crucial cytokine growth factor that significantly contributes to hematopoiesis, as well as the development of melanocytes and germ cells. This study aimed to compare the levels of SCF in serum and seminal plasma among patients with non-obstructive azoospermia (NOA), oligozoospermia, and men with normozoospermic controls, and to determine whether it could serve as a potential biomarker for the local testicular biomarker.

Materials and Methods: This cross-sectional comparative study enrolled 132 patients who were divided into three equal groups: normozoospermia, NOA, and oligozoospermia (n = 44 each). A comparison was made in all groups based on age, hormones, and SCF level. SCF level in serum and seminal fluid was measured by ELISA.

Results: Our research indicated the concentrations of SCF in both seminal plasma and serum across three groups. Seminal plasma SCF levels showed a significant difference between groups ($P = 0.002$), with higher levels observed in the oligozoospermia group compared with both NOA and normozoospermia groups; no significant difference was observed between NOA and normozoospermia. In contrast, serum ligand levels were significantly elevated in the NOA and normospermia groups compared to the oligospermia group ($P < 0.001$). Units are expressed as ng/mL.

Conclusions: Our findings identified that seminal plasma SCF, in contrast to its serum, was significantly elevated in oligozoospermia and may serve as a promising and direct biomarker of the active local testicular microenvironment in states of diminished sperm production.

Keywords: SCF, Kit ligand, Male infertility, Normozoospermia, NOA, Oligozoospermia, Paired samples, Sperm concentration

Introduction

Male infertility is a significant health issue, responsible for nearly 50% of all infertility cases among couples (1). It is typically defined as the male's inability to cause pregnancy in a fertile female partner after one year of unprotected intercourse (1). Diagnosis of male infertility usually relies on an analysis of standard semen parameters following WHO guidelines (1). Azoospermia, characterized by the absence of sperm in the ejaculate, affects approximately 1% of men and about 10% to 15% within the infertile demographic (2). This condition can be categorized into obstructive azoospermia (OA) or non-obstructive azoospermia (NOA), depending on whether there is an obstruction in the ducts or vas deferens (2).

Oligozoospermia is a pathological condition characterized by reduced sperm concentration, which exerts a significant impact on male fertility. It is recognized as one of the most prevalent etiological factors contributing to male infertility, with an estimated incidence of approximately 15% in the general population (3). Various factors influencing male fertility include steroid hormone imbalances, hypogonadism, genetic abnormalities, dysfunctions in spermatogenesis, ejaculation issues, and reproductive infections (3).

The stem cell factor (SCF), also known as kit ligand (KL), steel factor, or mast cell growth factor, is identified as a cytokine growth factor capable of activating multiple signaling pathways through its interaction with the c-kit receptor. This interaction plays a crucial role in regulating diverse processes such as cell proliferation, differentiation, migration, and apoptosis across various cells, including melanocytes, hematopoietic stem cells, gastrointestinal Cajal interstitial cells, mast cells, and reproductive cells respectively (4). The SCF/c-KIT system has been detected across several tissues within both males' and females' reproductive systems, specifically testis, prostate, breast, ovary & endometrium—and it holds vital significance concerning cellular survival regulation alongside other functions like proliferation, migration and differentiation (4).

Patients and Methods

This research was carried out at Al-Nahrain University, High Institute for Infertility Diagnosis and Assisted

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Key Messages

- SCF levels in seminal plasma differed significantly among normozoospermic, oligozoospermic, and non-obstructive azoospermic men, with higher levels observed in oligozoospermia.
- SCF levels in serum showed an opposite pattern, being elevated in normozoospermia and NOA compared with oligozoospermia.
- SCF levels in seminal plasma may serve as a more reliable biomarker of the local testicular microenvironment and impaired spermatogenesis.

Reproductive Technologies, Baghdad, Iraq, from May 2025 to October 2025. This cross-sectional comparative study included 132 patients, who were allocated into three distinct groups: 44 individuals with normozoospermia, 44 diagnosed with NOA based on clinical examination, testicular ultrasound, and hormonal profiling (serum follicle-stimulating hormone [FSH] and testosterone), and 44 with oligozoospermia. Each participant underwent comprehensive evaluations that included semen analysis as well as hormonal assessments for FSH, prolactin, and testosterone levels. Exclusion criteria were applied rigorously; individuals aged over 55 years old, those who had undergone previous surgeries or presented congenital anomalies, and patients suffering from uncontrolled systemic conditions such as diabetes mellitus or endocrine disorders were not considered eligible for this study. Additionally, cases of aspermia were excluded from the study. All selected participants received a thorough fertility assessment consistent with standard practices at the fertility center. This evaluation encompassed detailed medical history collection, physical examinations, semen analyses, and an assessment of male reproductive system functionality. The sample size was comparable to that of previous studies investigating similar biomarkers in male infertility, providing a reasonable basis for exploratory analysis, although no formal a priori power calculation was performed.

Blood Collection and Semen Sample Processing

Blood samples were obtained by a laboratory technologist utilizing a gel tube, which was subsequently centrifuged at 4000 RPM for 5 minutes to separate the serum. The isolated serum was then transferred into an Eppendorf tube and stored at -20 °C until the assay, ensuring that sample integrity is maintained. For semen collection, each patient was provided with a private and hygienic room where specimens were collected through masturbation following a period of sexual abstinence lasting between two and seven days. The samples were immediately handed over to the andrologist for evaluation. Upon collection, the semen was incubated at 37 °C before undergoing examination according to the WHO 2021 guidelines. This assessment aimed to confirm diagnoses such as normozoospermia (sperm count ≥15 million/mL), NOA (absence of sperm in ejaculate), or oligozoospermia (sperm count <15 million/mL). Additionally, relevant parameters, including volume, motility, morphology, and concentration, were measured utilizing a Makler chamber. Following this evaluation process, the seminal fluid underwent centrifugation at 1500 RPM for 15 minutes in order to separate the seminal plasma from cellular components. To maintain sample integrity during analysis, the resulting seminal plasma was immediately preserved at -20 °C. Storage duration did not exceed three months.

Measurement of SCF Levels in Serum and Seminal Fluid

Serum and seminal fluid were analyzed for SCF levels utilizing an ELISA Kit (Human Kit LG ELISA Kit®, Fine Test, China; Catalog No: EH0514), which enables precise quantification of SCF protein concentration. The assay was performed according to the manufacturer's instructions. The detection range of the assay was 31.25–2000 pg/mL, with a sensitivity of 18.75 pg/mL. The intra-assay and inter-assay coefficients of variation were <5% and <6%, respectively, indicating high assay precision.

Statistical Analysis

Data were collected, summarized, analyzed, and presented using Statistical Package for Social Sciences (SPSS) version 23 and Microsoft Office Excel 2010. Qualitative (categorical) variables were expressed as number and percentage, whereas, quantitative (numeric) variables were first evaluated for normality distribution using Kolmogorov-Smirnov test, and then accordingly normally distributed numeric variables were expressed as mean (an index of central tendency) and standard deviation (an index of dispersion), while those numeric variables that are not normally distributed were expressed as median (an index of central tendency) and inter-quartile range (an index of dispersion).

The following statistical tests were used: One-way ANOVA test was used to evaluate the difference in mean of numeric variables among groups, provided that these variables were normally distributed, which was followed by post hoc LSD test; otherwise, Kruskal-Wallis test would be used instead if those variables were not normally distributed, followed by post hoc Dunn's test. Post hoc comparisons were performed using LSD for ANOVA and Dunn's test for Kruskal–Wallis analysis. No additional correction for multiple comparisons was applied unless otherwise stated. Correlation analyses, where performed, were considered exploratory in nature and interpreted accordingly. The level of significance was considered at a *P* value of equal to or less than 0.05.

Results

Demographic Characteristics

In this study, age was the sole demographic variable recorded, and the comparison of mean age across groups

is presented in Table 1 and Figure 1. The mean age of patients enrolled was 31.75 ± 6.85 years, 32.59 ± 6.79 years and 32.89 ±4.88 years in the NOA group, oligozoospermia group and normozoospermia group, respectively. The age ranges were as follows: 20-55 years, 20-50 years and 21-43 years, respectively. Statistical comparison revealed no significant variation in mean age among the three groups enrolled in this study ($P = 0.676$).

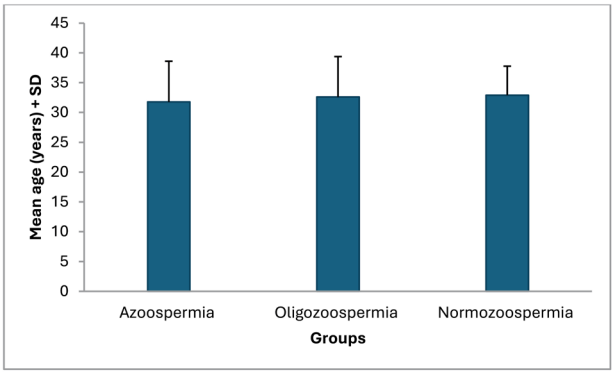

Figure 1. Bar Chart Showing Comparison of Mean Age Among Study Groups

Serum Hormonal Levels

Serum hormonal levels were compared among the study groups, and the results are presented in Table 2. The existence of a wide range and the relatively high values of standard deviations of serum FSH, prolactin and Testosterone and the significant p-value following performance of normality test, made the use of median and inter-quartile range as an obligatory condition to represent such data in the current project. As shown in Table 2 and Figure 2, the median (IQR) level and the range of serum FSH were as follows: 10.32, 5.59 and 5.83 IU/L and 0.86 -51.62, 0.72 -23.5 and 0.86 -9.45 IU/L in the NOA group, oligozoospermia group and normozoospermia group, respectively. Statistical comparison revealed significant variations among study groups ($P<0.001$). Post hoc analysis using Dunn's test revealed no significant difference in level between the oligozoospermia group and the normozoospermia group, but the level in the NOA group was significantly higher than those of the oligozoospermia group and the normozoospermia group.

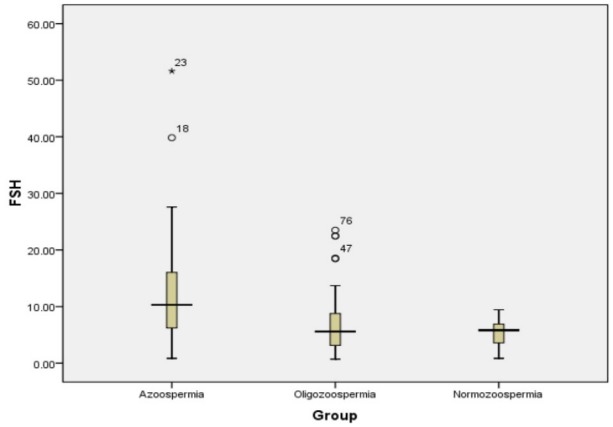

Figure 2. Box Plot Showing Comparison of serum FSH Level Among Study Groups

As shown in Table 2 and Figure 3, the median level

Table 1. Demographic Characteristics of Patients

Characteristic	Non-obstructive azoospermia (n = 44)	Oligozoospermia (n = 44)	Normozoospermia (n = 44)	*P*
Age				
Mean ± SD	31.75 ±6.85	32.59 ±6.79	32.89 ±4.88	0.676
Range	20 -55	20 -50	21 -43	

SD: standard deviation.

Table 2. Serum Hormonal Levels Compared Among Study Groups

Characteristic	Non-obstructive azoospermia (n = 44)	Oligozoospermia (n = 44)	Normozoospermia (n = 44)	*P*
FSH (IU/L)				
Median (IQR)	10.32 (10.21)	5.59 (5.62)	5.83 (3.52)	<0.001*
Range	0.86-51.62	0.72-23.5	0.86-9.45	
Prolactin (ng/mL)				
Median (IQR)	11.91 (6.33)	14.04 (7.88)	10.11 (7.7)	0.003*
Range	1-34.84	1.62-66.49	2.88-22.61	
Testosterone (ng/dL)				
Median (IQR)	3.07 (1.73)	4.37 (2.63)	3.38 (1.59)	0.018*
Range	0.45-7.11	1.71-8.51	1.22-7.15	

IQR: inter-quartile range. * Significant.

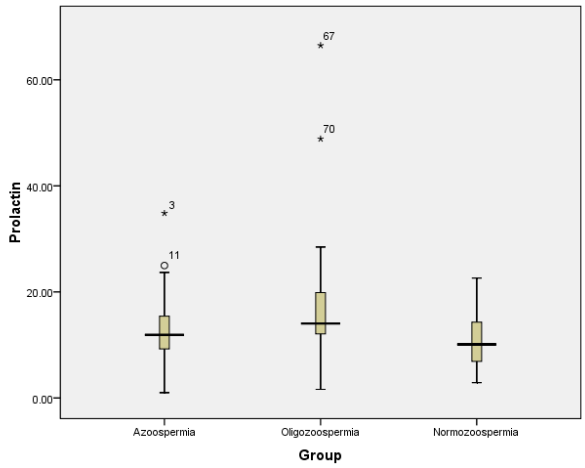

Figure 3. Box Plot Showing Comparison of Serum Prolactin Level Among Study Groups.

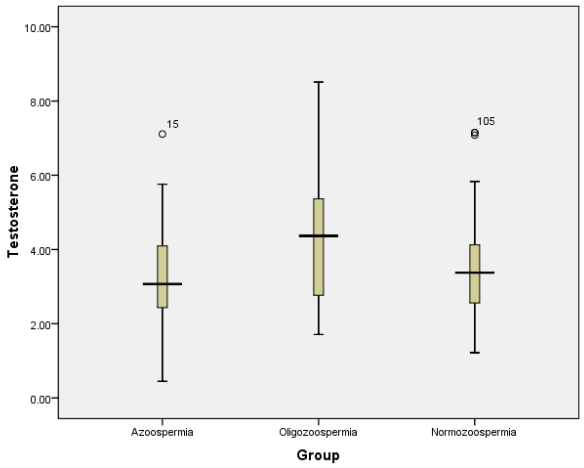

Figure 4. Box Plot Showing Comparison of Serum Testosterone Level Among Study Groups.

and the range of serum prolactin were as follows: 11.91, 14.04 and 10.11 ng/mL and 1 -34.84, 1.62 -66.49 and 2.88 -22.61 ng/mL in the NOA group, oligozoospermia group and normozoospermia group, respectively. Statistical comparison revealed significant variations among study groups ($P=0.003$). Post hoc analysis using Dunn's test revealed significant differences in level among groups in such a way that heights level was seen in the oligozoospermia group, followed by the NOA group and lastly by the normozoospermia group.

As shown in Table 2 and Figure 4, the median level and the range of serum testosterone were as follows: 3.07, 4.37 and 3.38 ng/dl and 0.45-7.11, 1.71 -8.51 and 1.22 -7.15 ng/dL in the NOA group, oligozoospermia group and normozoospermia group, respectively. Statistical comparison revealed significant variations among study groups ($P<0.018$). Post hoc analysis using Dunn's test revealed significant differences in level among groups in such a way that heights level was seen in the oligozoospermia group, followed by the normozoospermia group and lastly by the NOA group.

Comparison of Serum and Seminal Plasma Level of SCF Among Study Groups

As seen in Table 3 and Figure 5, the median level and the range of seminal plasma SCF were as follows: 0.12, 0.55 and 0.13 and 0.06 -0.15 pg/mL, 0.02 -1.78 and 0.1 -0.15 pg/mL in the NOA group, oligozoospermia group and normozoospermia group, respectively. Statistical comparison revealed significant variations among study groups ($P=0.002$). Post hoc analysis using Dunn's test revealed no significant difference in level between the NOA group and normozoospermia group, but the level in the oligozoospermia group was significantly higher than

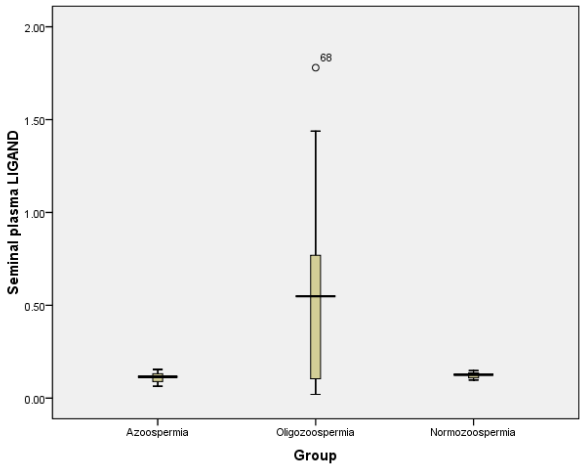

Figure 5. Box Plot Showing Comparison of Seminal Plasma SCF Level Among Study Groups.

Table 3. Serum and Seminal Plasma Level of SCF Among Study Groups

Characteristic	Non-obstructive azoospermia (n = 44)	Oligozoospermia (n = 44)	Normozoospermia (n = 44)	*P*
SCF (pg/mL) in seminal plasma				
Median (IQR)	0.12 (0.04)	0.55 (0.67)	0.13 (0.03)	0.002*
Range	0.06 -0.15	0.02 -1.78	0.1 -0.15	
SCF (pg/mL) in serum				
Median (IQR)	0.53 (0.23)	0.13 (0.43)	0.55 (0.28)	<0.001*
Range	0.29 -0.91	0.07 -0.95	0.33 -1.44	

SCF: stem cell factor; IQR: inter-quartile range. * Significant.

that of the NOA group and normozoospermia group.

As revealed in Table 3 and Figure 6, the median level and the range of serum SCF were as follows: 0.53, 0.13 and 0.55 pg/mL and 0.29 -0.91, 0.07 -0.95 and 0.33 -1.44 pg/mL in the NOA group, oligozoospermia group and normozoospermia group, respectively. Statistical comparison revealed significant variations among study groups ($P<0.001$). Post hoc analysis using Dunn's test revealed no significant difference in level between the NOA group and normozoospermia group, but the level in the oligozoospermia group was significantly lower than those of the NOA group and normozoospermia group.

Correlation Study

In the NOA group, no significant correlation was observed ($P>0.05$), as shown in Table 4. In the oligozoospermia group, significant positive correlations were observed between serum FSH levels and seminal plasma SCF concentrations ($P<0.05$) and significant negative correlation of FSH to serum plasma SCF ($P<0.05$), as explained in Table 5. In the normozoospermia group, a significant positive correlation was observed between serum SCF levels and age ($P<0.05$), as shown in Table 6.

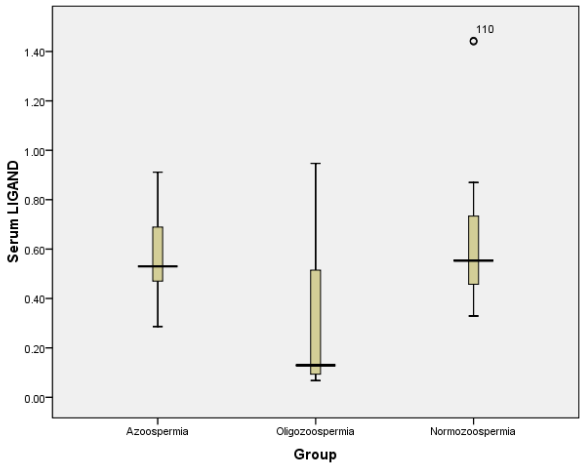

Figure 6. Box Plot Showing Comparison of Serum SCF Level Among Study Groups.

Discussion

In recent years, the concentration of SCF in women with infertility has attracted considerable attention due to its potential influence on pregnancy outcomes following assisted reproductive techniques. Despite this growing interest, conflicting and at times contentious findings have been reported, underscoring the need for further investigation in this area. For instance, Tan et al demonstrated that levels of SCF present in follicular fluid and granulosa cells were positively correlated with oocyte

Table 4. Correlations of Markers Levels to Age and Hormonal Levels in the NOA Group

Characteristic	Index	Age	FSH	Prolactin	Testosterone
Seminal plasma SCF (pg/mL)	*r*	0.232	-0.119	-0.257	-0.279
	P	0.129	0.440	0.092	0.067
Serum SCF (pg/mL)	*r*	-0.095	-0.119	0.168	0.057
	P	0.538	0.441	0.276	0.715

Table 5. Correlations of Markers Levels to Age and Hormonal Levels in the Oligozoospermia Group

Characteristic	Index	Age	FSH	Prolactin	Testosterone
Seminal plasma SCF (pg/mL)	*r*	0.130	0.300	0.169	-0.052
	P	0.401	0.048*	0.273	0.736
Serum SCF (pg/mL)	*r*	-0.040	-0.350	-0.228	-0.117
	P	0.797	0.020*	0.136	0.448

Table 6. Correlations of Markers Levels to Age and Hormonal Levels in the Normozoospermia Group

Characteristic	Index	Age	FSH	Prolactin	Testosterone
Seminal plasma SCF (pg/mL)	*r*	0.242	0.067	0.026	-0.055
	P	0.114	0.666	0.868	0.723
Serum SCF (pg/mL)	*r*	0.305	-0.086	0.000	-0.119
	P	0.044*	0.577	0.998	0.443

maturation, successful fertilization processes, embryo quality, and overall clinical pregnancy outcomes (5). Conversely, a study conducted by Firas et al (4) reported that SCF concentrations in follicular fluid from patients who did not achieve pregnancy were notably higher than those observed in individuals who successfully conceived. Additionally, other research indicated an absence of correlation between serum SCF levels measured on oocyte retrieval day and both cycle characteristics as well as resulting outcomes.

In the 2023 research conducted by Peng et al, various mouse models were utilized to investigate growth factor expression patterns in testicular somatic cells through single-cell RNA sequencing (scRNA-seq) (6). The study revealed that SCF, a crucial growth factor, exhibited a significantly wider expression profile within the testes than previously understood. This included its presence in Sertoli cells, endothelial cells, Leydig cells, smooth muscle cells, and Tcf21 stromal cells. The findings regarding SCF's distribution in the testis were validated using an SCF-GFP reporter system.

Peng et al employed genetic techniques to conditionally knock out or overexpress SCF specifically within SCF-expressing cell types. Results indicated that Sertoli cells serve as the primary physiological source of SCF necessary for spermatogenesis. Furthermore, scRNA-seq was applied to delve into the cellular and molecular mechanisms governing how SCF influences spermatogenic processes; it was discovered that Sertoli-derived SCF performs dual functions: sustaining differentiating spermatogonia while also facilitating their maturation (6). Additionally, another investigation assessed levels of SCF present in seminal plasma via reverse transcriptase polymerase chain reaction (RT-PCR) and found correlations suggesting that these levels could be indicative of sperm production capacity. Therefore, this particular growth factor is posited to play a significant role in spermatogenesis (7). In contrast to our finding of elevated seminal SCF levels in oligospermic, this discrepancy may be explained by differences in the study populations and underlying pathophysiology. Fujisawa et al included groups of men with a broader range of spermatogenic function, whereas our study specifically focused on oligospermia and NOA. It is plausible that oligospermia triggers a compensatory upregulation of SCF secretion from Sertoli cells or other sources in the male reproductive tract. Alternatively, differences in methodological such as ELISA vs. other detection methods, sample handling, or seminal plasma processing could contribute to the divergent results. More studies using standardized protocols and including patients with varying degrees of spermatogenic failure are needed to clarify these conflicting findings. Other studies have compiled substantial data—primarily from animal models—that highlight the importance of the relationship between the SCF/c-kit signaling pathway and spermatogenesis alongside its potential implications for issues related to fertility defects (8,9).

To our knowledge, this was the first study to compare blood serum and seminal plasma SCF concentrations in azoospermic and oligozoospermic individuals. The principal finding was that seminal plasma SCF levels in the oligozoospermia group were significantly higher than those in the NOA and normozoospermia groups, whereas serum SCF levels in the oligozoospermia group were significantly lower than those in the NOA and normozoospermia groups. Within the oligozoospermia group, significant positive correlations were observed between serum FSH and seminal plasma SCF, alongside significant negative correlations between FSH and serum SCF. In the normozoospermia group, a significant positive correlation was identified between serum SCF and age ($P < 0.05$).

The possible explanations for the current results of high seminal SCF but low serum SCF in oligospermic men may be due to the reduced number of germ cells (which express the c-Kit receptor) in oligozoospermia, which may trigger a feedback loop within the seminiferous tubules. Sertoli cells, the primary producers of SCF in the testis, might upregulate SCF production to stimulate the survival, proliferation, and maturation of the remaining germ cells. The condition causing oligozoospermia (e.g., inflammation, oxidative stress, hormonal imbalance) may compromise the integrity of the blood-testis barrier. This could lead to an increased leakage of SCF from the seminiferous tubule fluid into the seminal plasma, elevating its concentration there, while simultaneously affecting systemic production or clearance. Seminal SCF is primarily of testicular origin (Sertoli cells). Serum SCF comes from multiple sources (bone marrow stromal cells, endothelial cells, etc). The pathology in oligozoospermia may involve a systemic downregulation of SCF production or increased clearance, leading to lower serum levels. An isolated testicular pathology where the testis becomes insensitive to systemic SCF levels, developing its own compensatory local microenvironment. The negative correlation of serum FSH with serum SCF suggests that a complex systemic endocrine feedback loop is altered. The positive correlation between serum SCF and age in fertile men could be related to age-related subclinical inflammation (SCF is involved in mast cell and inflammatory pathways) or subtle, compensatory changes in the stem cell niche microenvironment with advancing age, even within a normal fertile range.

The strong correlations with FSH in the oligospermic group are pivotal. They directly tie the dysregulation of the SCF/c-Kit pathway to the well-established axis of spermatogenic failure. It positions SCF as a potential local mediator of FSH's effects on germ cell support. The negative correlation with serum SCF further suggests that the endocrine feedback governing this system is fundamentally altered in oligozoospermia. In NOA, the seminal SCF level was not elevated. This could indicate

a "burnt-out" or failed compensatory mechanism. If the primary defect is the absence of germ cell precursors (as in Sertoli-cell-only syndrome), the stimulus for SCF overproduction may be absent. Alternatively, in NOA, the SCF level might be normal, as spermatogenesis could be intact behind the blockage. In oligozoospermia, the body is still attempting to salvage spermatogenesis, leading to the observed active but dysregulated compensatory response (high local SCF).

Limitations of the Study

This study had several limitations. It was conducted at a single center with a relatively small sample size, which may have restricted the generalizability of the findings. In addition, the etiological heterogeneity within the NOA and oligozoospermia groups was not fully controlled, potentially contributing to variability in SCF levels. Furthermore, functional assessment of the SCF/c-Kit signaling pathway was not performed, thereby limiting mechanistic interpretation. Finally, the cross-sectional design without longitudinal follow-up precluded evaluation of temporal changes in SCF levels or their predictive value over time.

Conclusions

In summary, this study uncovered a unique and seemingly contradictory dysregulation of the SCF system in men with oligozoospermia. Notably, seminal plasma SCF levels were elevated, whereas serum concentrations were reduced. This compartmentalized phenomenon suggested a local compensatory mechanism within the testes; Sertoli cells appeared to have enhanced SCF production—potentially stimulated by increased FSH—in an ineffective attempt to salvage impaired spermatogenesis. The divergent observations noted in cases of NOA indicated alternative underlying pathophysiological processes or possibly an exhaustion of this adaptive response. Consequently, these results highlighted seminal plasma SCF as a potential direct biomarker reflecting the active microenvironment within the testes during periods of reduced sperm output, rather than relying on its serum counterpart. Furthermore, the substantial correlation with FSH intricately linked the SCF/c-Kit pathway to fundamental endocrine dysfunctions associated with male infertility. This study opened new avenues for exploring paracrine interactions involved in sperm development and may inform innovative therapeutic approaches aimed at manipulating this essential SCF-receptor system for the treatment of oligozoospermia.

Authors' Contribution

Conceptualization: Ghada Firas Faisal.
Data curation: Russel Taha Yaseen.
Formal analysis: Tuqa M. Abdul-Saheb.
Funding acquisition: All authors.
Investigation: Ghada Firas Faisal and Russel Taha Yaseen.
Methodology: Ghada Firas Faisal and Russel Taha Yaseen.
Project administration: Ghada Firas Faisal and Russel Taha Yaseen.
Resources: Ghada Firas Faisal, Russel Taha Yaseen and Tuqa M. Abdul-Saheb.
Software: Tuqa M. Abdul-Saheb and Shams Anmar Burhan.
Supervision: Ula M. Alkawaz.
Validation: Ula M. Alkawaz.
Writing–original draft: Ghada Firas Faisal.
Writing–review & editing: Ghada Firas Faisal and Russel Taha Yaseen.

Conflict of Interests

Authors declare that they have no conflict of interests.

Ethical Issues

The institution's reviewing board at High Institute for Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University, Iraq surveyed the research and licensed the latest installment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. A local ethical board reviewed the research protocol, topic data, and consent paperwork and gave its stamp of approval on May 4, 2025, with code:0701-Mf-2026G73.

Data Availability Statement

Data corroborating the outcomes of this study can be acquired from the corresponding author on a reasonable request.

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References

1. Mannucci A, Argento FR, Fini E, et al. The Impact of Oxidative Stress in Male Infertility. Front Mol Biosci. 2022;8:799294. doi:10.3389/fmolb.2021.799294
2. Sharma M, Leslie S. Non-obstructive azoospermia. StatPearls; 2023.
3. Ohanu EC, Lawson SD, Esiere RK, Onosakponome EO. Bacteriospermia: An Etiology of Oligospermia. Journal of Applied Health Sciences and Medicine. 2023;3(4):14-21.
4. Faisal GF, Al-kawaz U, Al-Anbari LA, Hussaini HAR, editors. Evaluation of the Level of Stem Cell Factor in Follicular Fluid and its Effect on Oocyte Maturity, Embryo Quality and Pregnancy Rate. Iraqi Journal of Embryos and Infertility Researches. 2020;9(2):65-77.
5. Tan J, Zou Y, Wu XW, et al. Increased SCF in Follicular Fluid and Granulosa Cells Positively Correlates With Oocyte Maturation, Fertilization, and Embryo Quality in Humans. Reprod Sci. 2017;24(11):1544-1550. doi:10.1177/1933719117697125
6. Peng YJ, Tang XT, Shu HS, Dong W, Shao H, Zhou BO. Sertoli cells are the source of stem cell factor for spermatogenesis. Development. 2023;150(6):dev200706. doi:10.1242/dev.200706
7. Fujisawa M, Kanzaki M, Okuda Y, Okada H, Arakawa S, Kamidono S. Stem cell factor in human seminal plasma as a marker for spermatogenesis. Urology. 1998;51(3):460-463. doi:10.1016/s0090-4295(97)00627-4
8. Loveland KL, Schlatt S. Stem cell factor and c-kit in the mammalian testis: lessons originating from Mother Nature's gene knockouts. J Endocrinol. 1997;153(3):337-344. doi:10.1677/joe.0.1530337
9. Mauduit C, Hamamah S, Benahmed M. Stem cell factor/c-kit system in spermatogenesis. Hum Reprod Update. 1999;5(5):535-545. doi:10.1093/humupd/5.5.535

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