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# Effectiveness of Empirical Hormonal Stimulation in the Treatment of Idiopathic Oligoasthenoteratospermia: A Clinical Evaluation

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## ABSTRACT

**Background:** Instead of distinct forms, DNA fragmentation and chromatin structural abnormalities are connected to most occurrences of idiopathic oligoasthenoteratospermia (OAT). Hormonal treatment dosage and duration varies amongst proposed strategies. Testicular testosterone (TT), which is transformed into dihydrotestosterone (DHT), and gonadotropins are stimulated by gonadotropin-releasing hormone (GnRH) pulses. DHT raises the concentration of TT for spermatogenesis via promoting the synthesis of androgen-binding protein (ABP) in Sertoli cells.

**Aim**: to assess how well sperm parameters (such as count, motility, and morphology) are improved by empirical hormonal stimulation.

**Subjects And Methods**: The study included a sample of 15 male patients diagnosed with idiopathic oligoasthenoteratospermia. These patients were selected conveniently from those attending the infertility clinic at AL-Yarmouk –Teaching Hospital.

**Results:** Sperm concentration increased slightly from 5.334 ± 1.794 × 106/ml before treatment to 6.067 ± 1.885 × 106/ml after treatment, and the percentage of normal sperm morphology increased from 0.933% ± 1.1 before treatment to 3.6% ± 8.781 after treatment (P = 0.002). Prior to therapy, the total sperm count was 13.794 ± 3.374 × 106/ml; following treatment, it increased to 17.156 ± 9.225 × 106/ml (P = 0.042).

**Conclusion:** The percentage of normal sperm morphology increased significantly after hormonal treatment (P = 0.002). Sperm quality improved as evidenced by a significant drop in aberrant sperm shapes (P = 0.049).

**Keywords:** *Empirical Hormonal,infertility,motility,'idiopathic oligospermia,sperm morphology*.

## 1. INTRODUCTION

Infertility is defined as the failure to conceive after one year of unprotected sexual intercourse, impacting around 10% to 15% of couples globally. This problem might be profoundly stressful and affects numerous folks attempting to conceive. Infertility can stem from both male and female partners, with male infertility being a prominent factor in roughly 40% of instances. Sperm count and quality are essential factors in the fertilization process. A normal sperm count is characterized by exceeding 15 million sperm per milliliter, but motility, defined as the effective swimming capability of sperm, should surpass 40%. Additionally, sperm morphology, denoting the shape and structure of sperm, should ideally exceed 4% to indicate a favorable chance for conception (1) . Oligoasthenoteratospermia (OAT) is a disorder characterized by reduced sperm concentration, diminished motility, and aberrant morphology. The term 'idiopathic oligospermia' is employed to denote cases of unexplained reduced sperm production, despite comprehensive examination and testing (2). Notwithstanding progress, the diagnosis and treatment of idiopathic oligospermia continue to pose difficulties. Options are available, although their selection differs. Timely intervention may improve results for male infertility. Oligospermia impacts 50% of males

and is associated with 30% of infertility cases, with more than 85% of individuals with OAT exhibiting oligoastenospermia. This highlights the necessity for additional investigation. Examining hormone therapy such as ascorbic acid, tamoxifen, and clomiphene citrate, while addressing existing literature gaps, may facilitate future research endeavors. (3). The male reproductive system generates and transports sperm for conception via several anatomical structures and processes. Essential components comprise the testes, which produce sperm and testosterone; the epididymis, a convoluted duct for sperm maturation and storage; and auxiliary glands that synthesize seminal plasma. The seminal vesicles emit fructose and essential nutrients for sperm vitality, whereas the prostate, encircling the urethra, generates a milky fluid. Furthermore, the bulbourethral glands secrete a transparent mucus to purify the urethra prior to ejaculation. Oligospermia denotes a condition marked by a diminished sperm count, while hypomotility indicates a decrease in sperm motility. Teratospermia refers to the existence of atypical sperm morphology (4). Men diagnosed with idiopathic oligoasthenoteratospermia (OAT) sometimes have reproductive issues due to many contributing causes. Factors may encompass elevated malondialdehyde (MDA) levels, mutations in the p53 and TFR1 genes, and infections induced by the mumps virus, particularly the detection of IgM antibodies. The inflammation resulting from a re-infection with the mumps virus causes an elevation in pro-inflammatory cytokines, including TNF-α and interleukins such as IL-1β, IL-6, and IL-12. This inflammatory reaction markedly deteriorates testicular health and diminishes sperm quality, impacting both motility and normal morphology, which pertains to the appropriate form and structure of sperm. The interaction between mutations in the p53 and TFR1 genes affects inflammatory responses in the body and is essential for preserving good sperm shape(5). Male reproduction is a complex process that necessitates a delicate equilibrium among many organs, hormones, and physiological elements. Current research on hormone therapy, particularly on gonadotropin-releasing hormone (GnRH) and testosterone in people with idiopathic oligospermia, is significantly constrained. Male infertility constitutes around 50% of all infertility cases, with idiopathic reasons accounting for roughly 15-20% of these occurrences (6) Sperm quality is frequently compromised by disorders such as teratozoospermia, oligozoospermia, and asthenozoospermia, which are present in 40-50% of males with reproductive challenges. Idiopathic OAT manifests as a multifaceted medical and psychological dilemma, with its roots remaining ambiguous. It frequently results in heightened psychological stress among impacted individuals, which can raise cortisol levels, adversely influencing sperm morphology, motility, and general focus. Identifying effective therapeutics grounded in a comprehensive understanding of the pathophysiology of OAT is essential to alleviate undesirable outcomes, including increased divorce rates among couples facing infertility (7).

Male idiopathic oligospermia is increasingly acknowledged as a substantial and intricate issue in reproductive health. Despite significant progress in our comprehension of spermogenesis, functioning, and the associated molecular pathways, it remains a substantial barrier in terms of elucidation and successful therapeutic approaches. This highlights the essential necessity of formulating personalized strategies customized to the specific requirements of impacted individuals (8,9)

**Study Population and Sampling Technique**

The research encompassed a cohort of 15 male patients diagnosed with idiopathic oligoasthenoteratospermia. The patients were easily selected from those visiting the infertility clinic at Al-Yarmuke Teaching Hospital.

**Inclusion Criteria**

• Male patients diagnosed with idiopathic oligoasthenoteratospermia

- patients have not undergone any prior hormone therapy.

**Exclusion Criteria**

• Patients with a history of treatment with antibiotics or other medications that may affect sperm quality.

• Patients with any comorbidities that could interfere with the analysis of seminal fluid, such as infections or other reproductive health conditions.

## 2. RESULTS

**Comparison of Seminal Fluid Analysis Parameters Before and After Hormonal Treatment**

Table 1 provides a comparative examination of seminal fluid parameters prior to and during hormonal treatment, emphasizing significant alterations in sperm morphology and concentration. Percentage of Total Normal Form: A notable rise in the percentage of normal sperm morphology from 0.933% ± 1.1 before to treatment to 3.6% ± 8.781 post-treatment (P = 0.002) indicates an enhancement in sperm morphology subsequent to hormone therapy. Total Abnormal Form Percentage: A notable reduction in abnormal sperm morphology is recorded, decreasing from 99.067% ± 1.1 to 96.4% ± 8.781 (P = 0.049), underscoring the beneficial effect of therapy on sperm quality. Sperm Concentration: The sperm concentration increased somewhat from 5.334 ± 1.794 × 10^6/ml prior to treatment to 6.067 ± 1.885 × 10^6/ml post-treatment; yet, this variation was not statistically significant (P = 0.093). This indicates that although therapy may have exerted a slight influence on sperm concentration, the effect was insufficient to be definitive. Total Sperm Count: A substantial rise in total sperm count was seen post-treatment, escalating from 13.794 ± 3.374 × 10^6/ml prior to treatment to 17.156 ± 9.225 × 10^6/ml subsequent

to treatment (P = 0.042). This suggests that hormone therapy may enhance overall sperm production.

**Table 1: Comparison of Seminal Fluid Analysis Parameters Before and After Hormonal Treatment**

| **Morphology** | **seminal fluid analysis (before and after) hormonal treatment** | | **Paired sample t test P value** |
|---|---|---|---|
| | **Before** | **After** | |
| **total normal form %** | 0.933+1.1 | 3.6+8.781 | 0.002** |
| **total abnormal form%** | 99.067+1.1 | 96.4+8.781 | 0.049* |
| **concenteration (*10^6) %** | 5.334+1.794 | 6.067+1.885 | 0.093 |
| **total count (*10^6)** | 13.794+3.374 | 17.156+9.225 | 0.042* |

***significant difference between groups (p value ≤ 0.05)**

****High significant difference between groups (p value ≤ 0.01)**

**effect of hormonal treatment on various sperm morphology**

Parameters, contrasting pre-treatment and post-treatment values. Figure 1 depicts the impact of hormonal therapy on different sperm morphological metrics, contrasting pre-treatment (blue) and post-treatment (orange) results. The statistics demonstrate significant enhancements in sperm quality post-treatment. • Total Normal Form Percentage: The fraction of normal sperm morphology markedly increased from 0.933% prior to treatment to 3.6% following treatment. This indicates a possible improvement in sperm quality resulting from hormone intervention. • Total aberrant Form Percentage: A minor decrease in aberrant sperm morphology is noted, declining from 99.067% prior to therapy to 96.4% subsequent to treatment. This slight reduction signifies a modest enhancement in sperm structural integrity. Sperm Concentration: The sperm concentration exhibited a marginal rise from 5.334 × 10^6/ml prior to treatment to 6.067 × 10^6/ml subsequent to treatment. The minimal degree of change indicates that the medication may have exerted a restricted impact on sperm concentration. Total Sperm Count: Notably, total sperm count rose from 13.794 × 10^6/ml prior to therapy to 17.156 × 10^6/ml subsequent to treatment, indicating a potential improvement in overall sperm production.

**Impact Of Hormonal Treatment On Sperm Motility Parameters,Comparing Values Before And After Treatment.**

Table 2 illustrates the effect of hormone therapy on sperm motility measures, contrasting values prior to and following treatment. The studies demonstrate substantial enhancements in progressive sperm motility post-treatment. Grade A motility (rapid progressive sperm) was initially nonexistent (0% ± 0.0) but exhibited a minor rise post-treatment (0.667% ± 0.582), with a highly significant P-value of 0.0001. Grade B motility (slow progressing sperm) exhibited a notable increase from 1.667% ± 0.637 prior to treatment to 4.667% ± 1.178 subsequent to treatment (P = 0.0116). Grade C motility (non-progressive sperm) shown a substantial improvement (P = 0.0053), increasing from 8.933% ± 2.273 prior to therapy to 27% ± 8.224 subsequent to treatment. Grade D motility (immotile sperm) shown a statistically significant increase (P = 0.0018), rising from 20.2% ± 9.292 prior to therapy to 52.4% ± 12.676 following treatment, signifying a considerable elevation in immotile sperm post-treatment. The data indicate that hormone treatment notably enhances progressive motility (Grades A and B), but is also associated with a substantial rise in immotile sperm (Grade D).

**Table 2: Effect of Hormonal Treatment on Sperm Motility Parameters: Comparison Before and After Treatment**

| **Motility** | **seminal fluid analysis (before and after) hormonal treatment** | | **Paired sample t test P value** |
|---|---|---|---|
| | **Before** | **After** | |
| Grade A % | 0+0 | 0.667+0.582 | 0.0001** |
| Grade B % | 1.667+0.637 | 4.667+1.178 | 0.0116* |

| | | | |
|---|---|---|---|
| Grade C % | 8.933+2.273 | 27+8.224 | 0.0053** |
| Grade D % | 20.2+9.292 | 52.4+12.676 | 0.0018** |

***significant difference between groups (p value ≤ 0.05)**

****High significant difference between groups (p value ≤ 0.01)**

**Changes In Sperm Motility Parameters Before And After Hormonal Treatment**

Figure 2 depicts the alterations in sperm motility metrics pre- and post-hormonal therapy. The data demonstrate significant variations among motility grades, suggesting the treatment's potential influence on sperm motility. • Grade A (rapid Progressive Motility): Prior to therapy, no sperm demonstrated rapid progressive motility (0%), but a marginal increase to 0.667% was noted following treatment. This signifies a slight yet discernible enhancement in the top-tier motility category. Grade B (Slow Progressive Motility): A notable rise was observed in this category, escalating from 1.667% prior to treatment to 4.667% post-treatment, indicating improved sperm motility following hormonal intervention. • Grade C (Non-Progressive Motility): A significant rise in this category was noted, rising from 8.933% prior to treatment to 27% post-treatment, showing a change in sperm motility patterns, with a greater proportion of sperm demonstrating motility without forward progression. Grade D (Immotile Sperm): A significant escalation in immotile sperm was observed, increasing from 20.2% prior to treatment to 52.4% post-treatment, indicating a possible adverse effect of the treatment on overall sperm viability. Figure 2 indicates that hormone therapy exerts a variable effect on sperm motility. Although there is a notable enhancement in progressive and non-progressive motility (Grades A, B, and C), the significant rise in immotile sperm (Grade D) prompts worries over potential detrimental effects of the treatment.

## 3. DISCUSSION

The results of our investigation on the effectiveness of hormonal stimulation for idiopathic oligoasthenoteratospermia demonstrate notable enhancements in sperm morphology and overall sperm count post-treatment. The percentage of normal sperm forms increased significantly from 0.933% to 3.6%, whereas the percentage of aberrant forms reduced from 99.067% to 96.4%. The alterations were statistically significant, underscoring the beneficial effect of hormone therapy on sperm quality. Prior studies corroborate these findings, indicating that hormone therapy can enhance normal sperm morphology and diminish aberrant forms, especially in individuals with idiopathic infertility (10).Conversely, although sperm concentration rose from 5.334 × 10^6/ml to 6.067 × 10^6/ml, the alteration was not statistically significant (P = 0.093), suggesting that the hormone therapy exerted a minimal effect on this parameter. Comparable results have been documented in other investigations, indicating that the impact of hormone therapy on sperm concentration was either negligible or ambiguous (11). The total sperm count exhibited a statistically significant increase from 13.794 × 10^6/ml to 17.156 × 10^6/ml (P = 0.042), indicating that hormonal therapy may enhance sperm production, consistent with findings from other studies regarding the impact of hormonal treatment on sperm count (12). These data indicate that hormone therapy can markedly enhance sperm morphology and total sperm count, however its impact on sperm concentration may be restricted. These findings endorse hormonal stimulation to enhance sperm quality in men with oligoasthenoteratospermia; nevertheless, additional research is required to investigate the long-term consequences and appropriate treatment procedures(13).Our work demonstrates that hormone therapy considerably influences sperm motility, resulting in major enhancements in progressive motility (Grades A and B) and a substantial rise in immotile sperm (Grade D). Initially, Grade A motility (rapid progressing sperm) was nonexistent (0%), but post-treatment, it increased slightly to 0.667% ± 0.582, with a highly significant P-value of 0.0001. This indicates that hormonal therapy may elicit a slight enhancement in the percentage of rapidly progressing sperm, which is crucial for fertility. Prior research, including Agarwal et al. (2015), has demonstrated enhancements in sperm motility subsequent to pharmacological intervention for male infertility. Grade B motility (slow progressive sperm) exhibited a notable increase from 1.667% ± 0.637 prior to treatment to 4.667% ± 1.178 post-treatment (P = 0.0116), indicating that hormonal therapy improves both fast and slow progressive sperm, thereby enhancing overall sperm functionality. This discovery corresponds with Moursy et al. (2013), who documented analogous enhancements in sperm motility after to hormone therapy. Grade C motility (non-progressive sperm) shown a considerable enhancement, rising from 8.933% ± 2.273 prior to therapy to 27% ± 8.224 post-treatment (P = 0.0053). While non-progressive sperm are suboptimal for conception, this enhancement suggests that hormone therapy may improve sperm motility, hence potentially facilitating greater sperm travel. This research corroborates the results of Colombo et al. (2016), wherein hormone treatment resulted in a decrease in non-progressive sperm. Grade D motility (immotile sperm) shown a statistically significant increase, escalating from 20.2% ± 9.292 prior to treatment to 52.4% ± 12.676 post-treatment (P = 0.0018). The significant rise in immotile sperm, however seemingly paradoxical, indicates that hormone therapy triggers intricate physiological alterations in sperm motility. The rise in immotile sperm, while necessitating additional research, may indicate transient modifications in sperm functionality. Ciftci et al. (2014) showed analogous tendencies in hormone therapy effects on sperm motility, indicating that alterations in motility were not consistently linear or advantageous. In summary, these findings demonstrate that hormonal treatment notably enhances progressive sperm motility (Grades A and B), yet it is also associated with a

substantial rise in immotile sperm (Grade D), underscoring the necessity for additional research to comprehend the ramifications of these alterations on fertility.

## 4. CONCLUSION

- The hormonal treatment led to a statistically significant enhancement in the percentage of normal sperm morphology (P = 0.002).
- A notable reduction in aberrant sperm morphology was noted (P = 0.049), signifying an improvement in sperm quality.
- Although sperm concentration exhibited a modest rise post-treatment, the alteration was not statistically significant (P = 0.093), indicating a minimal impact of the hormone therapy on this parameter.
- The total sperm count exhibited a substantial rise post-hormonal therapy (P = 0.042), suggesting a potential enhancement in sperm production.
- Fast progressive motility (Grade A) shown a modest enhancement following therapy.
- There was a substantial improvement in slow progressive motility (Grade B) (P = 0.0116), indicating improved sperm motility efficiency.
- Non-progressive motility (Grade C) exhibited a substantial increase (P = 0.0053), signifying a change in sperm motility patterns.

## 5. RECOMMENDATIONS

- Further research with expanded sample numbers and extended follow-up durations is essential to validate these results.
- Assessing the effects of various hormonal therapy protocols may enhance therapeutic results and reduce any adverse impacts on sperm motility.
- The integration of hormone therapy with additional fertility-enhancing interventions should be investigated to ascertain the optimal strategy for increasing male reproductive health.

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