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Pattern of some sperm profiles in infertile male subjects in Owerri

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Abstract

This study thoroughly assessed the semen profile of male infertile individuals and specific subgroups exhibiting defined sperm abnormalities; oligospermia, asthenozoospermia, asthenoteratozoospermia,oligoasthenozoospermia, oligoasthenoteratozoospermia (OAT), and azoospermia—relative to normozoospermic controls, to clarify the extent of spermatogenic dysfunction and its clinical ramifications. A total of 72 male infertile individuals and 72 age-matched controls were evaluated for total sperm count (TSC), progressive motility, total motility, and morphology utilising standard semen analysis techniques.The results showed that the quality of semen was much lower in male subjects who were infertile than in controls. The progressive motility was 14.76 ± 9.46% vs. 55.75 ± 8.68%, the total motility was 19.75 ± 12.91% vs. 65.75 ± 8.68%, the morphology was 4.86 ± 4.66% vs. 56.80 ± 9.35%, and the TSC was 25.84 ± 15.68 × 10^6 vs. 66.91 ± 5.12 × 10^6 sperm cells/ejaculate ($p = 0.001$). Subgroup studies revealed a steady decline in semen parameters throughout the various clinical groups.Men with oligospermia had significantly reduced total sperm count (TSC) (25.66 ± 3.44 × 10^6), progressive motility (24.66 ± 3.55%), and morphology (6.33 ± 1.77%) compared to controls ($p = 0.001$). Individuals with asthenozoospermia and asthenoteratozoospermia demonstrated similar reductions in sperm motility and morphology, with values of 5.00 ± 0.73% and 2.75 ± 0.45%, respectively ($p = 0.001$). Individuals with oligoasthenozoospermia had significant deficiencies across many metrics, with total sperm count (TSC) at 23.91 ± 6.52 × 10^6, progressive motility at 13.66 ± 6.80%, and morphology at 12.58 ± 5.66% ($p = 0.001$). The OAT group exhibited the most significant multi-parameter deficits among the non-azoospermic cohorts (TSC = 18.00 ± 1.04 × 10^6; progressive motility = 11.50 ± 2.61%; morphology = 2.50 ± 0.52%; $p = 0.001$), indicating severe combined quantitative, motility, and structural problems. Azoospermic men showed no sperm cells, motility, or morphology , which means that spermatogenesis completely failed. There were statistically significant differences ($p < 0.05$) between all infertile subgroups and controls.

In general, these results show a distinct range of spermatogenic failure, from partial impairment in oligospermia to total absence of spermatozoa in azoospermia. All of these diseases make it very hard to get pregnant naturally, however men with oligospermia and OAT still have some fertility potential that can be used clinically through assisted reproductive technologies. The study emphasises the diagnostic and predictive significance of complete semen analysis as a fundamental component in the evaluation and treatment of male infertility.

Keywords: *sperm profiles, infertile male, Owerri.*

Introduction

Infertility continues to emerge as an important public health concern affecting millions of couples globally. The intricacies inherent in this disorder stem from several connected variables, including genetic anomalies that affect normal reproductive functioning. These hormonal abnormalities restrict sperm production and quality, as well as lifestyle decisions such as food, physical exercise, and substance usage that adversely affect fertility. Furthermore, environmental exposures to numerous toxins and industrial pollutants are becoming recognized as significant variables in the genesis of

male infertility. A complete understanding of the intricate interplay among these factors is crucial for creating effective ways to address the issues of infertility faced by couples [1].

While infertility is not categorised as a life-threatening disorder, it often generates severe emotional, physical, and psychological distress, followed by social and socioeconomic repercussions. In Nigeria, the societal ramifications of infertility are deeply significant, since individuals may endure isolation, disinheritance, stigmatisation, and even divorce as a result of infertility-related issues. Historically, the perspective of infertility has been largely female-centric, which reinforces negative socio-cultural myths that allocate the responsibility for infertility solely to women. This perspective undermines the importance for men to receive good reproductive healthcare and obscures the truth that male factors contribute to couple infertility in about 20%-30% of instances and further impact female infertility in an additional 30-40% of cases [2]. Variations exist in regions, states, and townships, with a report from Channels TV documentary series called as “Health Matters”; displaying a male factor contribution rate of 3.4% in Owerri. Male infertility is defined as the inability of a man to impregnate a fertile female partner following at least one year of regular, unprotected sexual intercourse. This multidimensional condition demands a range of physiological activities, including healthy sperm generation, concentration, transit, and release, with functioning motility. Any interruption in these processes can dramatically lower the likelihood of successful fertilization and subsequent pregnancy. This aggravated scenario offers major implications for family planning, individual well-being, and broader public health concerns, reflecting numerous socioeconomic, medical, and environmental elements that influence reproductive health [3]. The cause of male infertility is multifaceted, covering sperm-related abnormalities, immune system illnesses, genetic anomalies, hormone imbalances, environmental exposures, and lifestyle variables such as obesity, nicotine, and alcohol usage. In certain circumstances, the cause of male infertility remains idiopathic. Increasing data suggests that oxidative stress plays a significant role in the aetiology of male infertility. Various factors, including environmental pressures, severe physical effort, and deficits in antioxidants, can alter the balance between oxidants and antioxidants, leading to oxidative stress [4] Understanding the sperm patterns of patients with male infertility is critical for discovering the underlying processes of this illness. By explaining the precise sperm patterns connected to male infertility, this research can equip policymakers to create targeted and effective solutions. Such initiatives could lead to improved healthcare results for impacted individuals, ultimately boosting the quality of care and support available for those facing infertility issues. This personalised strategy ensures that healthcare resources are utilised efficiently, addressing the special demands of this patient population [5].

Male infertility constitutes a serious public health challenge, impacting millions of families around the globe. In Nigeria specifically, the situation of male infertility is particularly alarming, with estimates estimating that roughly 32% of men face reproductive issues [6]. This study intends to shed light on this urgent topic and could play a vital role in educating and developing effective strategies to lessen the effects of male infertility, thereby increasing reproductive health and well-being for couples impacted by this condition.

MATERIALS AND METHODS

Study Area

The study was conducted at Imo State Specialist Hospital, Umuguma, Owerri West LGA. Owerri, the vibrant capital city of Imo State, is nestled in the enchanting south-eastern region of Nigeria, West Africa.

Ethical approval

The ethical approval was obtained the Imo State Specialist Hospital, Umuguma, Owerri, Informed consent was obtained from all participants before collecting blood samples. Participants were assured of confidentiality and anonymity. The study was conducted under the principles of respect for persons, beneficence, non-maleficence, and justice.

Study Population and Sample Size

A total of 144 participants were studied at the Imo State Specialist Hospital, Umuguma, Owerri. These participants involved 72 male infertile patients who came to seek treatment at the obstetrics and gynaecology clinic located at Imo State Specialist Hospital Umuguma, Owerri, and 72 healthy fertile male control subjects. The selection of patients was conducted through a thorough assessment process that included evaluating their medical history, a comprehensive physical examination, and relevant laboratory test results to ensure eligibility and suitability for the study.

Study Design

The research design was a cross-sectional study focused on male infertility. The study involved two groups: male subjects diagnosed with infertility and a control group of fertile men.

Semen Analysis

The semen analysis was performed on semen samples collected from participants following a standardized protocol, adhering to the World Health Organization's 6th edition guidelines. Participants were instructed to abstain from ejaculation for 2-7 days before sample collection. Semen samples was collected by masturbation into a sterile, wide-

mouthed container provided by the research team. Participants were advised to deliver the sample to the laboratory within one hour of collection, maintaining it at room temperature during transport. Upon arrival at the laboratory, the time of collection and time of receipt was recorded.

Macroscopic Examination

The semen samples were allowed to liquefy at a temperature of 37°C for up to 15 - 30 minutes. The time taken for liquefaction was noted. Semen volume was measured using a graduated cylinder or a calibrated pipette, and the pH of the semen determined using a pH meter. The appearance of the semen (e.g., homogenous, cloudy, etc.) were visually assessed and recorded.

Microscopic Examination

Sperm motility was assessed using a light microscope at 40x magnification. A minimum of 200 spermatozoa was counted, and the percentage of progressively motile, non-progressively motile, and immotile sperm were determined. Motility was categorized according to the WHO criteria (progressive motility, non-progressive motility, and non-motile).

Sperm concentration was determined using a Makler counting chamber or a similar validated method. The number of spermatozoa in a defined area were counted, and the concentration was expressed as millions of spermatozoa per millilitre (million/mL).

Sperm morphology was assessed using a stained smear (e.g., Papanicolaou stain) and examined under a light microscope at 100x magnification (oil immersion). A minimum of 200 spermatozoa were evaluated, and the percentage of morphologically normal forms was recorded, strictly adhering to the standard criteria for normal sperm morphology. Detailed assessment of the head, midpiece, and tail abnormalities were performed.

Quality Control

Strict quality control measures were implemented throughout the semen analysis process. This will include using calibrated equipment, regular participation in external quality assessment schemes, training of personnel performing the analyses, and blind duplicate analysis of a subset of samples.

Statistical Analysis

The data collected from this study were analyzed using the Statistical Package for the Social Sciences (SPSS) version 23. Descriptive statistics frequency distributions, and percentages, were used.

RESULTS

Table 1: Mean ± SD values of sperm profile (TSC, Progressive Motility, Total motility, and morphology) in male infertile and control subjects, n=72.

Variables (Units)	Male Infertile subjects n=72	Male control subjects n=72	t-value	p-value
Progressive Motility (%) Lower 95% C.I Upper 95% C.I	14.76 ± 9.46 12.54 16.98	55.75 ± 8.68 53.70 57.79	-28.480	0.001
Total motility (%) Lower 95% C.I Upper 95% C.I	19.75 ± 12.91 16.71 22.78	65.75 ± 8.68 63.70 67.79	-24.720	0.001
Morphology (%) Lower 95% C.I Upper 95% C.I	4.86 ± 4.66 3.76 5.95	56.80 ± 9.35 54.60 59.00	-39.640	0.001
TSC (10^6 sperm cells/ejaculate) Lower 95% C.I Upper 95% C.I	25.84 ± 15.68 22.16 29.53	66.91 ± 5.12 65.71 68.12	-21.400	0.001

Value is statistically significant at P<0.05

The above table 1 compared the sperm profiles between male infertile subjects and healthy controls. There were significantly lower levels of progressive motility (14.78 ± 9.46%), total motility (19.75 ± 12.91%), morphology (4.86 ± 4.66%), and total sperm count (25.84 ± 15.68 million sperm cells/ejaculate) in male infertile subjects compared to controls respectively (55.75 ± 8.68%, 65.75 ± 8.68%, 56.80 ± 9.35% and 66.9 ± 5.12 million sperm cells/ejaculate) (p=0.001 in each case).

Table 2: Mean ± SD of sperm profile in Asthenozoospermia vs. Control Subjects

Variables (Units)	Asthenozoospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate) Lower 95% C.I Upper 95% C.I	44.00±5.27 40.64 47.35	67.00±5.02 63.80 70.19	-12.803	0.001
Prog. Motility (%) Lower 95% C.I Upper 95% C.I	17.00±3.54 14.75 19.25	56.00±8.42 50.64 61.35	-14.030	0.001
Total motility (%) Lower 95% C.I Upper 95% C.I	24.25±4.33 21.49 27.00	66.00±8.42 60.64 71.35	-13.142	0,001
Morphology (%) Lower 95% C.I Upper 95% C.I	5.00±0.73 4.53 5.46	55.83±10.12 49.40 62.26	-16.990	0.001

Value is statistically significant at P<0.05

This table 2 compared key semen parameters—total sperm count (TSC), progressive motility, total motility, and sperm morphology—between men diagnosed with asthenozoospermia and normozoospermia control subjects. The results demonstrated statistically significant impairments across all evaluated sperm profile indices in the asthenozoospermia group.

The mean total sperm count (TSC) in asthenozoospermia subjects was significantly lower (44.00 ± 5.27 ×10^6 sperm cells/ejaculate) compared to control subjects (67.00 ± 5.02 ×10^6 sperm cells/ejaculate), with a t-value of -12.803 and a p-value of 0.001. This substantial reduction in sperm concentration highlights a major quantitative defect in the semen of affected individuals.

Progressive motility, a key indicator of sperm functionality and fertilizing potential, was markedly compromised in the asthenozoospermia group. Affected men exhibited a mean progressive motility of 17.00 ± 3.54%, while the control group showed a significantly higher value of 56.00 ± 8.42% (t = -14.030, p = 0.001). Similarly, total motility was significantly reduced in asthenozoospermia individuals (24.25 ± 4.33%) compared to controls (66.00 ± 8.42%), with a t-value of -13.142 and a p-value of 0.001.

Sperm morphology was also drastically affected, with the mean percentage of morphologically normal sperm cells in asthenozoospermia subjects recorded at 5.00 ± 0.73%, in contrast to 55.83 ± 10.12% in the control group. This difference was statistically significant (t = -16.990, p = 0.001), indicating a severe defect in sperm structure among the affected individuals.

Table 3.: Mean ± SD of sperm profile in Asthenoteratozoospermia vs. Control Subjects

Variables (Units)	Asthenoteratozoospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate) Lower 95% C.I Upper 95% C.I	43.50±2.15 42.13 44.86	67.00±5.02 63.80 70.19	-18.766	0.001
Prog. Motility (%) Lower 95% C.I Upper 95% C.I	21.75±9.11 15.95 27.54	56.00±8.42 50.64 61.35	-17.664	0.001
Total motility (%) Lower 95% C.I Upper 95% C.I	13.00±6.48 8.88 17.11	66.00±8.42 60.64 71.35	-16.876	0,001
Morphology (%) Lower 95% C.I Upper 95% C.I	2.75±0.45 2.46 3.03	55.83±10.12 49.40 62.26	-18.152	0.001

Value is statistically significant at P<0.05

Table 3 evaluated semen quality parameters in men with asthenoteratozoospermia compared to normozoospermia control subjects, focusing on total sperm count (TSC), progressive motility, total motility, and sperm morphology. The findings revealed statistically significant impairments across all measured sperm parameters in the asthenoteratozoospermia group.

Total sperm count (TSC) was markedly reduced in asthenoteratozoospermia subjects, with a mean value of 43.50 ± 2.15 $\times 10^6$ sperm cells/ejaculate, compared to 67.00 ± 5.02 $\times 10^6$ sperm cells/ejaculate in the control group. This difference was highly significant (t = -18.766, p = 0.001), indicating a profound deficit in sperm production.

Progressive motility, a crucial determinant of sperm fertilizing capability, was also significantly lower in the affected group (21.75 ± 9.11%) compared to the control group (56.00 ± 8.42%), with a t-value of -17.664 and a p-value of 0.001. Similarly, total motility was severely impaired in asthenoteratozoospermia individuals, who exhibited a mean total motility of 13.00 ± 6.48%, while the control group recorded 66.00 ± 8.42% (t = -16.876, p = 0.001).

Sperm morphology was the most severely affected parameter. The mean percentage of morphologically normal sperm cells in the asthenoteratozoospermia group was only 2.75 ± 0.45%, in stark contrast to 55.83 ± 10.12% observed in the control group. This difference was highly statistically significant (t = -18.152, p = 0.001), reflecting a serious structural abnormality in sperm cells of affected men.

Table 4: Mean ± SD of sperm profile in Azoospermia vs. Control Subjects

Variables (Units)	Azoospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate) Lower 95% C.I Upper 95% C.I	0.00±0.00 - -	67.00±5.02 63.80 70.19	-46.168	0.001
Prog. Motility (%) Lower 95% C.I Upper 95% C.I	0.00±0.00 - -	56.00±8.42 50.64 61.35	-23.037	0.001
Total motility (%) Lower 95% C.I Upper 95% C.I	0.00±0.00 - -	66.00±8.42 60.64 71.35	-27.157	0,001
Morphology (%) Lower 95% C.I Upper 95% C.I	0.00±0.00 - -	55.83±10.2 49.40 62.26	-19.102	0.001

Value is statistically significant at P<0.05

Table 4 assessed the sperm profile of men diagnosed with azoospermia in comparison to normozoospermia control subjects. The parameters evaluated included total sperm count (TSC), progressive motility, total motility, and sperm morphology. The findings revealed a complete absence of sperm in all measured indices among azoospermia individuals, with statistically significant differences observed across all sperm parameters when compared to the control group.

As expected in azoospermia, the total sperm count was 0.00 ± 0.00 $\times 10^6$ sperm cells/ejaculate, indicating a complete absence of sperm cells in the semen. This was significantly different from the mean TSC of 67.00 ± 5.02 $\times 10^6$ in the control group (t = -46.168, p = 0.001), reflecting a profound quantitative defect in sperm production.

Similarly, progressive motility and total motility were completely absent in the azoospermia group (0.00 ± 0.00%), in contrast to 56.00 ± 8.42% and 66.00 ± 8.42%, respectively, in the control group. These differences were highly statistically significant, with t-values of -23.037 (p = 0.001) for progressive motility and -27.157 (p = 0.001) for total motility. The absence of motile sperm further underscores the functional deficiency inherent in azoospermia.

In terms of morphology, azoospermia men exhibited 0.00 ± 0.00% normal sperm morphology, compared to 55.83 ± 10.20% in control subjects. This yielded a t-value of -19.102 and a p-value of 0.001, indicating an extreme morphological deficit due to the complete absence of spermatozoa.

Table 5 Mean ± SD of sperm profile in Oligospermia vs. Control Subjects

Variables (Units)	Oligospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate)	25.66±3.44	67.00±5.02	-31.000	0.001
Lower 95% C.I	23.47	63.80		
Upper 95% C.I	27.85	70.19		
Prog. Motility (%)	24.66±3.55	56.00±8.42	-12.009	0.001
Lower 95% C.I	22.41	50.64		
Upper 95% C.I	26.92	61.35		
Total motility (%)	38.00±1.70	66.00±8.42	-11.153	0,001
Lower 95% C.I	36.91	60.64		
Upper 95% C.I	39.08	71.35		
Morphology (%)	6.33±1.77	55.83±10.12	-16.183	0.001
Lower 95% C.I	5.20	49.40		
Upper 95% C.I	7.46	62.26		

Value is statistically significant at P<0.05

Table 5 examined the sperm profile of men with oligospermia in comparison to normozoospermia control subjects, focusing on total sperm count (TSC), progressive motility, total motility, and sperm morphology. The results demonstrated statistically significant reductions across all parameters in the oligospermia group, highlighting the extent of sperm dysfunction associated with this condition.

Total sperm count was significantly lower in oligospermia men, with a mean value of 25.66 ± 3.44 ×10^6 sperm cells/ejaculate, compared to 67.00 ± 5.02 ×10^6 in the control group. This difference was highly significant (t = -31.000, p = 0.001), indicating a severe quantitative deficiency in sperm output.

Progressive motility, which reflects the proportion of actively moving sperm, was also markedly reduced in the oligospermia group (24.66 ± 3.55%) relative to controls (56.00 ± 8.42%), with a t-value of -12.009 and a p-value of 0.001. Total motility followed a similar trend, with affected individuals showing 38.00 ± 1.70% motile sperm versus 66.00 ± 8.42% in the control group (t = -11.153, p = 0.001), indicating compromised sperm movement, which is crucial for natural fertilization.

Sperm morphology, which refers to the percentage of sperm with normal shape and structure, was also significantly impaired. The mean morphology value in oligospermia men was 6.33 ± 1.77%, whereas the control group exhibited a much higher value of 55.83 ± 10.12% (t = -16.183, p = 0.001), emphasizing a marked structural abnormality in sperm from the oligospermia group.

Table 6 Mean ± SD of sperm profile in Oligoasthenozoospermia vs. Control Subjects

Variables (Units)	Oligoasthenozoospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate)	23.91±6.52	67.00±5.02	-16.528	0.001
Lower 95% C.I	19.76	63.80		
Upper 95% C.I	28.06	70.19		
Prog. Motility (%)	13.66±6.80	56.00±8.42	-15.314	0.001
Lower 95% C.I	9.34	50.64		
Upper 95% C.I	17.98	61.35		
Total motility (%)	23.25±11.02	66.00±8.42	-12.403	0,001
Lower 95% C.I	16.24	60.64		
Upper 95% C.I	30.25	71.35		
Morphology (%)	12.58±5.66	55.83±10.12	-13.463	0.001
Lower 95% C.I	8.98	49.40		
Upper 95% C.I	16.18	62.26		

Value is statistically significant at P< 0.05

Table 6 compared the sperm profile of men diagnosed with Oligoasthenozoospermia to that of normozoospermia control subjects, focusing on total sperm count (TSC), progressive motility, total motility, and sperm morphology. The findings revealed significant impairments across all assessed semen parameters in the Oligoasthenozoospermia group.

Total sperm count in Oligoasthenozoospermia men was markedly lower, with a mean value of 23.91 ± 6.52 $\times 10^6$ sperm cells/ejaculate, compared to 67.00 ± 5.02 $\times 10^6$ in the control group. This difference was statistically significant (t = -16.528, p = 0.001), indicating a pronounced deficit in sperm production.

Progressive motility, which is crucial for effective sperm transport and fertilization, was significantly reduced in affected individuals (13.66 ± 6.80%) compared to the control group (56.00 ± 8.42%). The t-value of -15.314 and p-value of 0.001 confirm a substantial impairment in this functional parameter. Similarly, total motility was significantly compromised in the Oligoasthenozoospermia group (23.25 ± 11.02%) relative to controls (66.00 ± 8.42%), with a t-value of -12.403 and p = 0.001.

Sperm morphology, reflecting the proportion of structurally normal sperm cells, was also significantly reduced. The affected group exhibited a mean morphology of 12.58 ± 5.66%, whereas control subjects had a significantly higher value of 55.83 ± 10.12% (t = -13.463, p = 0.001). This indicates a high prevalence of abnormal sperm forms in Oligoasthenozoospermia men.

Table 7: Mean ± SD of sperm profile in Oligoasthenoteratozoospermia vs. Control Subjects

Variables (Units)	Oligoasthenotera tozoospermia n = 12	Control Subjects n = 12	t-value	p-value
TSC (10^6 sperm cells/ejaculate) Lower 95% C.I Upper 95% C.I	18.00±1.04 17.33 18.66	67.00±5.02 63.85 70.19	-31.180	0.001
Prog. Motility (%) Lower 95% C.I Upper 95% C.I	11.50±2.61 9.84 13.15	56.00±8.42 50.64 61.35	-23.709	0.001
Total motility (%) Lower 95% C.I Upper 95% C.I	20.00±4.17 17.34 22.65	66.00±8.42 60.64 71.35	-23.331	0,001
Morphology (%) Lower 95% C.I Upper 95% C.I	2.50±0.52 2.16 2.83	55.83±10.12 49.40 62.26	-17.833	0.001

Value is statistically significant at P< 0.05

Table 7 examined the sperm profile of men with oligoasthenoteratozoospermia (OAT) in comparison to normozoospermia control subjects. The evaluated parameters included total sperm count (TSC), progressive motility, total motility, and sperm morphology. The results revealed profound and statistically significant reductions across all indices in the OAT group, highlighting the severity of combined sperm abnormalities in this condition.

The total sperm count in men with OAT was significantly lower, with a mean value of 18.00 ± 1.04 $\times 10^6$ sperm cells/ejaculate, compared to 67.00 ± 5.02 $\times 10^6$ sperm cells/ejaculate in the control group. This difference was highly significant (t = -31.180, p = 0.001), indicating a marked impairment in sperm production.

Progressive motility, which measures the proportion of sperm moving actively in a forward direction, was also severely reduced in the OAT group (11.50 ± 2.61%) compared to the control group (56.00 ± 8.42%). This reduction was statistically significant (t = -23.709, p = 0.001), signifying major functional deficits. Similarly, total motility in the OAT group was 20.00 ± 4.17%, far below the control value of 66.00 ± 8.42%, with a t-value of -23.331 and a p-value of 0.001.

Sperm morphology, representing the percentage of sperm with normal structure, showed one of the most striking differences. Men with OAT had a mean morphology of just 2.50 ± 0.52%, compared to 55.83 ± 10.12% in the control group. This difference was highly significant (t = -17.833, p = 0.001), confirming severe structural abnormalities in sperm from the affected individuals.

Discussion

Men who were infertile had a significantly lower mean progressive motility (14.76 ± 9.46%) than the controls (55.75 ± 8.68%). A crucial component of natural conception, progressive motility indicates the sperm's capacity to travel effectively in the direction of the egg. Suboptimal progressive motility is defined as less than 32%. Oxidative stress, DNA fragmentation, and flagella structural anomalies can all impair sperm motility. [7] Given that male infertility frequently results from systemic oxidative and inflammatory stresses, the marked decrease in motility observed here suggested functional abnormalities. Compared to controls (65.75 ± 8.68%), infertile participants had considerably lower total motility (19.75 ± 12.91%), which includes both progressive and non-progressive motion. This decrease is consistent with other research demonstrating that genital tract infections, hormone abnormalities, and oxidative stress can all reduce sperm motility in general. Its therapeutic importance is further supported by the fact that total motility below 40% is frequently linked to lower rates of fertilisation[8]. The most noticeable variation between the groups was seen in sperm morphology. The mean morphology of infertile men was only 4.86 ± 4.66%, whereas that of controls was 56.80 ± 9.35%. Impaired spermatogenesis brought on by oxidative stress, heat exposure, toxins, or hereditary factors frequently results in morphological abnormalities. Even with assisted reproductive technology, abnormal morphology is strongly associated with poor fertilisation [9]. With a p-value of 0.001, the infertile group's TSC was substantially lower (25.84 ± 15.68 × 10^6/ejaculate) than the controls' (66.91 ± 5.12 × 10^6/ejaculate). Ejaculates with TSC levels less than 39 million have been deemed subfertile. A low TSC suggested reduced spermatogenic production, which could be brought about by hormonal imbalances, testicular disease, or environmental/lifestyle factors [10].

The severity of infertile men's reproductive dysfunction was shown by their 61.4% drop in TSC. TSC was considerably lower in all infertile groupings than in controls (67.00 ± 5.02 ×10^6 sperm/ejaculate). As anticipated, men with azoospermia had 0.00 ± 0.00 ×10^6 sperm cells in their ejaculate, indicating total obstruction or failure of the testicles. Additionally, oligoasthenotetratozoospermia (18.00 ± 1.04) and oligoasthenozoospermia (23.91 ± 6.52) showed severe reductions in TSC, indicating more complex spermatogenic abnormalities. Despite having a lower TSC, the Oligospermia group (25.66 ± 3.44) nevertheless had detectable sperm present. This pattern is compatible with either hormonal abnormalities impacting Sertoli cell function or partial spermatogenic arrest [11]. All infertile subgroups had considerably lower progressive motility, a crucial indicator of fertilisation potential, than controls (56.00 ± 8.42%). According to WHO standards for asthenozoospermia, the lowest values were reported in oligoasthenotetratozoospermia (11.50 ± 2.61%), oligoasthenozoospermia (13.66 ± 6.80%), and asthenozoospermia (17.00 ± 3.54%). The end of the male infertility continuum was further underlined by the complete lack of motile sperm in individuals with azoospermia [12]. Increased oxidative stress, mitochondrial malfunction, and structural abnormalities of the flagella are frequently cited as causes of poor motility. The trend for total motility was similar. Comparing all groupings to controls (66.00 ± 8.42%), there were highly significant decreases ($p < 0.001$). Patients who were azoospermic (0.00 ± 0.00%) and asthenotetratozoospermic (13.00 ± 6.48%) had the lowest overall motility. Despite having a comparatively higher overall motility (38.00 ± 1.70%), oligospermic individuals nevertheless displayed noticeably lower levels than controls. According to these findings, infertile males frequently experience a simultaneous decline in sperm quantity and quality, which is a defining feature of mixed diseases like Oligoasthenozoospermia. [13] All infertile subgroups had significantly reduced sperm morphology ($p < 0.001$), with azoospermia men once more exhibiting 0.00% normal forms. Extremely poor morphology was displayed by the oligoasthenotetratozoospermia group (2.50 ± 0.52%), asthenotetratozoospermia (2.75 ± 0.45%), and oligoasthenozoospermia (12.58 ± 5.66%). The control group, on the other hand, showed a sharp contrast with a mean morphology of 55.83 ± 10.12%. Defects in chromatin packaging, DNA breakage, and cytoplasmic retention are linked to abnormal morphology and have a detrimental effect on fertilisation and embryo development [14]. Those with oligoasthenozoospermia had a substantially greater TSC of 23.91 ± 6.52 × 10^6 cells/ejaculate ($p = 0.001$) than those with azoospermia, who as expected had no sperm cells at all in the ejaculate (0.00 ± 0.00 × 10^6 cells/ejaculate). The complete spermatogenic failure in azoospermia, which can result from either obstructive or non-obstructive reasons such testicular atrophy, genetic abnormalities (like Klinefelter syndrome), or hormonal imbalances, is highlighted by this striking difference [15]. Additionally, OA individuals had moderate progressive motility (13.66 ± 6.80%) and total motility (23.25 ± 11.02%), both of which were considerably greater ($p < 0.001$) than azoospermic men, who showed no sperm motility. A key factor in determining fertilisation potential is sperm motility, and a decrease in it in OA indicates functional sperm impairment that may be related to oxidative stress, mitochondrial malfunction, or abnormalities in seminal plasma [16]. Additionally, OA patients had considerably better sperm morphology (12.58 ± 5.66%) than azoospermic people without sperm cells to measure morphology ($p = 0.001$). Infertility is frequently linked to morphological defects, which are impacted by both genetic and environmental factors and frequently arise from faulty spermatogenesis [17]. These results highlight the severity of oligoasthenozoospermia, a partial functional compromise with some remaining reproductive potential, in contrast to azoospermia, a clinical disease marked by the total lack of spermatozoa. Therefore, prognosis assessments and therapeutic approaches need to be customised according to the level of sperm damage seen in these groups. The azoospermic patients had a total sperm count (TSC) of 0.00 ± 0.00 × 10^6 cells/ejaculate, which was completely absent. This confirmed the diagnosis and indicated either a non-obstructive or obstructive aetiology. The mean TSC of 43.50 ± 2.15 × 10^6 cells/ejaculate, on the other hand, was considerably greater in men with ATT, indicating maintained spermatogenesis despite functional limitations. The significant difference (t = -

69.983, p = 0.001) is consistent with research by [18], which highlighted that azoospermia is a more serious clinical state that has a typically worse prognosis for reproduction. While progressive motility was completely absent in azoospermic patients, it was statistically significant (t = -8.264, p = 0.001) and averaged 21.75 ± 9.11% in the ATT group. The capacity of sperm to reach and fertilise the oocyte is largely determined by progressive motility, and oxidative stress and mitochondrial dysfunction have been linked to its impairment in ATT [19]. The functional difference between the two groups was further supported by the identical trend in total motility, with 13.00 ± 6.48% motile sperm in ATT individuals compared to 0% in azoospermia. Additionally, the ATT group outperformed the azoospermic group, which lacked spermatozoa to evaluate morphology, by a substantial margin (2.75 ± 0.45%) in sperm morphology, another crucial factor for fertilisation success (t = -21.063, p = 0.001). The morphological percentage in ATT patients offers some hope for natural or aided conception, even if it is still below the WHO's standard reference level of 4% [20]. All of these results highlight how serious azoospermia is as a diagnosis that shows total lack of spermatogenesis or sperm blockage, whereas ATT indicates qualitative sperm abnormalities. The findings are consistent with the necessity of customised fertility treatments and customised diagnostic evaluations. Antioxidant treatment and sperm optimisation techniques may help ATT people, but more invasive procedures like testicular sperm extraction (TESE) and in vitro fertilisation (IVF) are sometimes needed for azoospermic patients. With a t-value of -9.746 and a p-value of 0.001, the total sperm count (TSC) was significantly greater in men with asthenotetratozoospermia ($43.50 \pm 2.15 \times 10^6$ sperm cells/ejaculate) than in men with oligoasthenozoospermia ($23.91 \pm 6.52 \times 10^6$). The reduced TSC seen in oligoasthenozoospermia points to both quantitative and kinetic inadequacies in these patients, indicating a compounding effect of oligozoospermia and asthenozoospermia [21]. With a p-value of 0.012, oligoasthenozoospermia participants had substantially lower progressive motility—a crucial factor in determining a sperm's capacity to reach and fertilise the oocyte—13.66 ± 6.80%) than asthenotetratozoospermic men (21.75 ± 9.11%). This finding implies that motility might be more negatively affected in oligoasthenozoospermia, possibly as a result of structural abnormalities of the flagella, increased oxidative stress, or mitochondrial malfunction [22]. Overall motility, on the other hand, was statistically significant (t = 3.090, p = 0.010) and strangely higher in the Oligoasthenozoospermia participants (23.25 ± 11.02%) than in the asthenotetratozoospermia subjects (13.00 ± 6.48%). This finding points to a complicated relationship between sperm count and motility, whereby a sufficient sperm concentration may maintain a greater overall proportion of motile sperm even in the face of decreased progressive motility. [23] With a p-value of 0.001, the most notable difference was seen in sperm morphology, where participants with oligoasthenozoospermia had a significantly larger percentage of normal forms (12.58 ± 5.66%) than their asthenotetratozoospermic counterparts (2.75 ± 0.45%). Teratozoospermia is characterised by morphological abnormalities, which have been closely associated with poor ART results, DNA fragmentation, and reduced fertilisation potential [24]. The ATT group's severe teratozoospermia component is highlighted by this notable discrepancy. These results demonstrate the variable character of male infertility, where underlying pathophysiology can cause individual sperm characteristics to vary. In order to customise efficient treatment plans, such as antioxidant therapy, hormone management, or assisted reproductive technologies (ART), the data suggest the necessity of thorough semen analysis and oxidative stress profile. In contrast to oligospermia participants ($25.66 \pm 3.44 \times 10^6$), azoospermia subjects ($0.00 \pm 0.00 \times 10^6$) exhibited a predictable and significant difference in TSC (t = -25.797, p = 0.001). Azoospermia is defined by the total lack of sperm cells in the ejaculate. The diagnostic criteria, which characterise azoospermia as a severe form of spermatogenic failure, are in line with this result. In terms of fertility potential, the existence of spermatozoa in oligospermia participants indicates continued spermatogenic activity, albeit at a lower concentration, providing a somewhat better prognosis. [25] Because they lacked spermatozoa, azoospermic people lacked both progressive motility and total motility. In contrast, males with oligospermia had significantly higher values—24.66 ± 3.55% for progressive motility and 38.00 ± 1.70% for total motility (p = 0.001 for both). These results highlight how important motility is to sperm function, especially during spontaneous conception when sperm migration and penetration of the zona pellucida and cervical mucus are crucial. Even though oligospermia's decreased motility might still affect fertility, it's a better option than azoospermia, which usually calls for more sophisticated reproductive procedures like intracytoplasmic sperm injection (ICSI) and testicular sperm extraction (TESE). [26] Additionally, oligospermia participants exhibited significantly superior sperm morphology (6.33 ± 1.77%) than azoospermia subjects, who scored 0.00 ± 0.00% (t = -12.358, p = 0.001). The comparatively low proportion of normal forms in oligospermia participants is compatible with the common occurrence of teratozoospermia in male infertility, whereas the lack of sperm in azoospermia precludes morphological evaluation. Unusual morphology is linked to increased sperm DNA fragmentation and decreased fertilising ability, which can result in unsuccessful fertilisation or early embryo arrest [27].

Conclusion

This study showed that male infertility is linked to decreased sperm motility, morphology, and concentration.

References

1. Roshan, K., Mahat, D., Vasantrao, B., & Vedika, R. (2023). Oxidative stress in seminal plasma negatively influences sperm quality in infertile males. International Journal of Integrated Health Sciences, 11(1), 27–31.
2. Aitken, R. J., Drevet, J. R., Moazamian, A., & Gharagozloo, P. (2022). Male infertility and oxidative stress: A focus on the underlying mechanisms. Antioxidants, 11(2), 306.
3. Banerjee, A., Sanyal, S., & Das, K. (2021). The role of oxidative stress and antioxidant balance in male infertility. Asian Pacific Journal of Reproduction, 10(2), 47–52.
4. Takeshima, T., Usui, K., Mori, K., Asai, T., Yasuda, K., & Kuroda, S. (2021). Oxidative stress and male infertility. Reproductive Medicine and Biology, 20, 41–52.
5. Zini, A., & Boman, J. M. (2022). Sperm morphology: Diagnostic value and relationship to fertility. Asian Journal of Andrology, 24(3), 223–230.
6. Edward, U., Marvis, U., Okehie, E. C., & Emmanuel, I. O. (2023). Studies on fertility hormone in azoospermic men attending Imo State Specialist Hospital, Owerri. Newport International Journal of Research in Medical Sciences, 2(4), 9–12.
7. Stormont, G. D., & Deibert, C. M. (2021). Genetic causes and management of male infertility. Translational Andrology and Urology, 10(3), 1365–1372.
8. Agarwal, A., Parekh, N., & Panner Selvam, M. K. (2019). Oxidative stress and male infertility: A clinical perspective. Reproductive Biomedicine Online, 38(6), 606–617.
9. Gharagozloo, P., & Aitken, R. J. (2017). The role of sperm oxidative stress in male infertility and the significance of oral antioxidant therapy. Human Reproduction, 26(7), 1628–1640.
10. Agarwal, A., Mulgund, A., Hamada, A., & Chyatte, M. R. (2019). A unique view on male infertility around the globe. Reproductive Biology and Endocrinology, 13(37), 1–9.
11. Cherry, N., Labreche, F., Collins, J. J., & Teschke, K. (2022). Occupational exposures and male infertility: A review. Occupational and Environmental Medicine, 65(10), 708–716.
12. Ko, E. Y., Sabanegh, E. S., & Agarwal, A. (2024). Male infertility testing: Reactive oxygen species and antioxidant capacity. Fertility and Sterility, 102(6), 1518–1527.
13. Abdullah, F., Khan Nor-Ashikin, M. N., Agarwal, R., Kamsani, Y. S., Malek, M. A., Bakar, N. S., Kamal, A. M., Sarbandi, S., Rahman, S. A., & Musa, N. H. (2021). Glutathione (GSH) improves sperm quality and testicular morphology in streptozotocin-induced diabetic mice. Asian Journal of Andrology, 23(3), 281.
14. Ricci, E., Bertawi, S., Cipriani, S., Candiani, M., Chiaffarino, F., Vigano, P., Noli, S., & Parazzini, F. (2019). Semen quality and alcohol intake: A systematic review and meta-analysis. Reproductive Biomedicine Online, 34(1), 38–47.
15. Nargund, V. H. (2021). Effects of psychological stress on male fertility. Nature Reviews Urology, 12(7), 373–381.
16. Guzick, D. S., Overstreet, J. W., Factor-Litvak, P., Brazil, C. K., Nakajima, S. T., Coutifaris, C., & Steinkampf, M. P. (2021). Sperm morphology, motility, and concentration in fertile and infertile men. New England Journal of Medicine, 345(19), 1388–1393.
17. Castleton, P. E., Deluao, J. C., Sharkey, D. J., & McPherson, N. O. (2022). Measuring reactive oxygen species in semen for male preconception care: A scientist perspective. Antioxidants, 11(2), 264.
18. Adams, J. A., Galloway, T. S., Mondal, D., Esteves, S. C., & Mathews, F. (2022). Effect of mobile telephones on sperm quality: A systematic review and meta-analysis. Environment International, 70, 106–112.
19. Hammoud, A. O., Gibson, M., Peterson, C. M., Hamilton, B. D., Carrell, D., Harley, A., Agarwal, A., & Gunes, S. O. (2023). Smoking and male infertility: An evidence-based review. The World Journal of Men's Health, 33(3), 143–160.
20. Caroppo, E., & Colpi, G. M. (2023). Male infertility: A review of key papers appearing in the reproductive medicine and andrology section of the Journal of Clinical Medicine. Journal of Clinical Medicine, 12(6), 2366.
21. Aditi, S., Suks, M., Waljit, D. S., & Channa, J. N. (2021). Male infertility due to testicular disorders. Journal of Clinical Endocrinology and Metabolism, 106(2), 442–459.
22. Chinyerum, S., Opuwari, E., Nnamso, A., & Swesme, E. (2023). Male infertility in Nigeria and South Africa: A ten-year observational study. Scientific Reports, 13(6819), 1–8.
23. Kais, K. H., Hanan, K. A., & Hasanain, A. H. (2024). Evaluation of some immune parameters in infertile men. Latin American Journal of Pharmacy, 43(3), 831–835.
24. Barati, E., Nikzad, H., & Karimian, M. (2020). Oxidative stress and male infertility: Current knowledge of pathophysiology and role of antioxidant therapy in disease management. Cellular and Molecular Life Sciences, 77, 93–113.
25. Cai, L., Gao, L., Wang, F., Zhang, L., & Guo, X. (2020). Association between hypertension and semen quality: A meta-analysis. Andrologia, 52(4), e13548.
26. Grande, G., Graziani, A., Scafa, R., Garolla, A., Santi, D., & Ferlin, A. (2024). FSH therapy in male factor infertility: Evidence and factors which might predict the response. Life, 14(8), 969.
27. Kaltsas, A. (2023). Oxidative stress and male infertility: The protective role of antioxidants. Medicina, 59(10), 1769.

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