# Impact of Testosterone propionate on semen quality and endocrine profile in male Wistar Rats

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## ABSTRACT

**Aim:** This study investigated the impact of testosterone propionate on semen quality and endocrine profile in male wistar rats.

**Study design:** Thirty-five (35) male wistar rats were used for this study. The rats were divided into seven groups (n =7) of five rats per group. Group 1, the normal control received feed and water only. The test groups 2-7 received different doses of testosterone propionate for varying periods. Groups 2 and 3 received 3mg/kg b. wt of TP for 11days and 22 days respectively. Groups 4 and 5 received 6mg/kg b. wt of TP for 11 days and 22 days respectively. Groups 6 and 7 received 9mg/kg b. wt of TP for 11 days and 22 days respectively.

**Place and duration of study:** The study was conducted in the Faculty of basic medical sciences animal house, University of Port Harcourt, Choba, Rivers state, Nigeria. Between October 2024 to March 2025

**Methodology:** The rats were subjected to 24 hours fasting prior to sacrifice. Blood samples were collected and used to determine hormonal assay. Seminal fluid was extracted from the rats' epididymis and was examined microscopically for spermatozoa evaluation.

**Results:** the induced groups with testosterone propionate showed a significant($P$ =.05) decrease in sperm motility and total sperm count when compared to the normal control group 1. Whereas the concentration of testosterone was elevated in all the testosterone induced groups when compared to the normal control group1, FSH and LH levels decreased in the induced groups when compared with the normal control group1 ($P$ =.05) The histology of the testis shows varied degrees of histological distortions in the testosterone induced groups when compared with normal control group 1.

**Conclusion:** The rat's induction with testosterone propionate resulted in dysregulation of both semen quality and hormonal parameters.

*Keywords*: Testosterone, follicle stimulating hormone, luteinizing hormone, semen fluid, hormonal assay.

## 1. INTRODUCTION

Infertility is one a rising problem in the world, it is a significant health challenge affecting millions pf couples worldwide with male infertility accounting to approximately 50% of cases [1] Numerous external and internal factors including environmental, genetic, and lifestyle factors can contribute to male infertility, including hormonal imbalances, oxidative stress, infections, and exposure to endocrine disrupting chemicals [2]. Testosterone plays a crucial role in male reproductive health, influencing sperm production, libido, and overall endocrine function [3]. Aging is characterized by testosterone deficiency due to decreasing testosterone levels associated with low testicular production, genetic factors, adiposity, and illness [4]. Low testosterone levels in men are associated with sexual dysfunction (low sexual desire, erectile dysfunction), reduced skeletal muscle mass and strength, decreased bone mineral density, increased cardiovascular risk and alterations of the glycometabolic profile [5]. Testosterone replacement therapy (TRT) has gained attention as a treatment for hypogonadism and other testosterone deficiencies. While TRT offers benefit such as improved libido, muscle mass and bone density, its long term impact on male fertility remains controversial, it shows several therapeutic effects while maintaining a good safety profile in hypogonadal men [6]. TRT restores normal levels of serum testosterone in men, increasing libido and energy level and producing beneficial effects on bone density, strength and muscle as well as yielding cardio protective effects [7]. Nevertheless, TRT could be contraindicated in men with untreated prostate cancer, different potential side effects, such as polycythemia, cardiac events and obstructive sleep apnea, should be monitored [8]. Testosterone has a positive correlation with semen parameters, meaning higher testosterone levels are generally associated with better sperm count and motility [9]. However, when testosterone levels are artificially elevated through exogenous administration (like testosterone therapy), it can suppress the hypothalamic-pituitary-gonadal (HPG) axis, which can negatively impact on sperm production by suppressing the production of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which are necessary for spermatogenesis, potentially leading to decreased semen quality and infertility [10]. Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are critical regulators of male reproductive function, primarily through their actions on the testes. These gonadotropins are secreted by the anterior pituitary gland under the control of gonadotropin-releasing hormone (GnRH) from the hypothalamus. LH plays a key role in stimulating the Leydig cells of the testes to produce testosterone, which is essential for spermatogenesis, libido, and secondary sexual characteristics. On the other hand, FSH primarily acts on the Sertoli cells within the seminiferous tubules, promoting sperm maturation and supporting spermatogenesis.[11].

The intricate balance between LH and FSH is crucial for maintaining normal reproductive function. Elevated testosterone levels, whether due to endogenous production or exogenous administration, exert negative feedback on the hypothalamic-pituitary-gonadal (HPG) axis, leading to reduced secretion of both LH and FSH. This suppression disrupts spermatogenesis, potentially leading to decreased sperm count and impaired fertility. In the context of testosterone propionate administration, the suppression of LH and FSH is expected, which may explain the observed alterations in semen quality and testicular histology in the study. Understanding the mechanistic role of these hormones provides insight into the complex interplay between exogenous testosterone and male reproductive health, emphasizing the importance of maintaining hormonal balance to preserve fertility.[11]

Long-term use of testosterone propionate causes enlarged prostate, decrease in sperm production and inconsistence in hormonal parameters [12]. The adverse effect of this is increasing on daily basis, making it a health concern as causes of infertility are on the rise. Despite the prevalence of male infertility in Nigeria, not much effort has been made at tackling the problem; therefore, if adequate measures are not taken there will be an increasing impact of male infertility[13].

### 1.1 Aim of Study

This study investigates the impact of testosterone propionate on semen quality and hormonal parameters in male wistar rats, aiming to provide insights into its potential risk concerning fertility.

### 1.2 Objectives

i. Determination of Hormonal Parameters
ii. Semen Analysis
iii. Histological Assessment of Testicular Structures
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## 2. Materials and Methods

### 2.1. Study Animals

The animal model employed for this study were thirty –five (35) adult male Wistar rats which were eight weeks' old weighing 180-250g). They were housed in clean plastic cages with controlled environmental temperature of 25°C-28°C, 12-hour light/dark cycle. The rats were allowed access to water and normal rat chow freely throughout the duration of the study. The rats were also acclimatized for 2 weeks before the commencement of the study. The procedure for animal handling and the study methodology conformed to the ethical Guideline on animal research as stipulated by guideline for Laboratory Animal Care (NIH Publication 1996, No 85-23).

### 2.2. Experimental Design

Rats were induced daily through subcutaneous injection (S.C) with testosterone-propionate adopting the method laid forth by Awajirih et el., [14] .They were randomly selected into seven groups of five animals each(n=5) ,Group 1 (normal control group) received feed and water only , Group 2 received TP (3mg/kg b.wt) for 11days , Group 3 received TP (3mg/kg b.wt) for 22days, Group 4 were induced with TP(6mg/kg b.wt) for 11 days , Group 5 were given TP (6mg/kg b.wt) for 22 days, Group 6 were administered with TP (9mg/kg b.wt) for 11 days, Group 7 were induced with TP (9mg/kg b.wt ) for 22 days. Following the methodology of Awarajih et al. [14], this dosage administration was implemented to study the effective dose of testosterone propionate, thereby providing insights into its potential to induce negative effects during replacement therapy.The animal grouping and induction protocol is summarized in Table 1.

| GROUPS | INDUCED AND NON INDUCED GROUPS |
|---|---|
| **Group 1** | Normal rats received feed and water only |
| **Grou*p* 2** | TP (3mg /kg. b. wt) for 11 days |
| **Group 3** | TP (3mg/kg. b. wt) for 22 days |
| **Group 4** | TP (6mg/kg. b. wt) for 11 days |
| **Group 5** | TP (6mg/kg. b. wt) for 22 days |
| **Group 6** | TP (9mg/kg. b. wt) for 11 days |
| **Group 7** | TP (9mg/kg. b. wt) for 22 days |

**Table 1: indicates the research design from group 1 to group 7 which were induced with the highest concentration of testosterone propionate**

### 2.3. Determination of Hormonal Parameters

The rats were subjected to 24- hours fasting period before sacrifice, and blood samples were collected in plain bottles. The blood samples were centrifuged at 3000 rpm for 20mins to separate the serum, which was then use to determine testosterone, FSH and LH concentration levels were estimated using bio check, ELIZA kit, with catalog number: BC-1115 FOR TESTOSTERONE, FSH catalog number: BC-1029 and LH with catalog number: BC-1031, following manufacturer's instructions.

### 2.4. Semen Analysis

Assessment of semen parameters, which include, the percentage of motile cells, dead cells, and morphological appearance of the sperm cells of the spermatozoa and total sperm count were determined by adopting method of Ogheneochuko et al.,2024. [15]. Semen fluid was extracted from the rat's epididymis onto a clean microscope slide, followed by the addition of few drops of normal saline. This preparation was used to evaluate sperm quality, including total sperm count, motility, and morphology.

The total sperm cells were counted in the five larger squares of the counting chamber using a high-magnification of the objective lens of the microscope. The motility assessment after sample preparation, the active sperm cells and dead cells were recorded accordingly

### 2.5. Histological Assessment of Testicular Structures

The testes were rapidly removed from the rats and fixed in 10% formalin for 24 hrs. The testicular tissue was then dehydrated and embedded in paraffin by standard procedures. Sections were deparaffinized and rehydrated. After hematoxylin and eosin (HE) staining, the histopathology of the testicle was observed with microscopic assessment (Leica Microsystems DM1000, Wetzlar, Germany).

### 2.6. Statistical Analysis

Data collected were analyzed using one-way analysis of variance (ANOVA) followed by Tukey's (HSD) multiple comparison. The significance of the data was assessed using a *P*-value of less than 0.05 and reported as Mean ± Standard Error of Mean (SEM).

## 3. RESULTS AND DISCUSSION

## 3.1 RESULTS

### 3.1.1. Effect of TP on Semen Quality Parameters

Individual results of seminal fluid analyzed is given in Table 2. The analysis of semen quality parameters showed that rats induced with testosterone propionate (TP) showed a significant($P$ =.05) decrease in sperm motility(mot), viability(viab) and total sperm count(TSC) for the experimental groups when compared to the normal control group 1. Higher doses and prolonged exposure to TP resulted to more pronounced negative effects with Group 7 induced with 9mg/kg b. wt of the TP for 22 days showed a more Significant decrease in the number of active sperm cells to 4.67±4.16% when compared to the normal control group with active cells of 24.67±24.03 However, the morphology showed an increase in the head defect, midpiece defect and tail defect which was statistically significant at ($P$ =.05).

### 3.1.2. Effect of TP on Hormonal Parameters

Hormonal analysis as shown in Fig 1. Shows the levels of testosterone(TEST),follicle stimulating hormone(FSH), and luetinizing hormone(LH) in testosterone propionate(TP) treated rats as shown in Fig 1. The concentration of TEST in the normal control was 3.67±0.81 ng/ml which significantly ($P$ =.05) increased to 10.07ng/ml (group 6) and 8.20ng/ml (group 7) indicating a significant increase in serum testosterone in all TP-treated groups. However, this was accompanied by a significant ($P$ =.05) decrease in the levels of FSH and LH for the treated groups when compared to the normal control.

### 3.1.3. Effect of TP on Testes Histoarchitecture

The testes of the control shows normal histological features. The experimental groups showed varied degrees of histological distortions.

**Table 2: Semen Analysis of Testosterone Propionate Treated Normal Male Wistar Rats**

| Grp | Mot(%) | Act(%) | Slu(%) | Dead(%) | TSC(* $10^6$ cells /mL) | Viab(%) | Hd Df(%) | Mid Df(%) | Tail Df(%) |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 85.00±13.23[a] | 73.33±11.55[a] | 11.67±2.89[a] | 15.00±13.23*a* | 113.33±31.53[a] | 94.33±2.89a | 2.33±2.31[a] | 2.00±0.00[a] | 0.67±0.57[a] |
| 2 | 30.00±26.46[b] | 18.33±18.93[b] | 11.67±7.64[a] | 70.00±26.46[b] | 30.00±4.00[b] | 54.00±47.15[b] | 8.00±3.46[b] | 9.00±9.64[b] | 6.33±3.21[b] |
| 3 | 15.00±13.23[c] | 11.67±10.40[b] | 3.33±2.89[b] | 51.67±44.81[c] | 22.67±25.32[b] | 51.00±44.17[b] | 2.67±4.62[a] | 4.00±5.29[b] | 2.33±3.21[c] |
| 4 | 48.67±8.08[b] | 24.33±8.15[c] | 24.33±4.04[c] | 51.33±8.08[c] | 39.33±14.01[b] | 79.00±6.08[c] | 5.67±1.53[b] | 5.67±2.08[c] | 5.67±1.53[b] |
| 5 | 21.67±7.53[b] | 16.67±28.87[c] | 5.00±8.66[b] | 11.67±20.21[d] | 13.33±23.09[c] | 28.67±49.65[d] | 1.67±2.89[a] | 1.33±2.31[a] | 1.67±2.89[a] |
| 6 | 21.67±18.93[b] | 6.67±7.64[d] | 15.00±13.23[d] | 78.33±18.93[b] | 26.00±7.21[b] | 89.00±3.40[c] | 5.33±5.58[b] | 3.33±2.52[c] | 2.33±1.53[c] |
| 7 | 24.67±24.03[b] | 4.67±4.16[d] | 20.00±20.00[c] | 75.33±24.03[b] | 25.00±4.58[b] | 86.67±5.77[c] | 4.33±6.58[b] | 2.67±3.06[a] | 9.67±5.51[b] |

**[a] Mot – Percentage of sperm cell motility, [b] Slu – Percentage of sluggish sperm cells**

**[c]Act – Percentage of active sperm cells, [d] TSC – Total sperm cell count**

**[e] Viab – Percentage of viable sperm cells , [f] Hd Df – Percentage of sperm cells with head defect**

**[g]Mid Df – Percentage of sperm cells with mid piece defect**

**[h]Tail Df – percentage of sperm cells with tail defect**

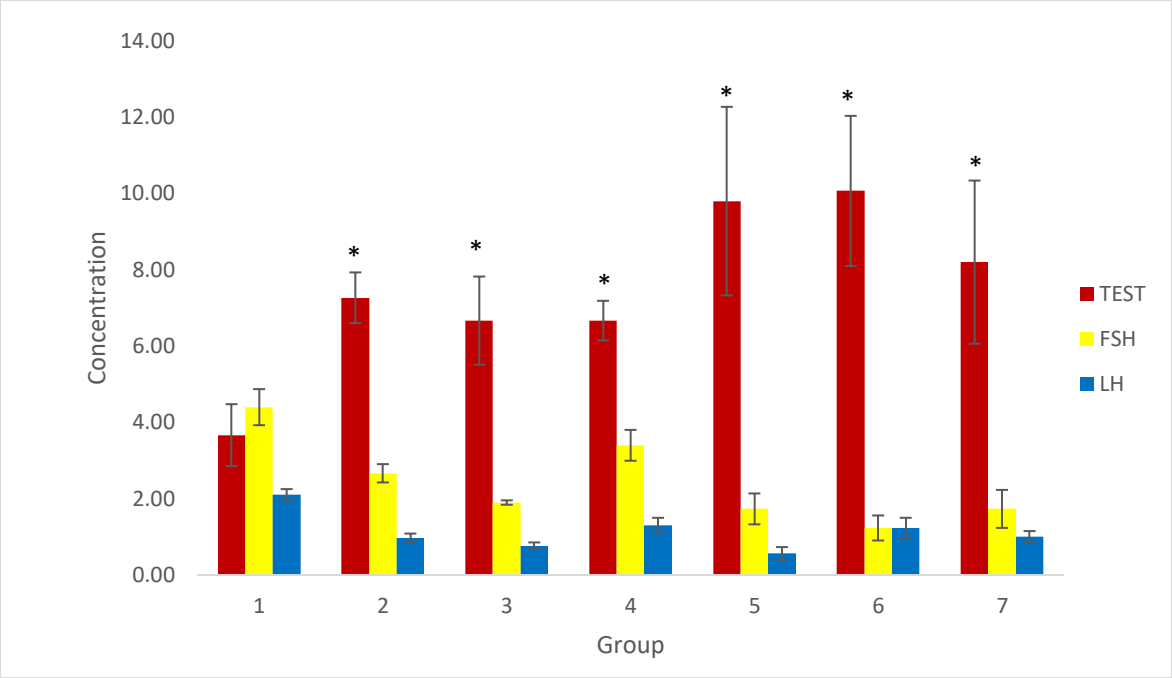


**Fig 1: Graphical representation of testosterone, FSH and LH concentrations of both the experimental group and the normal control group**

### 3.1.4 HISTOLOGY OF THE TESTES

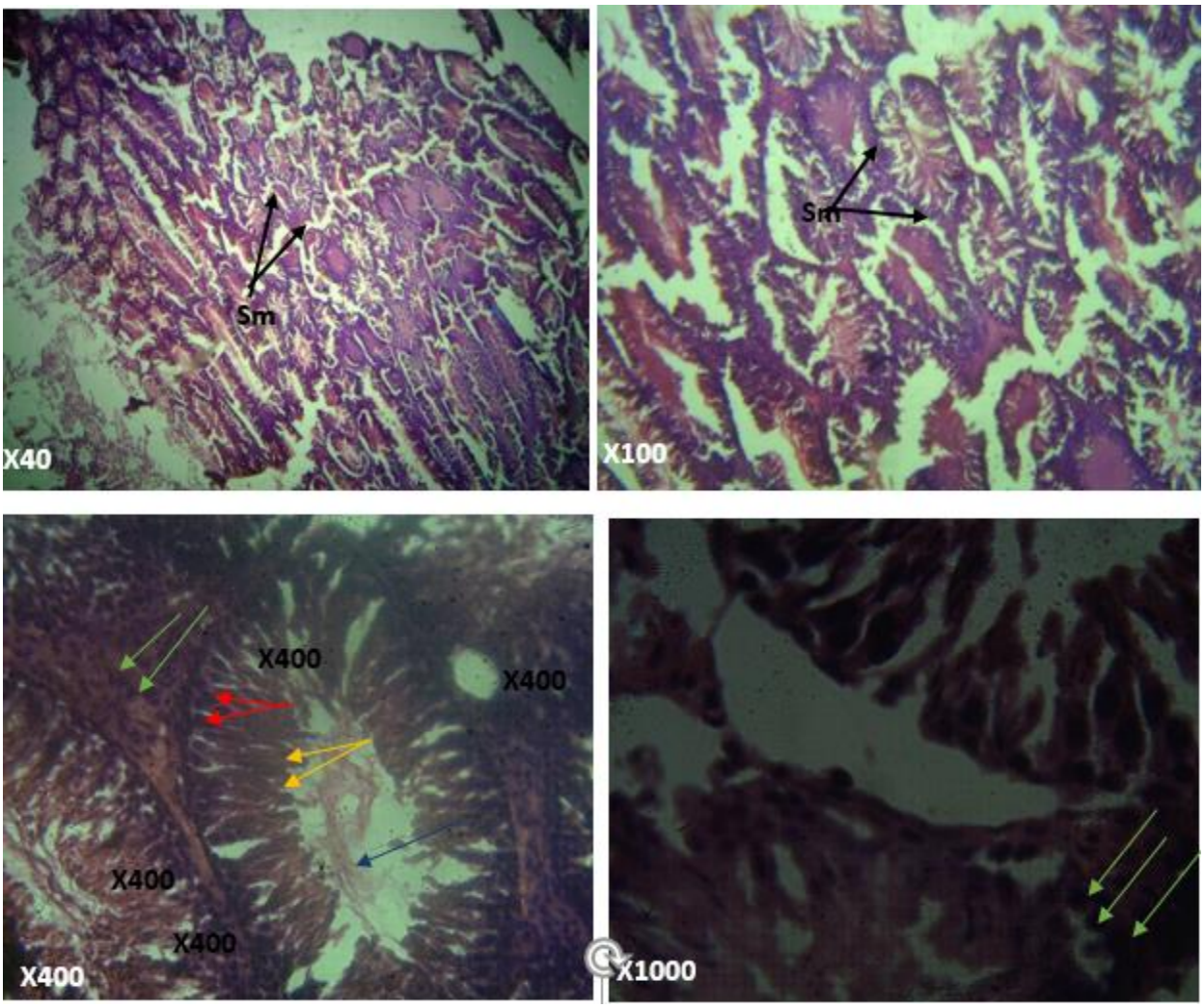


Plate 1: Photomicrograph of normal control(Group 1)

**The testes shows normal histological features with active spermatogenic activities occurring in the seminiferous tubules (Sm). Spermatogonia (red arrow), spermatocytes (yellow arrow), spermatids in the lumen (blue arrow) and interstitial cells of Leydig (green arrow)**

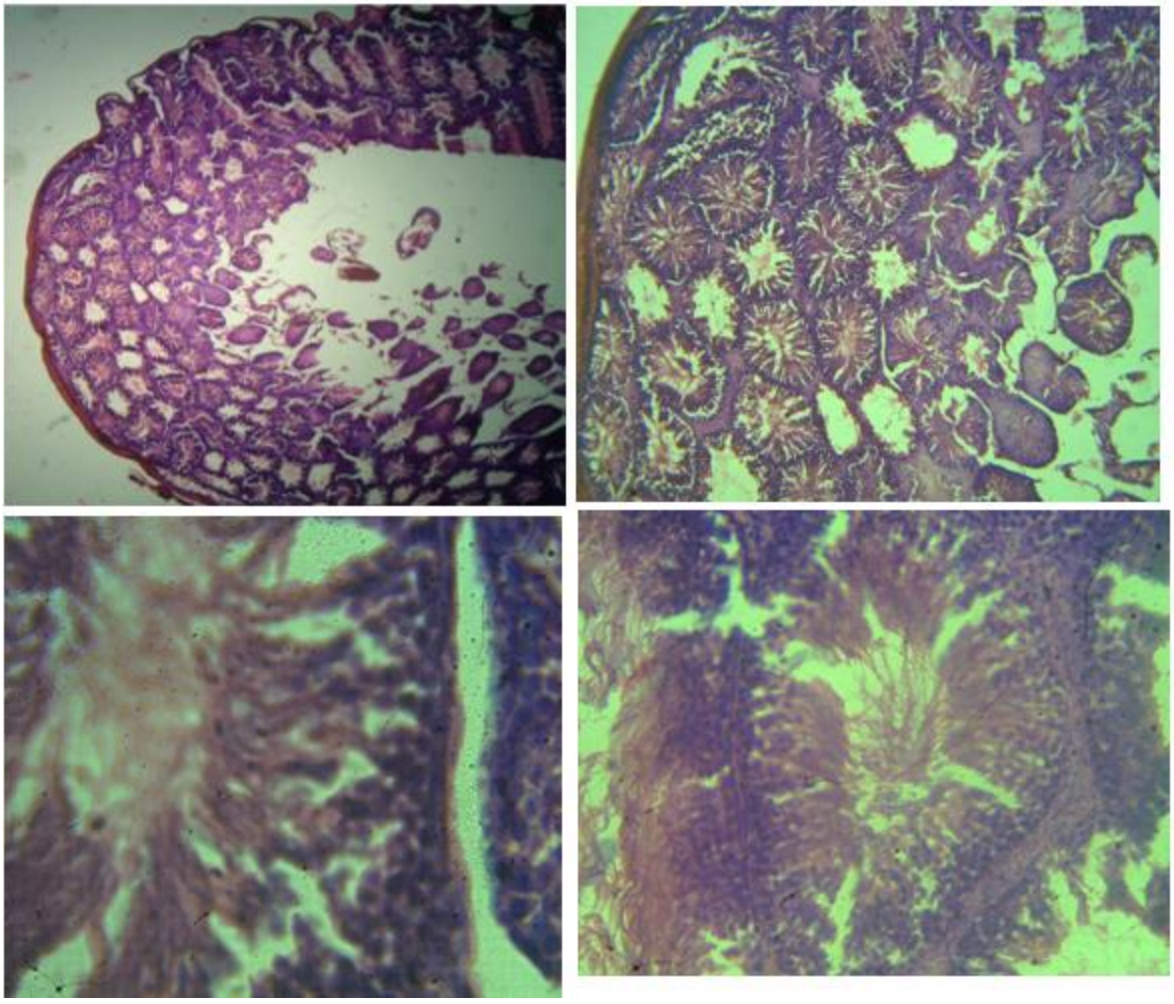

**Plate 2: Photomicrograph of Group 2: 3mg/kg b. wt TP 11 days- The testes shows normal spermatogenic activities occurring in the seminiferous tubules (black arrow). Spermatogonia (red arrow), spermatids in the lumen (blue arrow)**

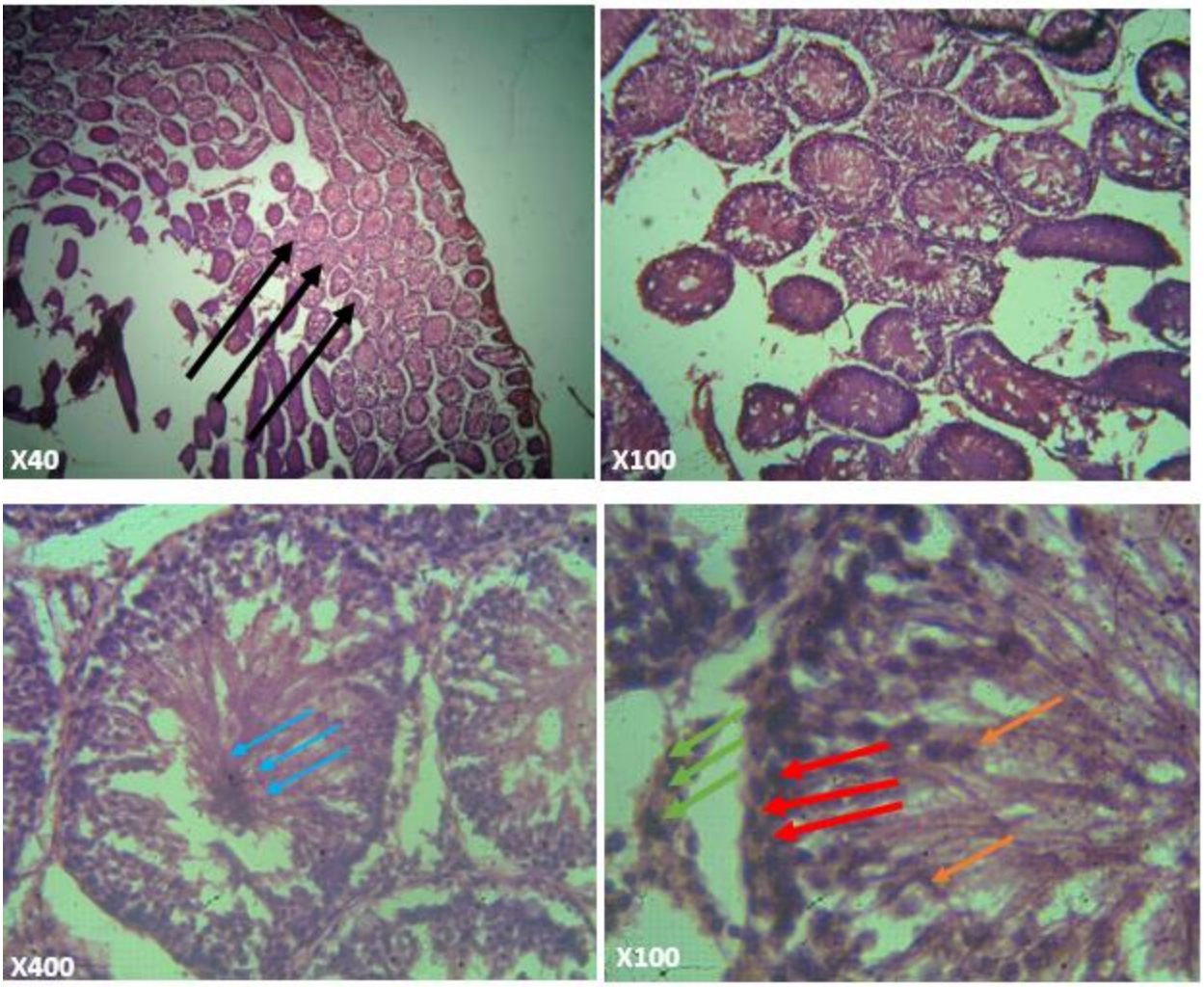


**Plate 3: Photomicrograph of Group 3: 3mg/kg b. wt 22days- The testes shows recovery with normal spermatogenic activities occurring in the seminiferous tubules (black arrow). Spermatogonia (red arrow), spermatocytes (yellow arrow), spermatids in the lumen (blue arrow) and interstitial cells of Leydig (green arrow)**

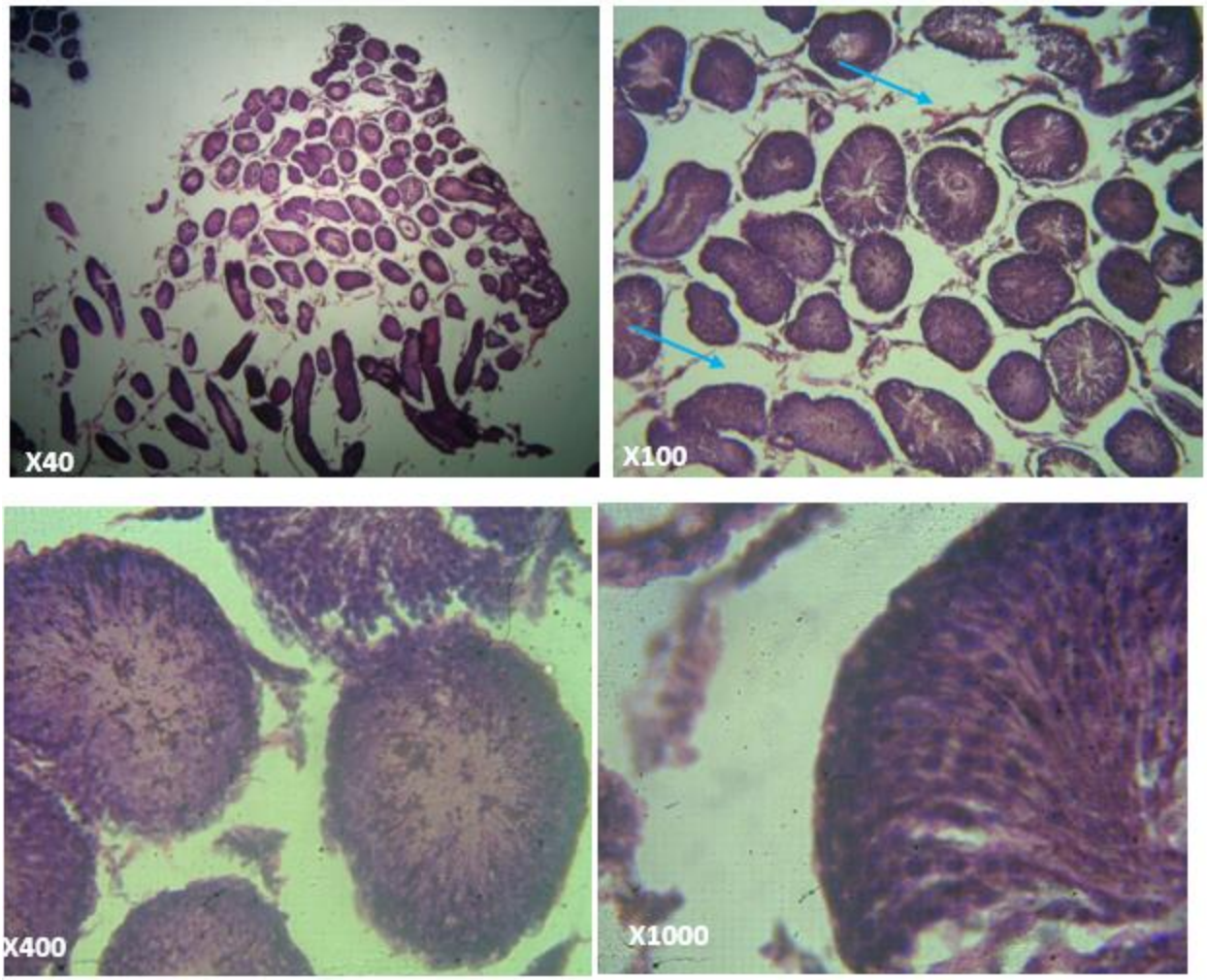


**Plate 4: Photomicrograph of Group 4: 6mg/kg b. wt TP 11days- Seminiferous tubules (Sm) are sparse, there is poor spermatogonia, spermatocytes spermatids differentiation.**

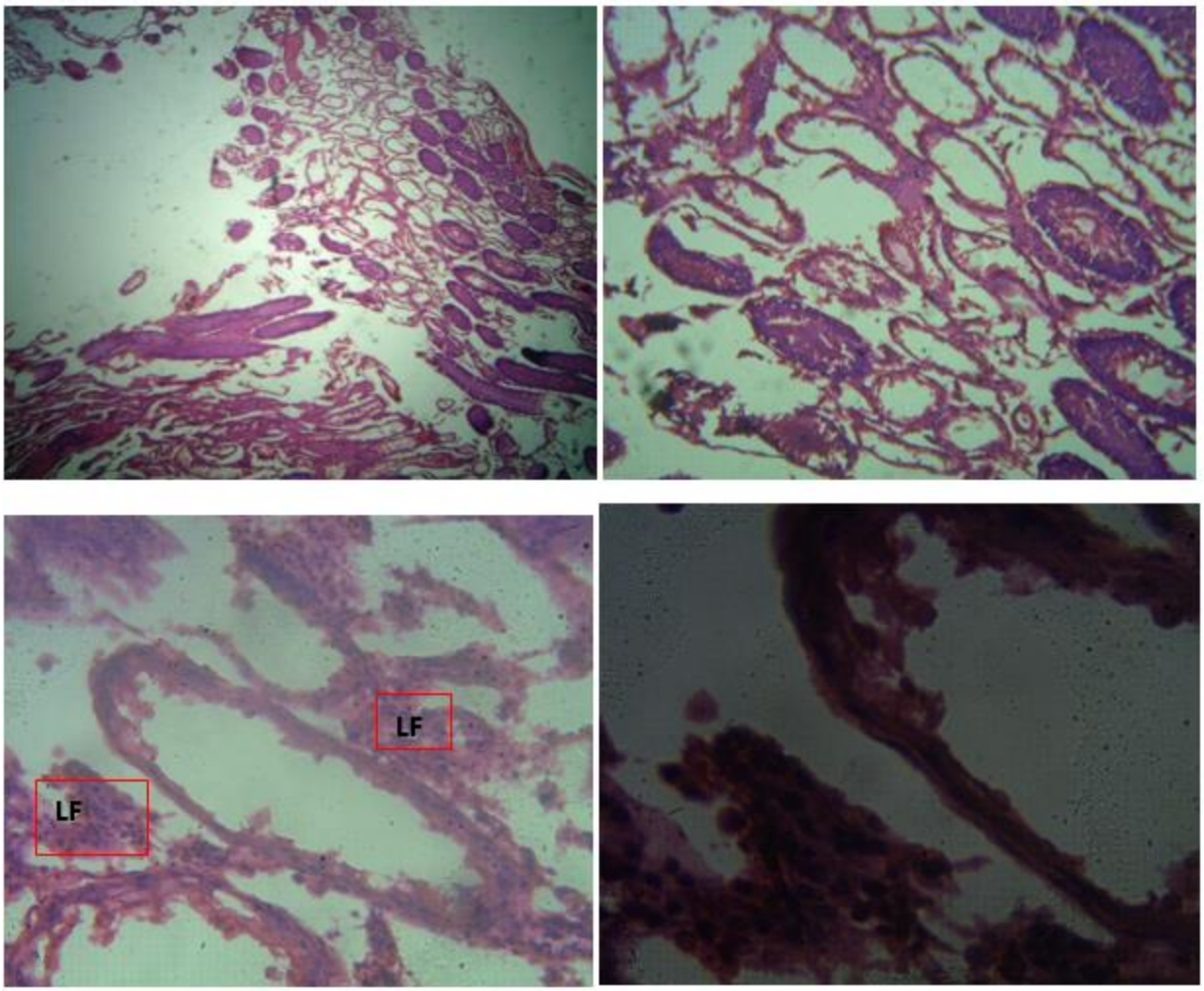


**Plate 5: Photomicrograph of Group 5: 6mg/kg b. wt 22days- The testes shows eroded spermatogenic epithelium indicating arrested spermatogenesis. There is mild lymphocytic cell infiltration [LF] of the interstitium indicating inflammation of tubules (Sm). Interstitial cells of Leydig are not distinguishable (green). There is lack of spermatogonia and spermatocytes and no spermatids in the lumen**

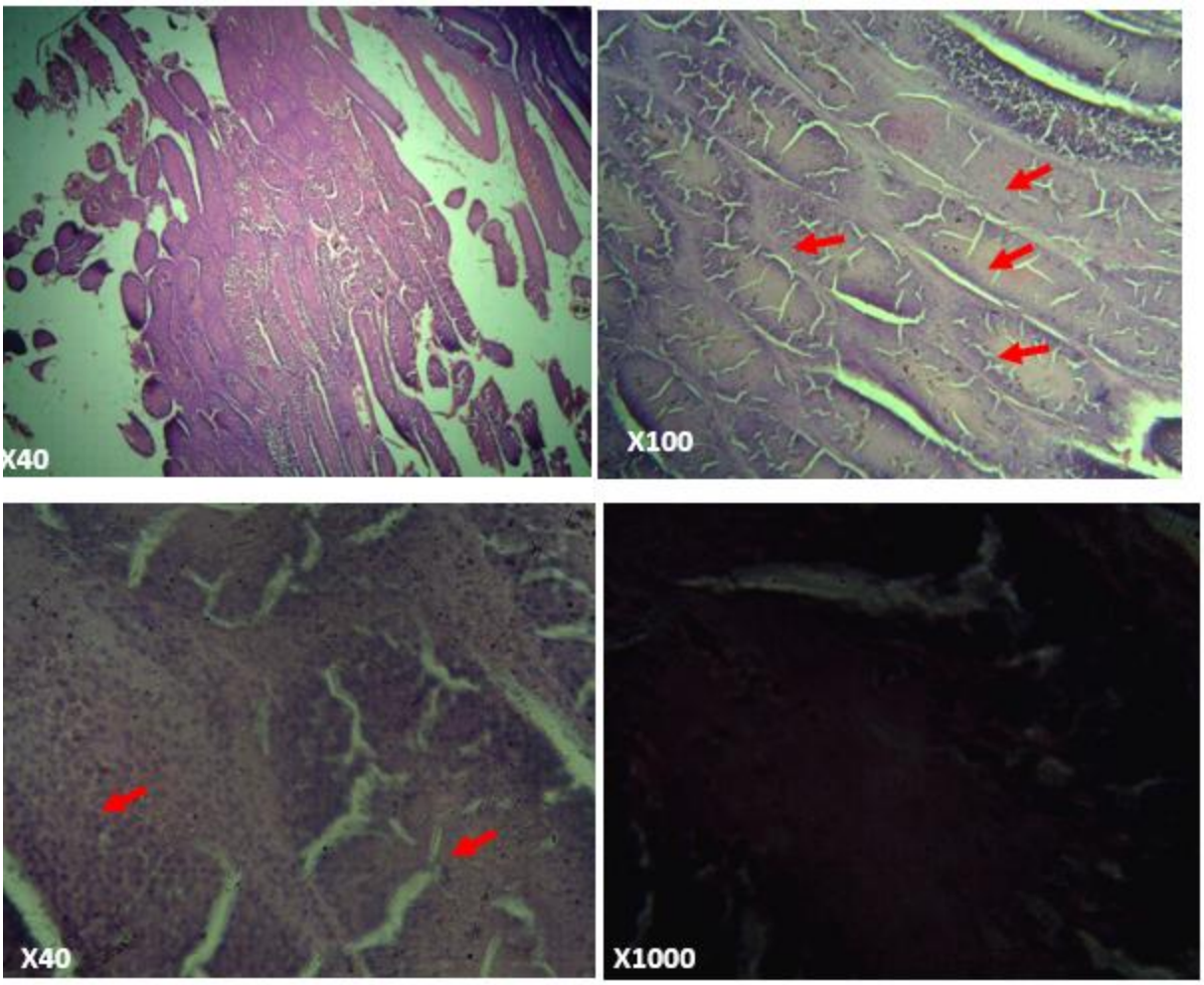


**Plate 6 :Photomicrograph of Group 6, 9mg/kg b. wt 11days- The testes shows hyalinization of the seminiferous tubules (red arrow) indicating distortion of spermatogenic activities. The Spermatogonia, spermatocytes, spermatids and interstitial cells of Leydig are obliterated.**

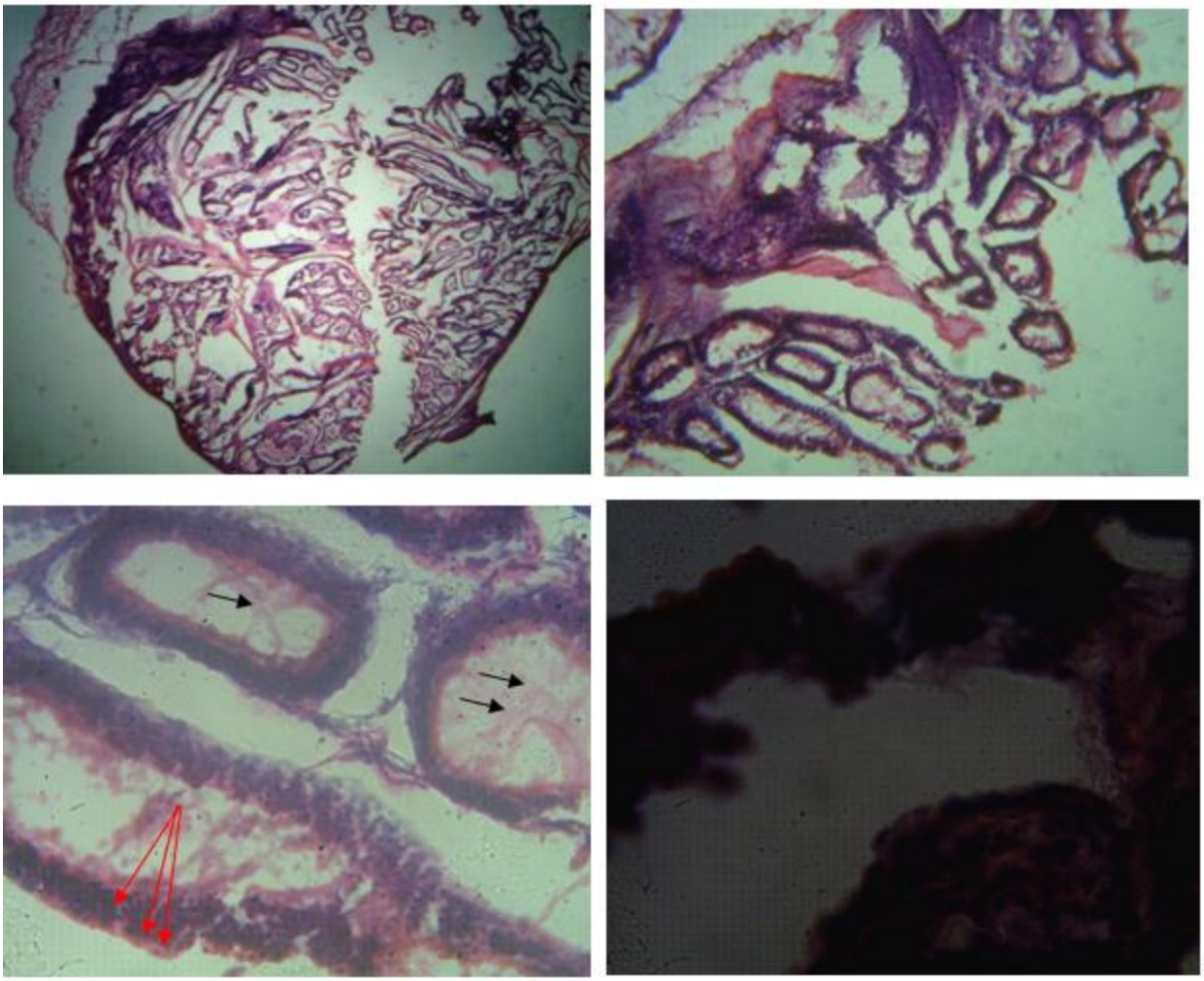

**Plate 7: Photomicrograph of Group 7: 9mg/kg b. wt 22 days- The testes shows altered spermatogenic epithelium with more of spermatogonia (red arrow) indicating distorted spermatogenesis. Spermatids in the lumen are few (black arrow)**

## 3.2 DISCUSSION

### 3.2.1. Semen Motility and Active Cells

The experimental groups treated with testosterone propionate (TP) exhibited a significant ($P$ =.05) decline in sperm motility and active cells compared to the normal control (Group 1). In Group 1, sperm motility was 85.00±13.23%, while in Group 7 (highest TP dose for 22 days), it dropped drastically to 24.67±24.03%. Similarly, the percentage of active sperm cells decreased from 73.33±11.55% (control) to 4.67±4.16% (Group 7). Impaired sperm motility is a recognized consequence of prolonged exogenous testosterone administration. This hormonal intervention can negatively impact the prostate gland, a key organ responsible for producing seminal fluid. Consequently, the provision of essential nutrients and the maintenance of an optimal pH environment for spermatozoa are compromised. The observed enlargement of the prostate following testosterone propionate treatment further suggests a disruption of its normal physiological function, ultimately contributing to poor sperm motility. This finding aligns with the observations of Yassin et al. (2025) [10], who reported that exogenous testosterone suppresses spermatogenesis by reducing follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels, thereby leading to impaired sperm motility. Similarly, Silveira et al. (2023) [12] have documented the detrimental effects of long-term testosterone administration on sperm function. Furthermore, the epididymis plays a crucial role in the acquisition of sperm competence, facilitating maturation, transport, and storage. Spermatozoa produced in the testes are initially immature and lack the capacity for effective fertilization. Their transit through the epididymis, an anatomically coiled structure connecting the seminiferous tubules to the vas deferens, is essential for undergoing critical maturational changes.

### 3.2.2. Sluggish and Dead Cells

A marked increase in sluggish and dead sperm cells was observed across the TP-induced groups. In Group 1, dead sperm cells were 15.00±13.23%, whereas in Group 7, the percentage rose to 75.33±24.03%. The increase in sluggish sperm suggests a deteriorating sperm quality, which has been previously linked to testosterone therapy-induced gonadal suppression [9]. Due to excessive administration of testosterone propionate generates a high activity of oxidative stress which affects the mitochondria of the cells resulting from the release of reactive oxygen species causes slowing celleluar activities and apotosis of the spermatozoa[16]

Prolonged administration of Tp triggers inflammatory cytokine leading to cellular damage and cellular fatigue (spermatozoa)

### 3.3.3. Total Sperm Count and Viability

The total sperm count (TSC) and sperm viability followed a decreasing trend. The control group had a TSC of 113.33±31.53 $\times 10^6$ cells/mL, but in Group 7, it plummeted to 25.00±4.58 $\times 10^6$ cells/mL. Viability also reduced from 94.33±2.89% (control) to 86.67±5.77% (Group 7). The results obtained from this study shows that testosterone propionate exhibit the negative effects

upon the cytosolic cells and the leydig cells, which are responsible for the production or synthesis of spermatozoa due to its direct effect on the hypothalamic-gonadal-axis, that negatively affect the anterior pituitary gland for the release of LH and FSH. The low level of LH and FSH, give a negative impact upon the testes and its cells to act positively for adequate reproduction of spermatozoa,in addition TSC and viabiulity decrease drastically resulting from alteration in epididermal function which is responsible for the storage of matured sperm cells being affected by the testposteropne linked to low level of FSH, the negative activity of the excess TP given to group 4-7 directly disrupts the sperm maturation process and reduces sperm viability as shown in the results.Prolonged administration of these TP leads to testicular athropy causing impairment of sperm production and viability as shown in this study. According to Agarwal et al. (2021), [13], testosterone therapy leads to oligospermia due to suppression of gonadotropins. study

### 3.3.4. Testosterone, LH, and FSH Levels

A paradoxical trend was observed: testosterone concentration increased significantly ((*P* =.05)) in TP-induced groups, with Group 7 showing 8.20±0.79 ng/mL compared to 3.67±0.81 ng/mL in the control. Conversely, FSH and LH levels were significantly lower in TP-treated groups, indicating HPG axis suppression, confirming Yassin et al. (2025).[10] findings that exogenous testosterone inhibits the hypothalamic-pituitary-gonadal axis, reducing endogenous FSH and LH levels. These findings suggest that exogenous hormone regulation, leads to impaired spermatogenesis and potential infertility. The suppression of LH and FSH highlights the negative feedback effect of exogenous testosterone on gonadotropin secretion. Testosterone concentration seems to be elevated due to the prolonged administration of this androgen as observed in the experimental group when compared to the normal control group. The results reveals that FSH and LH concentration are low resulting from the activity of excess exogenous TP which suppresses the physiological activities of the hypothalamic pituitary gonadal axis causing the pituitary gland to synthesize adequate amount of FSH and LH as reported in our study. It is possible that Architectural destruction of the testicular integrity caused by administration of TP as seen in the Plate 4-7 affects FSH receptors and LH receptors because these receptors are located on sertoli cells within the seminferal tubules promoting spermatogenesis. Therefore damge of this tubule affects the functioning of the sertoli cells which then destroys the potentiality of the FSH receptor leading to prevention of sperm production. LHR is also directly affected when the leydig cells that harbour the LH receptor resulting to functionality of the endogenous testosterone causing synthesis to drastically reduce because the LH receptor cannot function maximally to trigger leydig cells for testosterone production.

### 3.3.5. Histological Alterations

Testicular histology showed normal spermatogenesis in the control group as shown in plate 1, whereas testosterone-treated groups exhibited varying degrees of degeneration and structural distortions such as hyalinization of seminiferous tubules and depletion of spermatogenic cells, especially in the high dose as shown in plate 7 with 9mg/kg b.wt of testosterone propionate for 22 days, this is similar to findings by Awarajih, U. C et al., (2024) [14]. Plate 1 photomicrograph demonstrated normal histological appearance by presenting normal seminoferal tubule with adequate spermatocyte as well as spermatozoa, no architectural destruction of seminiferous studies as reported in this study. The photomicrograph of plate 2 with a lower concentatrion presented accurate morphological appearance pf the seminiferous tubule, haboring normal spermatozoa as reported in this study. From our results in plate 4 the photomicrograph shows poor spermatogonia, spermatiocytes in the SM tubules due to high dose of TP affecting the Seminoferal tubules where sperm production occur. The photomicrograph of plate 5 in our studies clearly shows that the epithelial lining of the seminiferal tubules eroded with the leydig cells altered beyond microscopic recognition, no spermatogonia and spermatocyte spotted resulting from architectural destructon or damage of the testicular tissue. However plate 6 photomicrograph with testosterone propionate concentration 9mg/kg indicates hyalinization of the seminiferal tubulkes linking to lacking of spermatogenic,spermatogonia spermatocyte activities of the testes due to Total alteration of the testes of the leydig cells as shown in this study. Photomicrograph of plate 7 with a highest dose of 9mg/kg for 22 days indicates disorganization or shading (desquamation) of spermatogenic cells within the seminiferal tubules. These observations are consistent with previous studies examining the impact of exogenous testosterone on testicular morphology. For instance, Ježek et al. (1993) reported that high doses of testosterone propionate in rats led to changes in morphology and length of seminiferous tubules.[17] This strengthens the interpretation that exogenous testosterone administration induces structural alterations within the testes.

## 4. Conclusion

In summary, this study clearly shows that giving male rats testosterone propionate causes a significant decrease in sperm quality, a reduction in FSH and LH hormones, and changes in the structure of their testicles. These results support what other research has found about the negative effects of extra testosterone on male reproductive health. The drop in FSH and LH suggests that the body's own testosterone production is being turned down, which can lead to poor sperm production and possibly infertility. These animal findings agree with reports from human studies that show testosterone therapy might contribute to fertility problems in men. For future work, it is important to study if these negative effects go away when testosterone treatment stops. Also, more research on how testosterone damages the testicles at a basic level is needed. This knowledge can help in finding ways to lessen the bad effects of using extra testosterone on male reproduction.These observations are consistent with previous studies examining the impact of exogenous testosterone on testicular morphology.

**ACKNOWLEDGEMENT**

My acknowledgement goes first to God almighty, for He has been my all sufficiency, my pillar and my rock at all times. I am grateful to ,Prof C.C Monago-Ighorodje, a mother indeed and Prof D.E Peters for all their support, constructive criticism and counsel throughout the course of this research work.

My sincere appreciation goes to my family, particularly my wife and my children for their support and assistance throughout the period of this research. My brothers, Dr Eruotor Ben, Engr. Dr Moses, Act. Eruotor James. I owe a depth of gratitude for their encouragement. I also appreciate my Co-authors Dr Ezendiokwere Emmanuel and Prof. Oghenemavwe, Ese Loveday for the immeasurable role they played in the course of this research work. I also appreciate Miss Kekachi Ekpendu for her immense contribution to the completion of this research work.

My gratitude also goes to the staff of Divic Medical Laboratory, University of Port Harcourt Teaching Hospital for their efforts and commitment towards the completion of this research work.

**COMPETING INTEREST**

Authors have declared that no competing interest exist.

**AUTHOR'S CONTRIBUTION**

Eruotor O.H designed the research work, performed the biochemical analysis, semen analysis and the write up of the work

Ezendiokwere, O. E designed the study and performed the statistical analysis of the work.

Oghenemavwe, Ese Loveday performed the histological interpretation of the histological slides.

## CONSENT

It is not applicable

## ETHICAL APPROVAL

The experimental protocol of the present study was thoroughly reviewed and approved by the University of Port Harcourt ethical Committee with Reference no: UPH/CEREMAD/REC/MM98/048, University of Port Harcourt, Choba, Rivers State, Nigeria

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