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ORIGINAL ARTICLE

Impact of daily administration of teriparatide on the histological pattern of testicular structure in adult male rats

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ABSTRACT

Aim Teriparatide, a synthetic analog of the active fragment of human parathyroid hormone (PTH 1–34), is a clinically approved anabolic treatment for osteoporosis. While its bone-forming capacity is well-documented, evidence regarding its systemic safety, particularly in relation to male reproductive health, remains limited. This study was designed to investigate the histological consequences of prolonged teriparatide exposure on testicular architecture in adult male rats, addressing a critical gap in its toxicological profile.

Methods Twenty adult male albino rats were randomly allocated into two groups. The control group received subcutaneous saline (1 mL/kg/day), and the treated group was administered teriparatide (10 µg/kg/day) daily for 60 days. Testes were collected post-euthanasia, fixed in formalin, and processed for histological examination. Spermatogenic activity was assessed using Johnsen's scoring system, and morphometric parameters, including seminiferous tubule diameter and epithelial height, were quantified. Statistical significance was determined using the Mann–Whitney U test ($p \leq 0.05$).

Results Histological findings revealed preserved cytoarchitecture with active spermatogenesis in control testes. The teriparatide-treated group exhibited significant degenerative changes, including epithelial detachment, interstitial edema, vascular congestion, and Leydig cell necrosis. Johnsen's scores were significantly reduced in the treated group (median = 4.5; IQR=1.5) compared to controls (median = 8.0; IQR=2.0; $p<0.001$). Seminiferous tubule diameter and epithelial height were markedly decreased in the treated animals.

Conclusion The findings suggest that prolonged teriparatide exposure may impair spermatogenesis and alter testicular structure. Re-evaluation of its reproductive safety is warranted, particularly for males of reproductive age.

Keywords: histopathology, reproductive toxicity, spermatogenesis, teriparatide, testis

INTRODUCTION

Osteoporosis represents a progressive metabolic skeletal disorder characterized by decreased bone mass and microarchitectural deterioration of bone tissue, ultimately predisposing individuals (particularly postmenopausal females) and elderly males to fragility fractures and associated morbidity (1,2). Among the Food and Drug Administration (FDA)-approved therapeutic agents for severe osteoporosis (2), teriparatide, synthetic recombinant fragment of human parathyroid hormone (PTH 1–34), has gained clinical prominence due to its potent anabolic effect on osteoblasts, in contrast to antiresorptive agents such as bisphosphonates (3,4). By stimulating bone formation and improving trabecular connectivity and mineral density, teriparatide has redefined treatment paradigms in osteometabolic care. However, beyond its osteogenic actions, teriparatide also exerts systemic effects by modulating calcium-phosphate homeostasis through enhanced intestinal calcium absorption and renal calcium reabsorption—mechanisms partially mediated via activation of vitamin D metabolism (5,6). These systemic influences raise concerns regarding their potential effects on other physiological systems, particularly those reliant on tightly regulated calcium dynamics, such as the male reproductive axis (7).

The testis, as the central organ in male fertility, exhibits high metabolic demand and functional dependence on both endocrine and paracrine signals. Sertoli cells within seminiferous tubules coordinate germ cell differentiation, while Leydig cells in the interstitial space maintain androgen production (8). Importantly, calcium ions regulate crucial reproductive functions, including spermatogonial maturation, steroidogenesis, and sperm motility (7,9). Hence, agents that alter calcium equilibrium may indirectly compromise testicular architecture and function (8).

Recent experimental evidence has increasingly implicated parathyroid hormone analogs in eliciting testicular pathology via multifactorial pathways. Among these, oxidative imbal-

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ance, compromised microvascular integrity, and interference with endocrine regulatory circuits have been highlighted as plausible contributors to gonadal dysfunction (10,11). Notably, extended exposure to teriparatide in preclinical models has been associated with pronounced histoarchitectural disruptions (most prominently), disorganization of the seminiferous epithelium and morphological degeneration of Leydig cells, thus raising concerns regarding its potential gonadotoxic effects (12,13).

Nonetheless, the reproductive safety landscape of teriparatide remains inadequately delineated, particularly in the context of therapeutic use among males with active fertility potential.

The aim of this study was a critical assessment of the impact of chronic teriparatide administration on testicular histology and spermatogenic outcomes within a controlled rodent model, thereby contributing mechanistic insights into the drug's broader endocrine footprint. By focusing on morphometric indices and degenerative patterns, this research seeks to elucidate whether systemic PTH modulation may pose an unrecognized risk to male reproductive health.

MATERIALS AND METHODS

Materials and study design

This controlled laboratory investigation was executed between March and May 2024 within the Department of Anatomy, College of Veterinary Medicine, University of Mosul, Iraq.

The study was structured to assess the histological implications of prolonged teriparatide exposure on testicular morphology and spermatogenic function in a standardized rodent model.

All procedures adhered strictly to the international guidelines for the ethical treatment of laboratory animals and received a formal approval from the Animal Research Ethics Committee of the College of Dentistry, University of Mosul (Approval No. UOM Dent 24/1029).

Twenty healthy adult male albino rats (*Rattus norvegicus*), aged 6–7 months and weighing between 300–350 grams, were housed under controlled environmental conditions (temperature: 22±2 °C, humidity: 55±10%, 12/12 h light/dark cycle) with unrestricted access to standard pellet feed and water. The animals received standard commercial rodent food while having unlimited access to filtered water. The researchers established these conditions to prevent outside factors from affecting testicular morphology and spermatogenesis while studying teriparatide effects.

The animals were randomly assigned into two equal groups (N=10 per group): Group I (Control) received subcutaneous injections of sterile normal saline (1 mL/kg/day) for 60 days; Group II (Teriparatide-treated) received daily subcutaneous injections of teriparatide at 10 µg/kg body weight for 60 days.

Methods

Teriparatide (recombinant human PTH 1–34) was prepared fresh under aseptic conditions prior to each administration, with dosage individually adjusted based on daily body weight measurements. At the end of the experimental period, animals were anesthetized using a ketamine–xylazine mixture (80 mg/kg and 10 mg/kg intraperitoneally, respectively, in both groups) and euthanized by cervical dislocation. Both testes were carefully dissected, trimmed of extraneous tissues, and fixed in 10% neutral-buffered formalin for no less than 48 hours.

Dose justification and human equivalence. A daily teriparatide dose of 10 µg/kg/day was used because this amount was established through preclinical research to assess the drug's safety outside bone tissue. The FDA-approved body-surface-area (BSA) conversion method (14) allows to calculate the human-equivalent dose (HED). The conversion is based on Km factors (rat Km = 6; human Km = 37), which are species-specific constants representing the ratio of body weight to surface area. Using this approach, the calculated HED was 0.00162 mg/kg, equivalent to a daily dose of about 1.62 µg/kg.

The calculated dose for a 70-kg adult amounts to approximately 113 µg per day. The rat dose was set above the therapeutic human dose of 20 µg/day administered subcutaneously, producing a 5.6-fold higher systemic exposure. This approach was intended to reveal subtle reproductive effects, yet remained within the accepted preclinical safety margins (15,16).

Histological processing and evaluation. Following fixation, samples were dehydrated using graded ethanol concentrations, embedded in paraffin blocks, and sectioned at 5 µm thickness using a rotary microtome (Leica RM2235, Leica Biosystems, Wetzlar, Germany). Sections were stained with haematoxylin and eosin (H&E) for general histopathological evaluation. The structural integrity of seminiferous tubules, germinal epithelium, interstitial tissue, vascular compartments, and Leydig cells was assessed via light microscopy (Olympus CX43, Olympus Corporation, Tokyo, Japan).

To semi-quantify spermatogenic activity, Johnsen's scoring system was employed. Ten seminiferous tubules per animal were randomly selected, and scores were assigned on a 1–10 scale based on the most advanced germ cell type present.

Morphometric parameters, including seminiferous tubule diameter and germinal epithelial height, were measured using ImageJ software (NIH, USA), with calibrated digital images captured from multiple fields.

Statistical analysis

Data were first examined for normality using the Shapiro–Wilk test. Depending on the distribution, results were expressed either as mean ± standard deviation (SD) or median with interquartile range (IQR). Comparative analysis between the two groups was conducted using the Mann–Whitney U test for nonparametric data and Student's t-test for parametric variables. A p-value ≤ 0.05 was considered statistically significant. Group size was set a priori following established guidance for adequately powered morphometric endpoints in rodent studies. A prospective power analysis did not perform, but the observed standardized effects for the main outcomes (seminiferous tubule diameter and epithelial height) resulted in post hoc power values greater than 0.99 at $\alpha=0.05$, which confirmed the adequacy of the selected sample size. Effect sizes and confidence intervals to complement p-value (17) were additionally reported.

RESULTS

Microscopic evaluation of testicular tissue in the Control group (Group I) revealed a well-preserved seminiferous epithelium with active spermatogenesis. Germ cells at various stages—spermatogonia, primary and secondary spermatocytes, spermatids, and spermatozoa—were arranged in an orderly, stratified manner. Sertoli cells were clearly visible at the

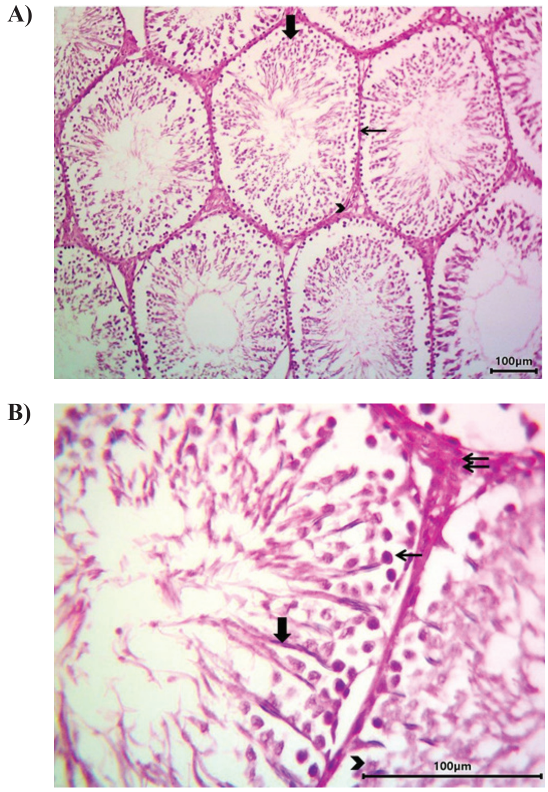

Figure 1. A) Histological section of the testis from a control rat showing preserved seminiferous tubule architecture with active spermatogenesis (arrow). The germinal epithelium is intact and displays an orderly arrangement of spermatogenic cells at various maturation stages (bold-arrow) (100×); B) intact seminiferous tubules with Spermatocytes (arrow), Spermatid (bold-arrow), Sertoli cells (arrowhead) and Leydig cell (double-arrow), (400×). H&E staining

basal compartment of the seminiferous tubules, while Leydig cells appeared structurally intact within the interstitial space. Vascular architecture was normal, and no signs of edema, congestion, or necrosis were observed (Figure 1A and 1B).

Conversely, rats treated with teriparatide (Group II) exhibited substantial histopathological alterations. Seminiferous tubules showed reduced cellular density and severe hypospermatogenesis. Key pathological features included germinal epithelial disorganization, vacuolization, interstitial edema, vascular congestion, and focal necrosis of Leydig cells. Spermatogenic arrest was evident in most tubules, with predominant degeneration of spermatocytes and detachment of the germinal epithelium (Figures 2A–D).

Semi-quantitative grading using Johnsen's scoring system revealed a statistically significant reduction in spermatogenic activity among the treated animals. The median Johnsen score in the control group was 8.0 (IQR=2.0), whereas the teriparatide-treated group exhibited a median score of 4.5 (IQR=1.5). Statistical comparison using the Mann–Whitney U test confirmed that the difference was highly significant ($p<0.001$), indicating disrupted spermatogenesis in response to prolonged teriparatide exposure.

Quantitative morphometric evaluation demonstrated a pronounced reduction in both seminiferous tubule diameter and germinal epithelial height in the teriparatide group compared to controls. The mean seminiferous tubule diameter in the control group was 224.6±8.2 μm, while in the treated group, it significantly decreased to 172.4±6.5 μm ($p<0.001$). The germinal

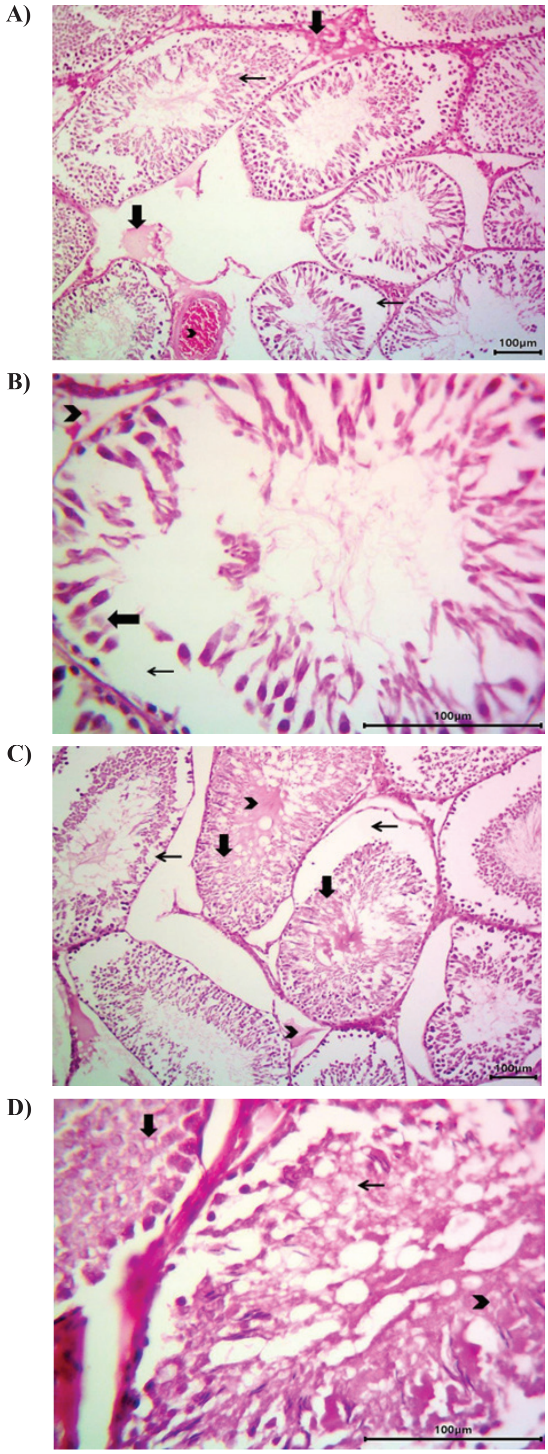

Figure 2. A) Testicular section from teriparatide-treated rats showing necrosis and degeneration of spermatogenesis cells with hypospermatogenesis (arrow), edema between seminiferous tubules (bold-arrow) and congested blood vessels (arrowhead), (100×); B) Higher magnification reveals hypospermatogenesis in the seminiferous tubules (arrow), necrosis and degeneration of the spermatocytes (bold-arrow) and necrosis of Leydig cells (arrowhead), (400×); C) hypospermatogenesis and detachment in the seminiferous tubules (arrow), necrosis and degeneration of the spermatocytes (bold-arrow) and edema (arrowhead), (100×); D) necrosis (arrow) and degeneration (bold-arrow) of the spermatocytes and edema (arrowhead), (400×). H&E staining.

epithelial height was also markedly reduced from 58.7±3.1 μm in controls to 39.2±2.4 μm in the treated rats ($p<0.001$).

These morphometric differences further substantiate the degenerative histological observations and suggest that teri-

Table 1. Morphometric parameters of seminiferous tubules in two groups

Parameter	Mean±SD		p
	Control group	Treated group	
Seminiferous tubule diameter (µm)	224.6±8.2	172.4±6.5	<0.001
Germinal epithelium height (µm)	58.7±3.1	39.2±2.4	<0.001

paratide may compromise testicular cytoarchitecture through direct or indirect mechanisms (Table 1). These histological and morphometric findings are suggestive of endocrine disruption, likely mediated through perturbations in the hypothalamic–pituitary–gonadal axis (Figure 3).

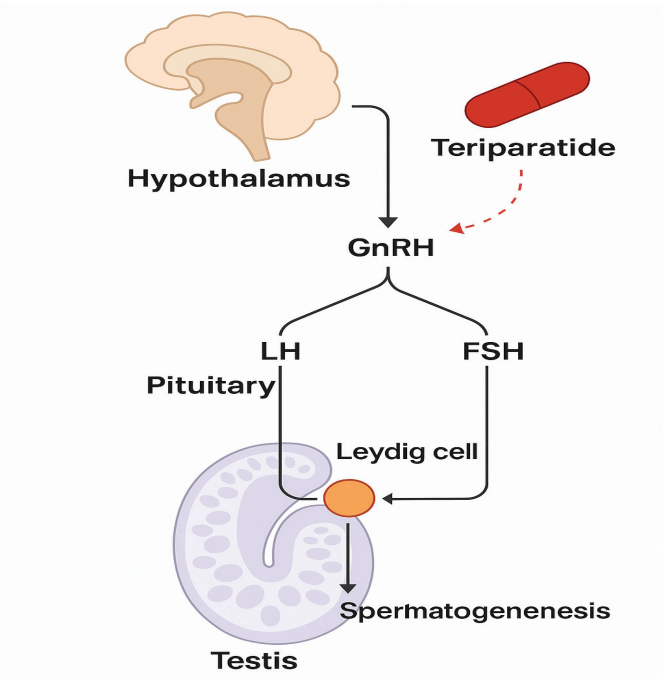

Figure 3. Schematic representation of the hypothalamic–pituitary–gonadal (HPG) axis showing the proposed inhibitory effect of teriparatide on GnRH signalling and downstream gonadotropin secretion, potentially impairing spermatogenesis (Created by the authors)

DISCUSSION

The current study presents compelling histopathological and morphometric evidence indicating that prolonged teriparatide administration induces marked disruptions in testicular architecture and spermatogenic function in adult male rats. These findings align with a growing body of preclinical data that suggest parathyroid hormone analogues may exert off-target effects beyond the skeletal system, particularly on hormonally regulated organs such as the testes (11,12). The 60-day exposure window was chosen to capture one complete spermatogenic cycle in rats. The seminiferous epithelium cycle lasts 12.8 days so a 58–60-day study period allows to measure chronic effects across the entire germ cell lineage which enhances the translational value for male fertility risk assessment (19). Previous research using rodents established that two months of experimental time in rodents equals several years of human exposure thus enabling the assessment of long-term teriparatide effects on testicular structure (20,21).

In the control group, the preservation of seminiferous tubule integrity, orderly germinal epithelium, and active spermatogenesis confirmed normal testicular function. Conversely, the teriparatide-treated group exhibited extensive degeneration, including epithelial detachment, interstitial edema, vascular congestion, and Leydig cell necrosis, features that collectively signal impaired spermatogenic progression and disrupted androgenic activity. The statistically significant reductions in both Johnsen scores and seminiferous tubule dimensions support these morphological observations and reinforce the hypothesis that teriparatide may interfere with the homeostatic regulation of male fertility. The underlying mechanisms through which teriparatide induces such testicular damage may be multifactorial. One plausible pathway involves its known effect on systemic calcium-phosphate metabolism, which may secondarily impact the hypothalamic–pituitary–gonadal (HPG) axis (22). Teriparatide stimulates calcium absorption via the gut and renal reabsorption, potentially altering neuroendocrine feedback loops involving gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), and follicle-stimulating hormone (FSH) (5,6). It is plausible that chronic perturbations within the hypothalamic–pituitary–gonadal (HPG) axis, potentially induced by teriparatide, may disrupt upstream neuroendocrine signals emanating from the hypothalamus and pituitary gland. Such dysregulation may ultimately compromise testicular steroidogenesis and germinal cell maturation. This hypothesis gains support from our histological observations, which revealed marked Leydig cell degeneration—an outcome that directly implicates diminished intratesticular testosterone synthesis. In turn, impaired androgenic support could destabilize Sertoli cell function, thereby attenuating the microenvironment required for spermatogenic progression (23,24).

The observed morphometric problems match the effects of low androgen because research has shown that decreased testosterone production leads to smaller seminiferous tubules and reduced epithelial cell height (25,26). Research studies conducted with rodent models show that spermatogenesis problems and germ cell death are associated with oxidative stress biomarkers malondialdehyde (MDA) and superoxide dismutase (SOD) (27, 28).

Moreover, oxidative stress may constitute an auxiliary mechanism contributing to testicular damage. The metabolic shifts triggered by prolonged teriparatide exposure could elevate intracellular reactive oxygen species (ROS), undermining cellular viability and promoting germ cell apoptosis (11). These mechanistic inferences align with prior findings by Li et al. (13), who reported structural disruption and reduced spermatogenic indices following extended administration of PTH analogs. Likewise, it was demonstrated that calcium–phosphorus dyshomeostasis could impair Leydig and Sertoli cell physiology, leading to subfertility (29). It was further emphasized that exogenous anabolic hormones may induce interstitial congestion and inflammatory infiltration within the testis—hallmarks mirrored in our treated cohort (30).

The convergence of these findings raises the possibility that teriparatide, when administered chronically, may function as a pharmacological endocrine-disrupting compound (EDC). This potential classification is particularly salient in clinical contexts involving younger male patients, where long-term reproductive implications must not be overlooked.

It is the first experimental study which evaluated how daily teriparatide treatment affects testicular tissue structure in adult rats. Our research introduces new findings about teriparatide reproductive safety through complete morphometric and histological assessments which differ from previous studies that concentrated on skeletal results. The study's original findings demonstrate why healthcare providers should track parathyroid hormone analogue side effects that occur outside the skeleton when patients use these medications for extended periods. There are some limitations of the study. Despite the robustness of histological and morphometric data, the study is constrained by the absence of serum hormonal profiling (e.g., testosterone, LH, FSH) and oxidative stress biomarkers (e. g., MDA, SOD), which limits mechanistic inference. Furthermore, the use of a single dose and fixed exposure duration precludes evaluation of dose-dependent or reversible effects. Finally, extrapolation to human physiology remains tentative, necessitating further translational research to validate these findings.

In conclusion, the present investigation established a histomorphological link between prolonged teriparatide administration and testicular degeneration in adult male rats. The observed alterations, including epithelial atrophy, Leydig cell necrosis, and reduced seminiferous tubule dimensions, strongly implicate HPG axis dysregulation and androgenic insufficiency. Accordingly, these findings urge clinicians to adopt a cautious approach when prescribing teriparatide to individuals of reproductive potential and emphasize the necessity for longitudinal evaluation of its gonadotoxicity. Further research should include hormonal profiling and molecular markers to clarify the mechanisms underlying testicular damage. Evaluating dose dependency, treatment reversibility, and long-term reproductive safety, especially in clinical settings, will be crucial for guiding therapeutic use of teriparatide in reproductive-age individuals.

Author Contributions: OMA, conceptualization, methodology, experimental work, data analysis, manuscript writing. GAT, histological evaluation, supervision, manuscript review and editing.

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TRANSPARENCY DECLARATION

Conflicts of interest: None to declare.

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