Evaluation of Physiological Parameters in Male Rats with Dexamethasone-Induced Osteoporosis: Vitamin D, Parathyroid Hormone, Calcium, Phosphorus, and Reproductive Hormones

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ABSTRACT

This study examined how dexamethasone, a commonly used corticosteroid, affects key biochemical, hormonal, and physiological functions in male rats. We focused on its impact on body weight, kidney function, mineral balance, and hormonal regulation. Our results showed that dexamethasone caused significant weight loss and impaired kidney function, evidenced by increased levels of urea, creatinine, and uric acid, along with reduced total protein. Additionally, dexamethasone disrupted mineral metabolism by lowering vitamin D and calcium levels while raising parathyroid hormone and phosphorus, indicating potential bone metabolism disturbances. Hormonal analysis revealed elevated follicle-stimulating hormone and luteinizing hormone but reduced testosterone, suggesting interference with reproductive hormone regulation. These findings demonstrate that dexamethasone induces widespread physiological disruptions, highlighting the need for careful monitoring of these parameters during corticosteroid treatment to minimize adverse effects.

1. Introduction

Glucocorticoids are commonly described for their potent anti-inflammatory as well as immunosuppressive impacts [1]. They play an important role in managing conditions as asthma, allergic reactions in addition to autoimmune diseases. Despite their therapeutic benefits, the use of glucocorticoids for prolonged period is a main cause of secondary osteoporosis, significantly raising patients' risk of fractures and associated complications [2]. Osteoporosis caused by glucocorticoid use develops in two distinct phases. The first is an early and rapid phase characterized by a marked decrease in bone mineral density, largely because of increased bone resorption. The second is a slower and more persistent phase, in which bone mineral density decreases further, mainly due to inhibition of bone formation. While glucocorticoids exert indirect influences on bone metabolism, their direct actions on osteoblasts, osteoclasts, and osteocytes have a pivotal role in the disease process [3].

Glucocorticoids alter the quantity, differentiation, and function of osteoblastic cells. Glucocorticoids stimulate osteoclast formation by upregulating the expression of receptor activator of nuclear factor-κB ligand as well as colony-stimulating factor-1, while downregulating osteoprotegerin. Nevertheless, the main effect of glucocorticoids on bone is the inhibition of bone formation. This inhibition results from a reduction in osteoblast numbers due to a diversion in mesenchymal cell differentiation away from the osteoblastic lineage, coupled with an increase in the mortality of mature osteoblasts. Glucocorticoids diminish the activity of residual osteoblasts both directly and indirectly by inhibiting the expression of insulin-like growth factor I. The initial bone loss following glucocorticoid exposure is likely attributable to the stimulation of bone resorption. In the long run, stopping bone formation will lead to less bone remodelling and a higher chance of breaking bones [4].

Glucocorticoids lead to osteoporosis, which elevates the risk of bone fracture and makes a contribution to significant death [5]. Glucocorticoids can lead to localised changes in bone microarchitecture, causing micro-lesions that diminish bone strength. It has been shown that changes in regional microarchitecture are linked to the use of glucocorticoids [6]. Osteoporosis is a challenge with bone remodelling caused by an imbalance in the activity of osteoblasts and osteoclasts. This imbalance is affected by factors like inflammation and oxidative stress [7, 8]. Osteoporosis is a long-term bone disease recognized by decreased bone mineral density, deterioration of bone architecture, and heightened vulnerability to fractures. Glucocorticoids are responsible for one of the major serious health issues, after cardiovascular diseases [9-11].

Even though we know how glucocorticoids cause osteoporosis in general, we still lack enough information about the specific changes that occur in the body, especially in male models

that use dexamethasone. It is important to understand these changes in order to improve clinical observation, pathological changes, and the creation of specific treatments that may prevent bone loss throughout corticosteroid therapy.

So, the idea of this study is to have an insight into the way dexamethasone-induced osteoporosis affects the body's hormonal systems as well as the clinical changes in the bone tissue of male rats. By making these changes easier to understand, the study provides the information that could be a better guide to manage bone diseases linked to glucocorticoids.

2. Materials and Methods

2.1. Ethical approval

The study was approved by Ethics Committee of the Faculty of Veterinary Medicine at the University of Basrah and all treatments strictly adhered to ethical standards governing the humane care of animals (No. 48/37/2024).

2.2. Animals model and experimental design

Twenty-eight adult male rats, aged 8–10 weeks and weighing 209-231g were randomly divided into two equal groups, each consisting of fourteen rats. The animals were obtained from the Central Animal House Facility of Faculty of Veterinary Medicine, University of Basrah, which maintains a specific pathogen-free status. Upon arrival, rats were acclimatized for one week before the start of experiments to minimize stress-related variability.

The animals were housed in standard polycarbonate cages measuring (40 × 25 × 20 cm) with standard bedding material, with a maximum of 5 animals per cage to prevent overcrowding. Animals were housed under standardized conditions with a 12-hour light/dark cycle [12-16], ambient temperature of 22 ± 2°C, and relative humidity of 50-60%. Rats had ad libitum access to standard laboratory chow with unrestricted access to water. Twice a week, the cages were washed and sterilized, the bedding was changed to keep clean and comfort the animals.

The groups and their respective treatments were as follows:

1. Control Group: Rats in this group received an intraperitoneal injection of normal saline (0.9%) at a volume equivalent to the treatment group, once daily for 28 consecutive days.

2. Second Group: Rats in this group were administered dexamethasone sodium phosphate intraperitoneally at a dose of 0.5 mg/kg body weight once daily for 28 days. The injection volume was adjusted according to individual body weight per administration. This dosage was chosen by the authors as a relatively high exposure level to detect the possible biological

alterations to investigate biological responses under elevated exposure conditions rather than to replicate a previously reported therapeutic dose and to evaluate potential subacute toxicological within the 28-day experimental period while remaining within a non-lethal and ethically acceptable range.

All injections were performed between (9:00 and 10:00 am) to reduce variability related to circadian rhythms.

2.3. Body weight, Blood Collection

The body weight of each rat was measured before and after the experimental period. Blood samples were obtained via cardiac puncture with a sterile 23-gauge needle and 5 mL syringe, withdrawing around 5 mL from the left ventricle for analysis of biochemical and hormonal parameters.

2.4. Biochemical and Hormonal Analyses

Serum samples were analyzed to determine a range of biochemical and hormonal parameters relevant to bone metabolism and reproductive function.

Vitamin D (25-hydroxyvitamin D) levels were measured using a specific Rat 25-Hydroxy vitamin D ELISA Kit, following the manufacturer's protocol from (MyBioSource, USA). Parathyroid hormone (PTH) concentrations were measured using a rat-specific ELISA kit, employing sandwich ELISA with detection at 450 nm. following the manufacturer's protocol from (LS Bio, USA).

Serum calcium and phosphorus levels were assessed using standard colorimetric assays in which performed with commercially available diagnostic kits (BIOLABO, France), Calcium was measured by the o-cresolphthalein complexone method, and phosphorus was measured by ammonium molybdate method.

Urea, creatinine, and uric acid levels were evaluated enzymatically by using automated analyzers and commercial kits from (AGAPPE, India), following to all the manufacturer's instructions.

A commercially available assay kit from (Sigma-Aldrich, USA) to measure serum total protein levels by using the biuret method.

Reproductive hormones, involving follicle-stimulating hormone (FSH), luteinizing hormone (LH), as well as testosterone, were determined using rat-specific ELISA kits from (BT

LAB, China). Levels of hormones were evaluated according to kit protocols, at absorbance 450 nm.

To ensure accuracy all assays were achieved in duplicate, The standard curves were generated for analyte concentrations quantification.

2.5. Bone Histomorphology

After euthanasia, Femur bones were collected and kept in 10% formalin. Decalcification of bones were performed, then paraffin-embedded, and sectioned for histological examination. Finally, hematoxylin and eosin (H&E) staining was performed to assess any trabecular bone microarchitecture.

2.6. Statically Analysis

Statistical analysis was achieved by using SPSS version 26.0. The data were expressed as mean ± standard deviation (SD). An independent t-test was used in order to measure the differences between the two groups, the p-value (≤ 0.05) was considered as statistically significant.

3. Results

3.1 Effect dexamethasone on body weight as well as body weight gain in male rats

The results reveled in (Table 1) indicated that the administration of dexamethasone had a significant effect on the body weight as well as body weight gain of male rats. The initial body weight (g) was comparable between the control group and the dexamethasone-treated group, revealing no significant differences ($p > 0.05$). However, the final body weight (g) of the dexamethasone-treated group was significantly lower compared to the control group, demonstrating a statistically significant reduction ($p < 0.05$). Similarly, body weight gain differed significantly between the groups. While the control group revealed a positive body weight gain, the dexamethasone-treated group exhibited a negative body weight gain, reflecting a significant loss of body weight over the experimental period ($p < 0.05$).

3.2 Effect of dexamethasone on urea, creatinine, uric acid as well as total protein in male rats

The results were highlighted in **(**Table **2)** indicated the detrimental effects of dexamethasone on renal and protein metabolism in male rats.

The administration of dexamethasone significantly altered the levels of urea, creatinine, uric acid, as well as total protein in male rats compared to the control group. Urea levels (mg/dL) were markedly elevated in the dexamethasone-treated group compared to the control group ($p < 0.05$). Similarly, creatinine levels (mg/dL) showed a significant increase in the dexamethasone-treated group relative to the control group, further suggesting renal dysfunction ($p < 0.05$). Uric acid levels (mg/dL) also exhibited a significant rise in the dexamethasone group compared to the control group ($p < 0.05$).

Conversely, total protein levels (g/dL) were significantly reduced in the dexamethasone-treated group compared to the control group ($p < 0.05$).

3.3 Effect of dexamethasone on vitamin D, parathyroid hormone, calcium as well as phosphorus concentrations in male rats

(Table 3) reveals that the administration of dexamethasone significantly affected the concentrations of vitamin D, PTH, calcium, and phosphorus in male rats.

The dexamethasone-treated group exhibited significantly lower vitamin D levels compared to the control group. Conversely, parathyroid hormone levels were significantly elevated in the dexamethasone-treated group compared to the control group, reflecting a compensatory response to reduced calcium levels.

Calcium concentrations were significantly reduced in the dexamethasone group relative to the control group ($p < 0.05$). Phosphorus concentrations were markedly elevated in the dexamethasone-treated group relative to the control group ($p < 0.05$).

3.4 Effect of dexamethasone on FSH, LH as well as Testosterone in male rats

The results showed in (Table 4) elaborate dexamethasone had a significant effect on the levels of FSH, LH, in addition to testosterone in male rats.

FSH levels were significantly elevated in the dexamethasone-treated group in comparison to the control group ($p < 0.05$). LH was significantly elevated in the dexamethasone group as compared to the control group ($p < 0.05$).

Testosterone was significantly lower in the dexamethasone-treated group when compared with control group ($p < 0.05$).

Table 1. Effect of dexamethasone treatment on body weight and body weight gain in male rats. Data presented as Mean±SD, (N = 14)

(Groups)	(Parameters)		
	Intial Body Weight (g)	**Final Body Weight (g)**	**Body Weight Gain (g)**
Control (Normal saline 0.9% NaCl)	220.00±11.0 a	260.40± 2.01 a	40.40± 7. 2 a
Dexamethasone (0.5mg/Kg)	234.70± 6.14 a	175.10± 6.05 b	-59.60±2.04 b

N = total number of animals. Distinct letters denote significant differences between groups at $P \leq 0.05$ relative to the control.

Table 2. Effect of dexamethasone on urea, creatinine, uric acid as well as total protein in male rats. Data presented as Mean±SD, (N = 14)

(Groups)	Parameters			
	Urea mg/dl	**Creatinine mg/dl**	**Uric acid mg/dl**	**Total Protein g/dl**
Control (Normal saline 0.9%NaCl)	35±1.41 b	0.29± 0.03 b	5.00±2.00 b	6.00±0.83 a
Dexamethasone (0.5mg/kg)	52.16±8.61 a	5.66±0.87 a	8.3±2.94 a	4.00±0.89 b

N = total number of animals. Distinct letters denote significant differences between groups at $P \leq 0.05$ relative to the control.

Table 3. Effect of dexamethasone on vitamin D, parathyroid hormone, calcium as well as phosphorus concentrations in male rats. Data presented as Mean±SD (N = 14)

(Groups)	Parameters			
	vitamin D (ng/ml)	**parathyroid hormone (Pg/ml)**	**calcium (mg/ml)**	**phosphorus (mg/ml)**
Control (Normal saline 0.9%NaCl)	50.83±6.40 a	4.28±0.11 b	13.70±1.35 a	6.33±0.56 b
Dexamethasone (0.5mg/kg)	15.73±1.72 b	16.06± 2.02 a	9.45±1.44 b	11.78±0.03 a

N = total number of animals. Distinct letters denote significant differences between groups at $P \leq 0.05$ relative to the control.

Table 4. Effect of dexamethasone on FSH, LH as well as Testosterone in male rats. Data presented as Mean±SD (N = 14)

(Groups)	Parameters		
	FSH (mlU/ml)	**LH (mlU/ml)**	**Testosteron (ng/ml)**
Control (Normal saline 0.9%NaCl)	5.50±2.42 b	3.30±1.35 d	9.80±1.30 a
Dexamethasone (0.5mg/kg)	11.83±1.94 a	11.08±1.42 a	5.57 ±1.95 b

N = total number of animals. Distinct letters denote significant differences between groups at $P \leq 0.05$ relative to the control.

3.5 Effect of dexamethasone on femur bone histology

Control Group:

Histological examination of femur bone section from the control group reveals normal bone architecture as shown in (Figure 1). The photomicrograph reveals well-developed Haversian systems (green arrow), which are made up of central Haversian canals bordered by circular lamellae of dense bone matrix. This shows that the osteons are still physically functional. There are a lot of osteoblasts (purple arrows) that can be observed covering the bone surfaces. These

cells look like cubes and have basophilic cytoplasm. They are spread out evenly, which suggests that bones are actively forming and remodelling. The lamellar structure of bone tissue and the lack of degenerative changes show that the normal bone architecture when dexamethasone treatment is not administered.

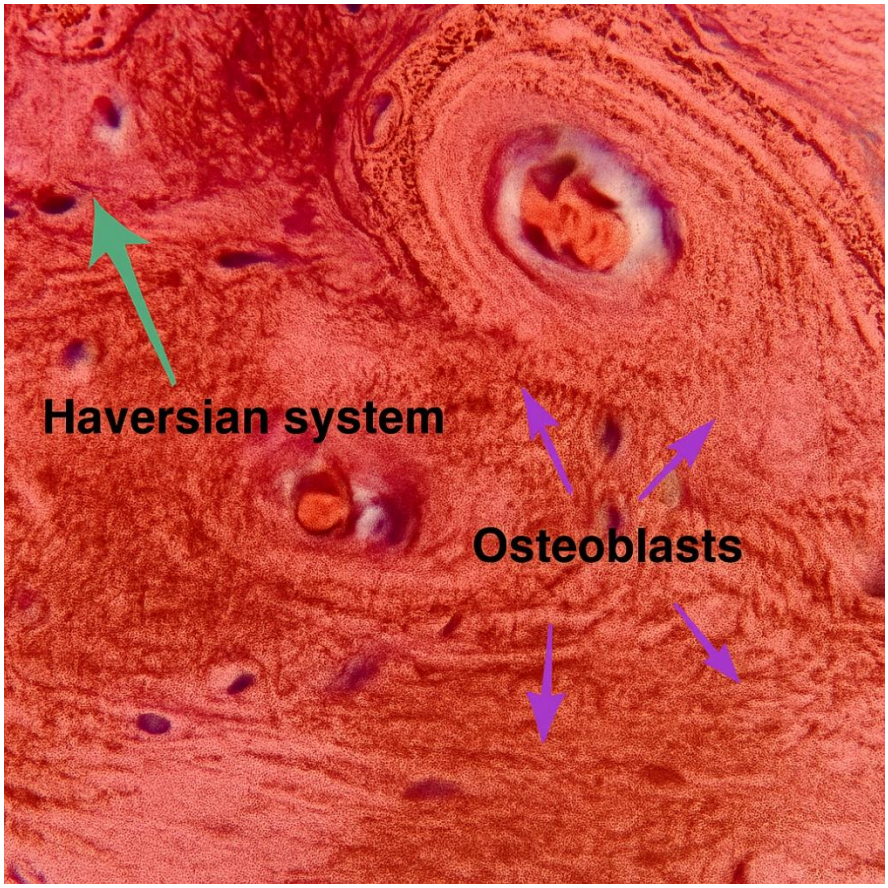

Figure 1. Section of femur bone of male rats of control group (Hematoxylin & Eosin stain, 125X)

Dexamethasone-Treated Group:

Histological examination of femur bone sections from male rats that treated with dexamethasone as shown in (Figure 2) demonstrated a pronounced structural damage when compared to control group. The spaces in the bone marrow are noticeably greater but the amount of trabeculae is noticeably reduced as shown by the orange arrows. There are fewer or no bone struts in these areas, which means that the trabecular bone is breaking down. Also, some of the remained trabeculae appeared highly thin and fragile, as shown by the blue arrows. These changes show that bone mass and structural support have been lost, which is consistent with osteoporosis caused by glucocorticoids. There is more space between the trabeculae, and the structure of the trabeculae is disrupted. According to these findings, dexamethasone breaks down bone tissue, mostly by stopping osteoblastic activity and speeding up osteoclastic resorption.

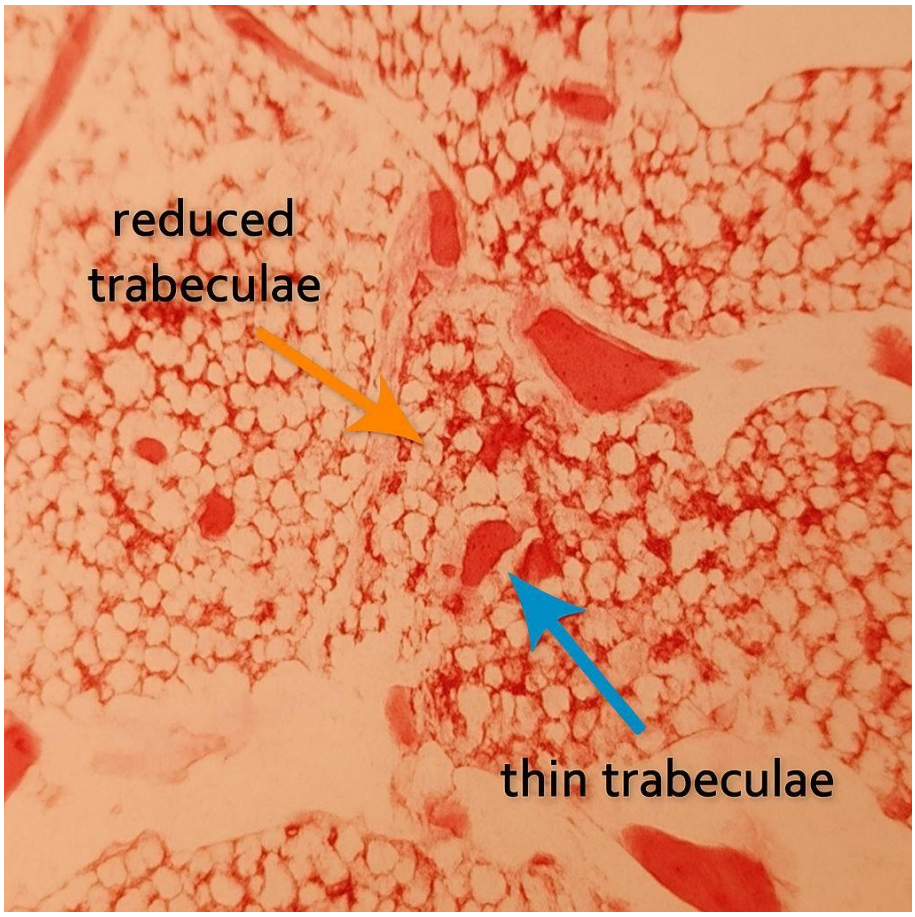

Figure 2. Section of femur bone of male rats of Dexamethasone treated group (Hematoxylin & Eosin stain, 125X)

4. Discussion

The present investigation shows the considerable physiological and biochemical influence of dexamethasone administration in male rats, particularly in body weight regulation, renal function, bone metabolism, and hormonal balance. These findings align with the well-documented effects of glucocorticoids in preclinical studies but also highlight areas requiring further exploration.

Effect of Dexamethasone treatment on body weight as well as body weight gain in this study indicates that dexamethasone administration adversely affects body weight regulation in male rats. This result is explained by the catabolic actions of glucocorticoids, which inhibit appetite and encourage the breakdown of proteins. Furthermore, it has been shown that glucocorticoids directly catabolise muscle, causing atrophy and weakness [17, 18]. Steroid myopathy is the most common and serious side effect of manufactured glucocorticoids that are linked to drug-induced myopathies [19, 20]. The gastrocnemius muscle significantly atrophied after a brief 10-day course of dexamethasone therapy [21].

Elevations in serum urea, creatinine, and uric acid, in conjunction with a decrease in total protein, in this study, indicate that dexamethasone may have impaired renal function and altered protein metabolism. This suggests that dexamethasone may have caused disturbances in purine metabolism or renal clearance, and that it may have had a negative impact on protein synthesis or increased protein degradation. Additionally, Mobeen et al., (2022) was reported that total protein levels were significantly reduced in the dexamethasone-treated group [22]. These changes probably happen because of glucocorticoids damaging the glomeruli and tubules and having systemic catabolic effects like lowering protein production in the liver and increasing protein breakdown, as demonstrated in rat models treated with dexamethasone [23]. However, the lack of functional kidney clearance tests makes it hard to come to a firm conclusion. This suggests that future studies need to include more thorough nephrological assessments.

Low amounts of vitamin D and calcium were observed along with high levels of parathyroid hormone as well as phosphorus. This indicated that there was an important issue with bone metabolism. These changes reflected an imbalance of minerals, which is typical of osteoporosis caused by glucocorticoids. The amount of phosphorus in the group that administered dexamethasone was significantly greater than in the control group. This could be because the dexamethasone changed the kidney's function or imbalance of calcium and phosphorus metabolism in the body. Sabeeh and AL-Saeed, (2025) also states that glucocorticoids exert lower levels of vitamin D and raise levels of parathyroid hormone [24].

Hypocalcaemia and secondary hyperparathyroidism are caused by glucocorticoids, which also inhibit renal calcium reabsorption and intestinal calcium absorption [2]. Ultimately, bone formation is diminished as a result of this hormonal imbalance, which accelerates bone resorption and suppresses osteoblast function. Although the biochemical data presented here supports these mechanisms, the histological analysis of the femur bone further elucidates the structural and cellular changes in bone, providing sufficient proof of glucocorticoid-induced bone loss. The hormonal profile showed decreased testosterone and increased gonadotropins (FSH, LH), suggesting a disturbance in the hypothalamic-pituitary-gonadal axis. The findings show that dexamethasone directly affects Leydig cell activity and disturbs the hypothalamic-pituitary-gonadal axis, which in turn inhibits testicular production of testosterone. Perhaps the elevated gonadotropin levels indicate a feedback mechanism triggered by low androgen levels, as the pituitary and hypothalamus attempt to re-establish hormonal equilibrium. Hampl and Stárka, (2020) suggests that glucocorticoids achieve these effects by inducing apoptosis and

inhibiting steroidogenic gene expression in Leydig cells, thereby decreasing testosterone production [25].

It's important to be aware of the study's restrictions, such as the small sample size and the fact that there wasn't a group that received vitamin D supplements so that any possible therapy effects could be assessed. Also, the 28-day treatment time may not fully show the long-term effects of glucocorticoid exposure, even though it was long enough to create changes that could be evaluated. Another limitation includes the body weight and food intake were not monitored every week, which could have supplied more information about how the long-term effects developed over the duration of the experiment. Because of all these factors, the data should be interpreted with care, and highlight for future researches.

In conclusion, this study shows that dexamethasone causes major changes in the functions of male rats. These changes are similar to the results when glucocorticoids cause osteoporosis and problems with reproduction. Adding direct measurements of bone density and turnover to experiments, along with treatments that target mineral and hormonal changes, will be very important for developing improved strategies to mitigate the negative impact of glucocorticoid therapy.

5. Conclusions

The results of this study reveal a significant data about how dexamethasone affects a number of morphological, physiological, and biological factors in male rats. Dexamethasone led to significant drops in body weight as well as weight gain, indicating how important it is for general growth and healthy metabolism. A rise in urea, creatinine, as well as uric acid levels alongside a fall in total protein levels indicate that the kidneys are not functioning as well and that the breakdown of protein is slowing down. Low levels of vitamin D as well as calcium, in addition to high levels of parathyroid hormone and phosphorus, and changes in the structure of the bones, reveal that the bones are not as dense or strong as they should be. This means that bone and mineral homeostasis have been significantly disrupted, which is a feature of osteoporosis. The data of the study has shown that the administering of dexamethasone may damage sexual health. This is shown by higher levels of FSH and LH and lower levels of testosterone. The findings indicate that dexamethasone has serious negative effects on the metabolism, muscle, kidney, reproductive and systems, which suggests that it may be toxic to the whole body.

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تقييم المعايير الفسيولوجية في ذكور الجرذان المصابة بهشاشة العظام الناجمة عن الديكساميثازون: فيتامين د، هرمون جار الغدة الدرقية، الكالسيوم، الفوسفور، والهرمونات التناسلية

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المستخلص

تناولت هذه الدراسة تأثير الديكساميثازون، وهو كورتيكوستيرويد شائع الاستخدام، على الوظائف البايوكيميائية والهرمونية والفسيولوجية الرئيسية لدى ذكور الجرذان. ركزنا على تأثيره على وزن الجسم، ووظائف الكلى، وتوازن المعادن، والتنظيم الهرموني. أظهرت النتائج أن الديكساميثازون تسبب في فقدان ملحوظ لوزن الجسم وقصور في وظائف الكلى، ويتضح ذلك من خلال ارتفاع مستويات اليوريا والكرياتينين وحمض اليوريك، بالإضافة إلى انخفاض البروتين الكلي. علاوة على ذلك، أدى الديكساميثازون إلى اضطراب ايض المعادن عن طريق خفض مستويات فيتامين د والكالسيوم، بينما رفع مستويات هرمون الغدة الدرقية والفوسفور، مما يشير إلى اضطرابات محتملة في ايض العظام. كما وكشف التحليل الهرموني عن ارتفاع مستوى الهرمون المنبه للجريب والهرمون اللوتيني، وانخفاض مستوى التستوستيرون، مما يوحي بتداخله مع تنظيم الهرمونات التناسلية. تُظهر هذه النتائج أن الديكساميثازون يُحدث اضطرابات فسيولوجية واسعة النطاق، مما يُبرز الحاجة إلى مراقبة دقيقة لهذه المؤشرات أثناء العلاج بالكورتيكوستيرويدات لتقليل الآثار الجانبية.

الكلمات المفتاحية: ديكساميثازون، كلوكوكورتيكويدز، توازن المعادن، هشاشة العظام، فيتامين د.