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RESEARCH ARTICLE

Misuse of dexamethasone for cosmetic purposes boosts hyperthyroidism and hepatotoxicity in albino rats

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Abstract

Background: The use of dexamethasone (Dex) for cosmetic purposes, particularly for skin lighting, is widespread and is associated with a high incidence of skin cancer in several populations. This study aimed to investigate the misuse of Dex for skin whitening, particularly its influence on thyroid, liver, and kidney function in female albino rats.

Materials and methods: In the in vivo comparative experiments I and II, 36 female albino rats, each weighing 140–162 g, were used. Thyroid function, liver enzyme activity, and renal function were assessed using enzyme-linked immunosorbent assay (ELISA). Liver and kidney sections were fixed and stained with hematoxylin and eosin (H&E).

Results: The groups administered high and low doses of Dex exhibited significant increases in thyroid hormone levels, liver enzyme activities, creatinine, and urea levels compared to the control group. In contrast, thyroid-stimulating hormone (TSH) levels were significantly lower ($P < 0.05$). Kidney sections displayed ghost glomeruli, partially necrotic tubular cells, and chronic inflammation at both doses. Liver sections showed binucleated cells, infiltration, and focal necrotic cells relative to the control.

Conclusion: The misuse of Dex for cosmetic purposes influences hyperthyroidism, hepatotoxicity, and renal impairment, with dose- and duration-dependent effects.

Keywords: Thyroid, hepatocyte, kidneys, dexamethasone, cosmetics.

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Introduction

Dex is a widely used synthetic steroid with anti-inflammatory and immune-suppressant properties [1]. In contrast, Dex is commonly used to lighten skin colors and is available in various forms, including creams, lotions, soaps, tablets, and injections. Approximately 25–67% of adult women regularly use skin-whitening cosmetics [2,3]. However, cultural standards and beauty norms can influence the use of cosmetics, and some populations find lighter skin tones more appealing [4,5]. Women use skin lighteners because they are dissatisfied with their natural skin [6]. Despite its potential risks and adverse effects, Dex is often misused as a cosmetic in many African and Asian countries, leading to the illegal distribution of unverified cosmetic products containing Dex [7, 8].

Dex decreases melanocyte-stimulating hormone production [9], which may make the skin more susceptible to Ultraviolet (UV) radiation from sunlight, consequently increasing the risk of skin cancers such as melanoma [10]. It has been proposed that Dex may cause a decrease in TSH, free triiodothyronine (FT3), and free tetraiodothyronine (FT4) levels due to damage to the pituitary gland, leading to secondary hypothyroidism [11]. Additionally, Dex elevates transaminase activities and creatinine levels [12]. Moreover, steroid drugs cause fatty liver and necrosis, as well as dil-

atation and glomerular congestion [13]. There is a need for immediate research to highlight the discrepancies between previous studies and the increasing improper use of Dex as a cosmetic product. Therefore, we used experimental female albino rats to evaluate the impact of Dex on thyroid, liver, and kidney function at varying doses and durations.

Materials and Methods

Drug Preparation

Dex 0.5 mg was obtained from a local pharmacy in Khartoum State. The tablet was crushed, and 1 mL of 50% methanol was added to form a stock suspension. The mixture was homogenized using an Ultrasonic Sonicator and diluted to achieve the desired concentrations at low and high doses.

Experimental Animals

The study utilized female albino rats, aged 3–4 months and weighing 140–162 g, were obtained from the Pharmacy College Animal House. Rats were acclimated to laboratory conditions for 7 days. The rats were housed under standard conditions and were healthy, fed a nutrient-rich diet. In the in vivo comparative animal study, 36 rats were divided into six groups of six rats each, with 18 rats per experiment, as follows:

In Experiment I, Group I received a placebo and served as the control group. Group II was treated with a low dose of Dex at 8.3 µg/kg/day, while Group III was treated with a high dose of Dex at 24.9 µg/kg/day for 30 days.

In Experiment II, Group I received a placebo once again. Group II was treated with the same low dose of Dex (8.3 µg/kg/day), and Group III received the high dose of Dex (24.9 µg/kg/day) for 60 days. All doses are administered orally.

Ethical Approval and Euthanasia

This study was approved by the local Committee of the Faculty of Medical Laboratory Sciences, Al-Neelain University, and all experiments were conducted at the Pharmacy College of King Abdulaziz University. It adhered to the guidelines set by the US National Institutes of Health and the Declaration of Helsinki for the ethical treatment of animals. After an 18-hour overnight fast, the rats were euthanized using 1.9% diethyl ether in a closed chamber through inhalation, in accordance with the American Veterinary Medical Association (AVMA) Guidelines for the Euthanasia of Animals: 2020 Edition. Blood samples were collected via cardiac puncture, and the rats were then sacrificed to collect liver and kidney tissues. Liver and kidney tissues were examined histologically, and blood samples were centrifuged at 3000 rpm to obtain serum. Biochemical parameters were measured using an ELISA Dynex Best 2000 analyzer.

Estimation of Thyroid Hormones

TA double-sandwich ELISA was performed using T3 (Cat. No. MBS261285), T4 (Cat. No. MBS704309), and TSH (Cat. No. MBS726442) competitive enzyme immunoassay kit (MyBiosource, Inc., Germany). The T3 assay utilized a specific antibody coated on a surface and an avidin-HRP combination. After incubation, the free components were washed, and the substrate was added. The color intensity was proportional to the T3 concentration. T4 and TSH are coated explicitly with biotin-conjugated antibodies. After incubation, the unbound T4 and TSH were washed. Then, the substrate was added. Color intensity was inversely correlated with TSH and T4 levels.

Estimation of Liver Enzymes

The double-sandwich ELISA method was used to quantify the activities of aspartate transaminase (AST) (Cat. No. MBS264975), alanine transaminase (ALT) (Cat. No. MBS269614), alkaline phosphatase (ALP) (Cat. No. MBS269614), and γ-glutamyl transpeptidase (GGT) (Cat. No. MBS9343646) (MyBiosource, Inc., Germany). The antibodies were tagged with biotin and monoclonal pre-coated antibodies. Samples and biotin-labeled antibodies were added to each well. After rinsing, the avidin-peroxidase conjugates were added. The color was proportional to the activities of AST, ALT, ALP, and GGT.

Estimation of Urea and Creatinine

The ELISA (Cat. No. MBS2600001) (MyBiosource, Inc., Germany) was used to measure the urea levels. Briefly, according to the manufacturer, color development was based on the urea concentration. A competitive enzyme immunoassay (Cat. No. MBS749827) from MyBiosource, Inc. (Germany) was used to measure creatinine levels. Color development was inversely proportional to the creatinine concentration.

Histological Methods

The experimental rats were euthanized, and the liver and kidney organs were excised. Biopsies were obtained for histopathological analysis and fixed in 10% neutral-buffered formalin. The samples were then processed into paraffin wax blocks, and 5µm sections were prepared [14]. These sections were stained with hematoxylin and eosin (H&E), a standard pathological stain. Two experienced pathologists conducted a systematic, anonymous, and blinded analysis of all samples. Findings were reported blindly, without knowledge of the treatment groups, and later matched to the respective rat groups. Once identified, the findings were correlated with specific treatment exposures, and representative histology for each of the four experimental groups was selected and is shown in Figures 4 and 5.

Statistical Analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) software (version 21.0; SPSS Inc., Chicago, IL, USA). Data are expressed as mean ± standard error of the mean (SEM) and compared with controls using a t-test and analysis of variance (ANOVA). Statistical significance was set at $p<0.05$.

Results

Thyroid Hormones

After 30 days of Dex treatment, TSH levels were significantly decreased in the low-dose (2.17 ± 0.32 mIU/L) and high-dose (0.60 ± 0.15 mIU/L) groups compared to the control

group (4.27 ± 0.37 mIU/L) (p<0.05). The T3 levels were significantly higher in the low-dose (132 ± 5.70 ng/ml) and high-dose (209 ± 20.2 ng/ml) groups than in the control group (103 ± 4.26 ng/ml) (p<0.05). The T4 levels were significantly increased in the low-dose (15.2 ± 0.72 µg/dL) and high-dose (25.0 ± 1.75 µg/dL) groups compared to the control group (9.06 ± 0.43 µg/dL) (p<0.05). After 60 days of Dex treatment, the TSH in the low-dose (1.40 ± 0.40 mIU/L) and high-dose (0.66 ± 0.15 mIU/L) groups decreased significantly compared to the control group (3.11 ± 0.27 mIU/L) (p<0.05). There were significant increases in the T3 levels in low-dose (131 ± 13.05 ng/ml) and high-dose (275 ± 28.49 ng/ml) compared to the control group (103 ± 4.24 ng/ml) (p<0.05. The T4 levels were significantly increased in the low-dose (18.1 ± 0.90 µg/dL) and high-dose (27.1 ± 2.45 µg/dL) groups compared to the control group (9.10 ± 0.53 µg/dL) (p<0.05), as shown in Figure 1.

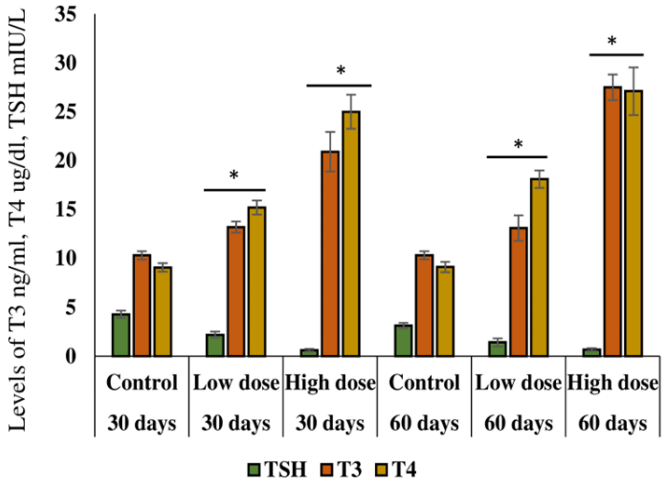

Figure 1. Comparison of mean TSH, T3, and T4 levels among Group II (low-dose Dex), Group III (high-dose Dex), and the control group following Dex administration for 30 and 60 days. (*) Indicates a significant difference (p<0.05).

Liver Enzymes

The administration of Dex for 30 days resulted in significantly increased ALT, AST, ALP, and GGT activities in both the low-dose and high-dose groups compared to the control group (p < 0.05). After administering Dex for 60 days, ALT activities were significantly increased in both the low-dose (56.2 ± 5.60 U/L) and high-dose (126 ± 23.0 U/L) groups compared to the control group (16.2 ± 1.30 U/L) (p < 0.05). The AST activities were significantly increased in the low-dose (71.5 ± 8.60 U/L) and high-dose (144 ± 18.7 U/L) groups compared to the control group (22.5 ± 2.17 U/L) (p<0.05). The ALP activities were significantly higher in the low-dose (105 ± 7.20 U/L) and high-dose (168 ± 20.5 U/L) groups than in the control group (52.6 ± 3.29 U/L) (p<0.05). The GGT activities were significantly increased in the low-dose (61.0 ± 3.66 U/L) and high-dose (98.0 ± 6.40 U/L) groups compared to the control group (16.0 ± 1.18 U/L) (p<0.05), as depicted in Figure 2.

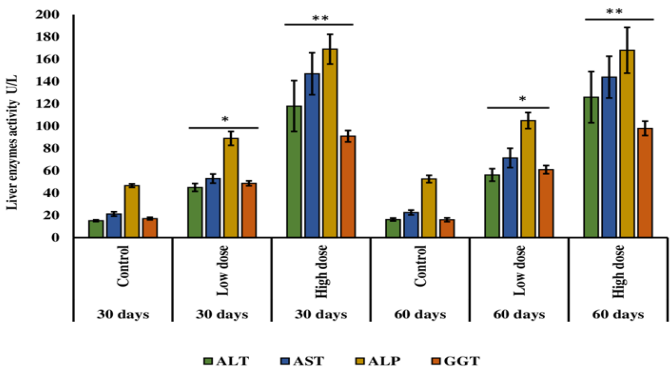

Figure 2. Comparison of liver enzyme activity levels (ALT, AST, ALP, and GGT) among Group II (low-dose dex), Group III (high-dose dex), and the control group over 30 and 60 days. (*) Indicates a significant difference (P < 0.05).

Renal Functions

After administering Dex for 30 days, the mean urea levels were significantly increased in the low-dose (4.18 ± 0.25 mmol/L) and high-dose (7.98 ± 0.63 mmol/L) groups compared to the control group (2.09 ± 0.07 mmol/L) (p<0.05). The creatinine levels were significantly higher in the low-dose (119 ± 12.3 µmol/L) and high-dose (167 ± 10.6 µmol/L) groups than in the control group (53.9 ± 5.30 µmol/L) (p<0.05). Following 60 days of Dex treatment, the urea levels were significantly increased in the low-dose (4.48 ± 0.34 mmol/L) and high-dose (7.80 ± 0.49 mmol/L) groups compared to the control group (2.32 ± 0.11 mmol/L) (p<0.05). The creatinine levels were significantly higher in the low-dose (123 ± 12.3 µmol/L) and high-dose (176 ± 15.0 µmol/L) groups compared to the control group (61.8 ± 7.07 µmol/L) (p<0.05), as shown in Figure 3.

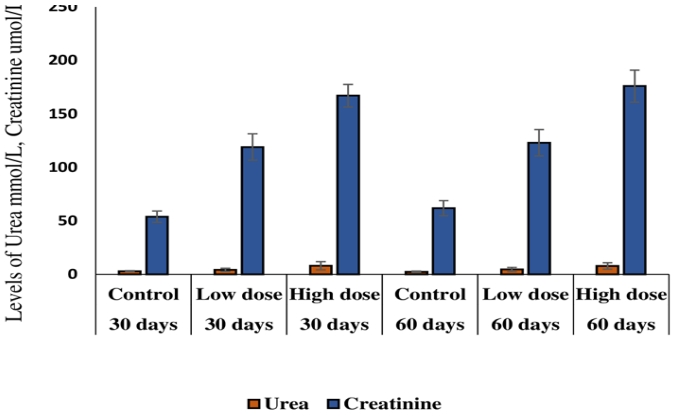

Figure 3. Comparison of mean urea and creatinine levels among Group II (low-dose Dex), Group III (high-dose Dex), and the control group over 30 and 60 days. A significant difference was defined as P ≤ 0.05.

Histological findings

Experiment 1: Control group A displayed normal hepatocytes (Figure 4). Groups B and C showed mild congestion, focal hepatocyte necrosis, nuclear lysis, dilated blood sinusoids with mononuclear inflammatory cells, active hepatocytes with significant numbers of

inflammatory cells (primarily lymphocytes), and numerous apoptotic cells.

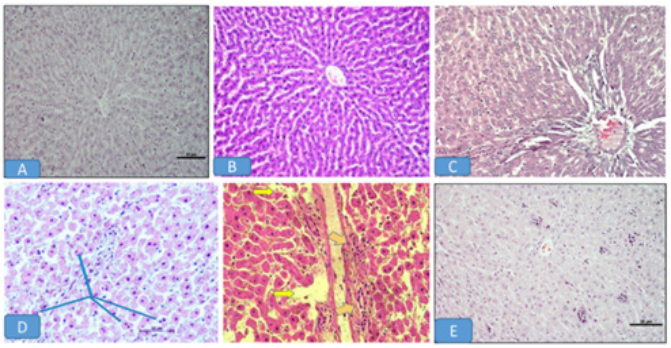

Figure 4. Histological section of liver stained with H and E (×10,40,40,40,20): A: Group I as control, B: Group II, which received low dose Dex for 30 days, C: Group II, which received low dose Dex for 60 days, D: Group III which received high dose for 30 days, E: Group III which received high dose Dex for 60 days.

Experiment 2: Group D showed increased binucleated cells due to hepatocyte toxicity and acute inflammatory cell infiltration (mainly neutrophils) with focal necrotic regions. Group E showed significant central vein congestion, loss of hepatocyte outlines, nuclear lysis, and dilated blood sinusoids filled with mononuclear inflammatory cells.

Experiment 1: A: Normal kidney sections from the control group (Figure 5). B: Mild chronic tubular inflammation with lymphocytic infiltration and tubular necrosis, reduced corpuscle and glomerular size, dilated tubular lumen, and slight parenchymal disorganization. C: Vascular and kidney tubular degeneration with a balanced cellular appearance.

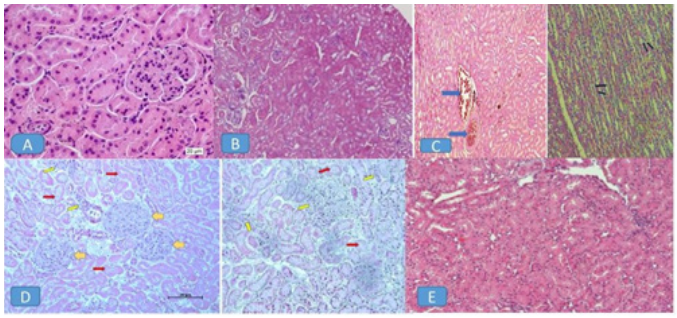

Figure 5. Histological section of kidney stained with H and E (×40, 10, 10, 20, 20): A: Group I Control, B: Group II treated with low dose Dex for 30 days, C: Group II which was treated with low Dex dose for 60 days, D: Group III treated with high dose Dex for 30 days, E: Group III which received high dose Dex for 60 days.

Experiment 2: D: Ghost glomeruli and tubular cells exhibiting necrosis, chronic inflammation, hemorrhage, reduced corpuscle and glomerular size, and a dilated tubular lumen with significant parenchymal disorganization. E: Significant lymphocytic infiltration, tubular necrosis, chronic inflammation, and hemorrhage with reduced corpuscle and glomerular size, dilated tubular lumen, and marked parenchymal disorganization.

Discussion

Despite its many adverse effects, Dex is commonly administered orally for skin lightening in African and Asian countries and has been associated with higher rates of skin cancer, especially in regions with elevated ultraviolet (UV) exposure. However, only a few studies have examined the direct effects of Dex on liver toxicity and thyroid functions. Therefore, this study aimed to investigate the effects of Dex on thyroid, liver, and kidney function in albino rats, simulating its potential misuse for cosmetic purposes. Dex dosages elevated T3 and T4 levels while suppressing TSH levels with dose- and duration-dependent effects. The findings partially align with earlier research that reported elevated levels of T3 and T4 following Dex treatment [15, 16]. However, other studies contradict the notion that Dex treatment leads to secondary hypothyroidism [17]. This contradiction is attributed to the fact that prolonged Dex treatment causes pituitary cell damage, resulting in low TSH levels and subsequently reduced stimulation of the thyroid gland [18,19]. The steroid hormone Dex may account for this inconsistency, as low doses of Dex synergize with thyroid hormones to enhance female fertility, given that estrogen increases the production of thyroid-binding globulin [20].

The cytotoxic effect of Dex on liver function was investigated and compared to that of the control group. Both low- and high-dose treatments resulted in higher activities of AST, ALT, GGT, and ALP. Our investigation indicated the potential hepatotoxic effect of Dex, which may lead to the leakage of transaminases and cholestasis biomarkers into the bloodstream due to hepatocellular damage. Prior research on Dex abuse has revealed liver necrosis, which was attributed to the damaged structural integrity of the liver caused by long-term Dex treatment [21,22]. Furthermore, our findings were supported by histological examination of liver sections, which revealed fatty liver and hepatocyte necrosis, consistent with previous reports that Dex administration leads to increased AST and ALT activities, indicating hepatocellular damage [13,23,24] . Moreover, increased serum ALP activity indicates disturbances in biliary flow, liver damage, or cholestasis [21]. This is further supported by elevated GGT activity, which is known to increase in response to liver injury or biliary obstruction [25]. Hence, previous studies have demonstrated that chronic Dex treatment leads to increased activity of cholestatic biomarkers [26, 27], which results in hepatotoxicity and liver injury [28].

To demonstrate the effect of Dex on renal function, urea and creatinine levels were measured, and kidney histological sections were analyzed. Our findings showed elevated urea and creatinine levels after Dex administration, indicating renal dysfunction [23]. Typically, these increases are associated with impaired renal function resulting from glomerular and tubular damage [27, 29] and a reduction in the number of nephrons 30. These observations were reinforced by histological results, which showed increased

renal corpuscles and glomerular size, along with necrosis, following Dex treatment. Previous studies have also reported that Dex raises urea and creatinine levels [12, 13, 22, 27] and induces renal necrosis [31]. Elevated serum creatinine levels indicate impaired glomerular clearance, whereas increased plasma urea levels may result from impaired renal excretion due to glomerular and tubular damage from prolonged Dex administration [32]. Additionally, another study reported that corticosteroids increase protein catabolism and blood urea nitrogen (BUN) levels [33].

This study provides insights into the misuse of Dex for skin lightening and its effects on thyroid, liver, and kidney function. However, the lack of assessment of FT3 and FT4 levels, which are more specific to thyroid functions, and the absence of clinical data from experimental animals are considered limitations.

Conclusion

The study investigated the effects of oral Dex administration and found that Dex increased T3 and T4 levels while decreasing TSH levels. Dex also increased ALT, AST, ALP, and GGT activities, as well as urea and creatinine levels. The findings indicated marked necrosis and inflammation in the liver and kidney tissues. Thus, oral misuse of Dex for cosmetic purposes has the potential to induce hepatotoxicity, renal dysfunction, and hyperthyroidism in a dose—and duration-dependent manner. Further studies are required to understand the mechanisms underlying these adverse effects.

Authors' contributions

Conception and design F.Y.A., A.M.I.; Supervision A.M.I., A.A.A., S.M.A.; Conducting experiments F.Y.A., S.M.A., A.A., Z.E., A.A.A., A.M.I.; Writing original research F.Y.A.; Writing review and editing A.M.I., F.Y.A., S.M.A., A.A.A., Z.E., A.A., O.A.M. All authors have read and approved the manuscript.

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Disclosure statement

None to declare.

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