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Skin Changes in Iatrogenic Cushing's Syndrome: A Detailed Case Analysis

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Abstract

Cushing's syndrome can lead to various skin complications. Iatrogenic Cushing's syndrome is a condition caused by excessive exogenous glucocorticoid exposure. This case report presents a patient with a history of Cushing's syndrome and long-term corticosteroid use who developed multiple skin manifestations. A 20-year-old male presented with erythematous purulent lesions on the face, neck, chest, and back. Concurrently, the patient developed purplish-red striae on the chest, abdomen, arms, and legs. Physical examination revealed erythematous nodules and pustules, along with atrophic striae. Laboratory tests showed leukocytosis with a left shift and hypokalemia. Radiological investigations revealed lumbar spondylosis and cardiomegaly. The patient was treated with a combination therapy, including topical and systemic medications, and showed significant improvement in skin lesions. This case highlights the importance of a comprehensive evaluation for patients presenting with unusual skin lesions. A thorough history, physical examination, and laboratory investigations are crucial to identify underlying conditions and initiate appropriate treatment.

1. Introduction

Iatrogenic Cushing's syndrome is a rare condition caused by excessive exogenous glucocorticoid exposure, which disrupts the hypothalamic-pituitary-adrenal (HPA) axis and leads to systemic hypercortisolism. This condition can result from long-term or high-dose use of corticosteroids prescribed for various medical conditions, including autoimmune diseases, respiratory disorders, and dermatologic ailments [1]. In resource-limited settings, the accessibility and affordability of over-the-counter corticosteroids further exacerbate the risk of this condition [2]. For instance, topical or oral steroids are frequently used for non-specific skin conditions without proper medical supervision, leading to systemic side effects. Skin manifestations are among the hallmark features of Cushing syndrome, often presenting as striae, easy bruising, delayed wound healing, and thinning of the skin [3]. These symptoms can significantly impact a patient's quality of life and may serve as a visual clue for clinicians to investigate underlying hypercortisolism. Despite being a well-recognized consequence of glucocorticoid therapy, the clinical presentation of iatrogenic Cushing syndrome remains underreported in

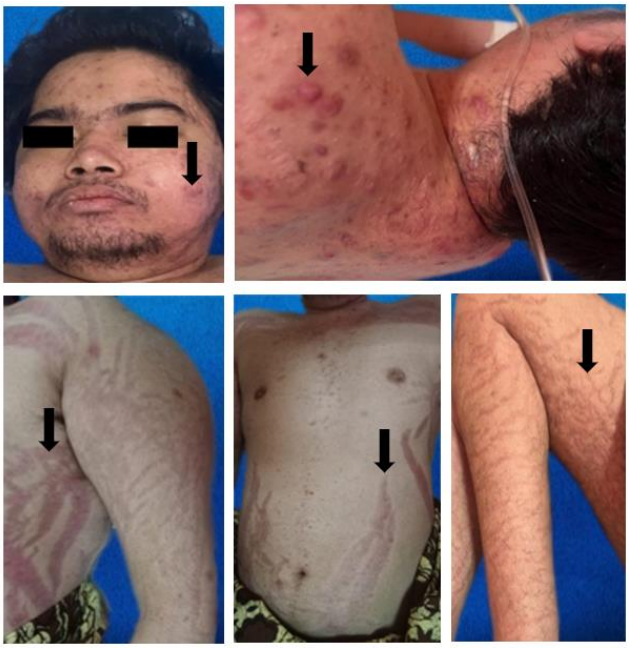

Figure 1. Clinical photograph of the patient when first came to the hospital. Physical examination of dermatology status showed Acneiform eruptions are seen on the face and back, as well as striae on the trunk and extremities regions.

certain populations [4]. A study in Denmark from 1985 to 1995 reported the incidence of Cushing's syndrome in as many as 166 patients, with an incidence of two to three cases per 1 million people per year [5]. The highest mortality was reported to occur in the first year [6]. This is particularly true in cases where skin disorders dominate the symptomatology, potentially delaying diagnosis. Understanding such presentations is critical for timely intervention and prevention of long-term complications [4].

Diagnostic confirmation often involves biochemical tests, including serum cortisol levels and suppression testing, which can distinguish iatrogenic from endogenous causes [7]. Imaging studies are rarely required unless an endogenous source of cortisol excess is suspected. Therefore, clinicians must integrate clinical findings with patient history to arrive at an accurate diagnosis [8]. Management of iatrogenic Cushing syndrome requires a multi-prolonged approach, including tapering or discontinuing corticosteroids and addressing complications [9]. Gradual withdrawal of glucocorticoids is essential to allow recovery of the HPA axis and prevent adrenal insufficiency [10]. Concurrently, symptomatic management of skin manifestations, such as using emollients and topical antibiotics for secondary infections, is crucial. Patient education plays a pivotal role in preventing recurrence by emphasizing the risks of unsupervised steroid use.

This case report aims to detail a unique presentation of iatrogenic Cushing syndrome with prominent skin manifestations. Skin manifestations of Cushing syndrome can mimic or exacerbate primary dermatologic conditions, posing a diagnostic challenge for clinicians [11, 12]. It highlights the challenges faced by clinicians in diagnosing and managing such cases, particularly in resource-constrained settings. The report also explores strategies to prevent similar occurrences, including patient education and regulatory measures.

2. Cases

A 20-year-old man came to the Department Dermatology and Venereology of dr. Zainoel Abidin General Hospital with complaint of purulent red spots on the face, neck, chest, and back area. Complaints appeared along with the emergence of purplish-red stretch marks on the chest, stomach, hands, and feet. Another symptom is that the face of the patient becomes swollen or looks rounded. Since 2022, the patient began to complain of acne on the face containing pus that was itchy and painful. Initially, acne only appeared on the face, but the acne increased to the chest and back over time. The patient also complained of skin that seemed to stretch, which was purplish red on the chest, stomach, arms, legs, and buttocks. This rash sometimes itches. The patient had a history of falling from a tree, so he often complained of lower back pain. The patient has been taking dexamethasone 0.5 mg twice daily to manage pain for the past four years. The medication was obtained from a pharmacy without a prescription or doctor's supervision.

Physical examination of dermatology status showed erythematous nodules and pustules, multiple, discrete, and distributed generally in fascial, Colli, posterior thoracoabdominal, and trunk regions. Other lesions showed purplish atrophic scars, well-defined plaque size, discrete, and distributed generally in anterior thoracoabdominal, superior, and inferior extremity dextra, and sinistra regions (Figure 1). Blood pressure of admission was 174/102 mmHg which can be diagnosed with hypertension stage 2. Laboratory examination showed 21,670 cells/mm^3, an increase in segment neutrophils (77%), and a decrease in lymphocytes (14%), or what is called a shift to the left. A lumbar x-ray was performed in November 2024, with the result shown in Figure 2, and a conclusion was reached regarding lumbal spondylosis. The thorax x-ray in Figure 3 showed cardiomegaly results and increased pulmonary bronchovascular markings.

Patients were given cetirizine tablets 10 mg twice a day, a mixture of retinoic acid 0.05% cream, Vaseline album for

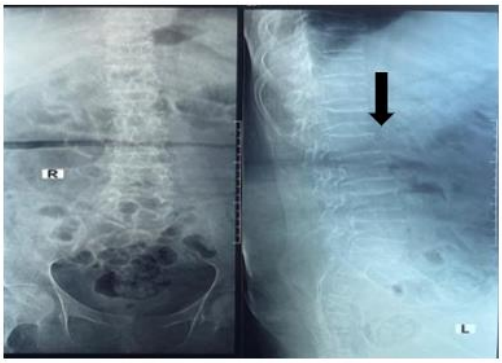

Figure 2. Lumbar X-ray. Lumbar Spondylosis overview.

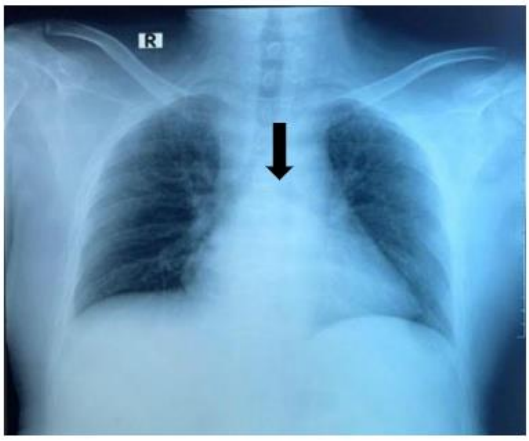

Figure 3. Thorax X-ray. Cardiomegaly overview and increased pulmonary bronchovascular markings.

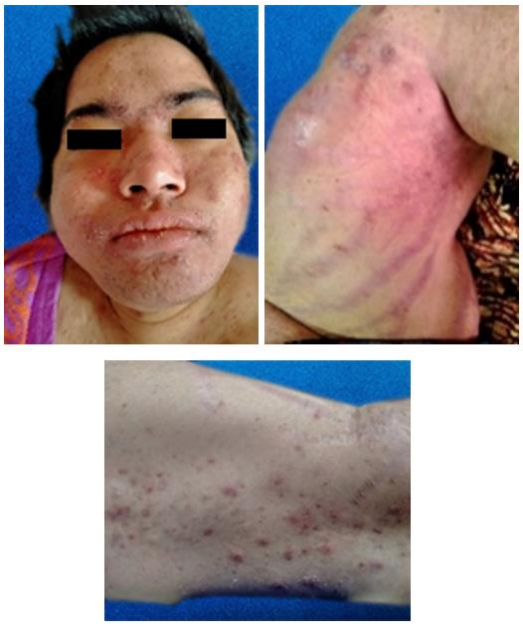

Figure 4. Follow up at 12 days. Pustule and striae began to decrease.

striae, and gentamicin 0.1% for pustules. The patient was also consulted by the Department of Internal Medicine due to hypertension and hypokalemia and was given an amlodipine tablet of 10 mg once a day and a potassium chloride tablet of 600 mg once a day. The patient was also consulted by neurology due to low back pain and was given mecobalamin tablet 500 mg twice a day and gabapentin tablet 100 mg twice a day. The patient consulted an orthopedic due to low back pain and was given a ketorolac injection of 1 ampoule per 8 hours and a lumbar corset. After 12 days of treatments, the lesions showed improvement. Pustule and striae began to decrease (Figure 4). Treatment continues, steroids should be tapered off, and patients should be given the education to avoid prolonged and uncontrolled use of corticosteroids.

3. Discussions

The patient was referred to the Zainoel Abidin Hospital with complaints of lesions containing pus on the face, neck, back, and chest, the most severe complaints felt on the neck. Complaints were accompanied by complaints of a purplish-red rash spread throughout the body. The patient also complained of pain in the waist that was felt after falling 7 months ago. The patient has a history of oral steroid use without dosage guidelines for 4 years.

The patient's face also looks rounder. These symptoms are typical clinical manifestations of Cushing's syndrome. Clinically, patients with this syndrome often show a full face and edema that looks like a "moon face," as well as fat accumulation in the supraclavicular area and upper back that forms a "buffalo hump" [13]. Complaints in the form of a purplish-red rash all over the body in patients are called striae. Striae are caused by long-term use of corticosteroids without dosage guidelines. One of the causes of this condition is due to exposure to exogenous corticosteroids [14]. This is also supported by the patient's statement that in the past 4 years, he has used oral steroids without any dosage guidelines and duration of use of this drug, so there is suspicion of Cushing's syndrome.

Cushing's syndrome is a reversible endocrinological disorder characterized by increased levels of cortisol or other glucocorticoids in the bloodstream. This syndrome can result from endogenous factors, such as excessive steroid secretion due to adrenal or pituitary tumors, or exogenous factors, such as prolonged corticosteroid use, which is the primary cause of iatrogenic Cushing's syndrome [15]. It specifically arises from long-term exposure to glucocorticoids. There is no specific exposure time, which varies between 1 and 72 months [16].

A series of tests are performed to evaluate cortisol levels and establish an initial diagnosis. Tests that can be used include 24-hour urine-free cortisol (UFC) measurement,

overnight 1 mg dexamethasone suppression test (DST), midnight salivary cortisol measurement, or low-dose dexamethasone suppression test for 48 hours, with a minimum of two repetitions in some tests. If the test results show normal cortisol levels, then Cushing's syndrome can be ruled out [17].

This patient was suspected of having iatrogenic Cushing's syndrome based on the history physical examination, and available supporting examinations. In the patient, there was a moon face, central obesity, purplish striae, acneiform eruption, grade II hypertension, dyslipidemia, and signs of bone degeneration. In this patient, 24-hour urine-free cortisol and midnight salivary cortisol levels were not performed because of the unavailability of test reagents. In addition, the dexamethasone suppression test was also avoided because there was concern that the patient's general condition could worsen due to the administration of oral dexamethasone. This step was taken to reduce the risk of further complications, given the potential side effects that could arise from additional exposure to glucocorticoids in patients with an already vulnerable condition. Due to the wide variation of symptoms, remission criteria for Cushing's syndrome are still difficult to determine. In addition to checking cortisol levels, the success of treatment can be seen in the improvement of symptoms and the general condition of the patient [6].

Cushing's syndrome can cause striking dermatological manifestations, such as thinning of the skin due to increased collagen degradation. Although little research has focused on the skin manifestations caused by systemic glucocorticoids, the common dermatological manifestations of Cushing's syndrome are very significant. These disorders include skin and hair problems, including purple striae, hyperhidrosis, hyperpigmentation, acanthosis nigricans, and acne, while hair disorders include hirsutism and alopecia [18].

In this patient, wide purple striae were spread almost all over his body, starting from the chest, stomach, back, hands, and feet. Wide purple striae, often exceeding 1 cm, are typical signs of hypercortisolism. This is influenced by the dilation of blood vessels that are getting thinner. Striae in Cushing's syndrome will differ from narrow pink or pale striae due to rapid weight gain or pregnancy. Excessive glucocorticoids in Cushing's syndrome damage keratinocytes and inhibit the proliferation of dermal fibroblasts, reducing the synthesis of collagen types I and III, which are important for skin strength and elasticity. As a result, the dermis and epidermis are thinning, making the skin more fragile, easily stretched, and prone to striae formation, especially in areas with fat accumulation, such as the abdomen, thighs, and arms [19].

This condition also damages the skin's lipid barrier, increasing water loss through the epidermis (transepidermal water loss), which causes the skin to become dry and resembles natural skin aging [20]. Another dermatological manifestation of Cushing's syndrome in this patient is the appearance of acne on the back, neck, shoulders, and upper arms. Acne that occurs after chronic exposure to steroids is called steroid-induced acne. Steroid-induced acne, called acneiform eruption, is a group of disorders characterized by papules, pustules, nodules, and cysts resembling acne vulgaris. Acneiform eruption often occurs on the trunk and extremities, with less facial involvement, by the lesions that arise in patients. In addition, this acneiform eruption occurs suddenly, according to the results of the patient's anamnesis and control of treatment, where new lesions appear suddenly. The sudden onset, uniformity of the lesions, and distribution beyond the seborrheic areas distinguish this acneiform eruption from acne [21].

Although the pathogenesis of acneiform eruptions is unclear, the development of acne due to corticosteroids has been reported for more than 70 years. The underlying mechanism is believed to be a direct effect of steroids on epithelial degeneration and inflammation or an increase in free fatty acids (FFAs) in the skin, leading to increased bacterial levels. The onset of these eruptions can vary widely, from manifestation immediately after administration of the drug to several months later [20]. Figure 5 shows the various functions of systemic corticosteroids in the immune system. Suppression of immune function in cases of Cushing's syndrome can lead to infections in various body locations, including the skin. Pathophysiologically, corticosteroids can increase the expression of toll-like receptors (TLRs) on keratinocytes, which can increase the inflammatory response to bacteria [21].

The primary management of iatrogenic Cushing's syndrome focuses on the gradual reduction or discontinuation of glucocorticoid therapy (tapering) to prevent adrenal insufficiency secondary to hypothalamic-pituitary-adrenal (HPA) axis suppression. Glucocorticoid withdrawal should be done slowly, especially in patients who have been on high doses or long durations of therapy, to allow the HPA axis to recover gradually and prevent adrenal crisis. Tapering protocols vary depending on the dose and duration of glucocorticoid use but often involve weekly or monthly dose reductions with close clinical monitoring [22].

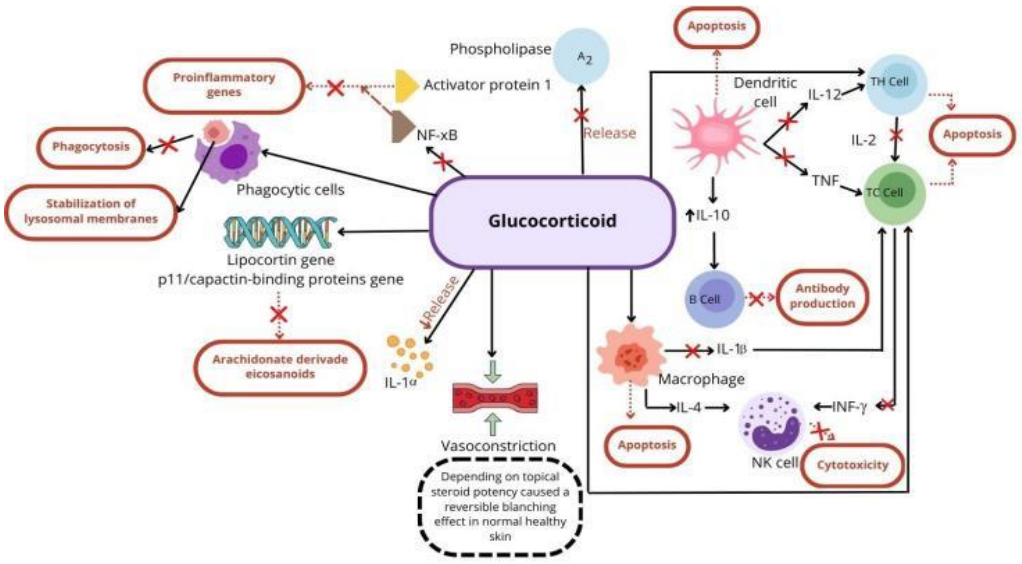

Figure 5. The role of corticosteroids in suppressing the immune system. Adapted from [21].

These patients are required to undergo corticosteroid dose tapering to prevent adrenal crisis, given their long-term use of high doses of corticosteroids. In addition, benzoyl peroxides, topical retinoids, and oral or topical antibiotics, such as doxycycline and clindamycin, as in acneiform eruptions, may be recommended [23]. These agents are known to decrease sebum production and resolve eruptions quickly [24]. Skin cleansers such as salicylic acid or benzoyl peroxide may be recommended to reduce oily skin. Itching is a common symptom in patients with acne-like eruptions, and these patients may benefit from antihistamines. For nocturnal itching, first-generation antihistamines are recommended as they also induce sleep [25]. By this therapeutic approach, the patient was managed with 0.05% retinoic acid, a topical retinoid formulation, and the patient was discontinued from systemic steroids, which were the trigger for her acneiform eruptions. The patient was also given 3% salicylic acid as a skin cleanser, Vaseline as a moisturizer, and cetirizine 10 mg tablets to manage the itching symptoms of the acneiform eruption lesions.

Striae management varies and has mixed and controversial results. Various modalities of striae therapy include topical therapy, laser, light therapy, physical or chemical peels, filler or collagen injections, and microneedle radiofrequency [26]. Topical therapy is the most common method of treating Striae Distensae, but few topical agents have been studied. One modality of topical therapy that can be chosen for striae is retinoid. Retinoids can increase the amount of dermal collagen through modulation of tumor growth factor-beta (TGF-β), which regulates fibroblast synthesis in the extracellular matrix. Stimulation of these fibroblasts causes an increase in collagen types I and III. The retinoid used in this therapy is topical tretinoin at a concentration of 0.025-0.1% [27].

The use of moisturizing creams such as hyaluronic acid and ceramides, in addition to helping to overcome the side effects of tretinoin, also increases resistance to mechanical traction and stimulates collagen formation. The ceramide component in moisturizing cream restores barrier function and skin permeability, and it is anti-inflammatory [28]. The patient was treated with 0.05% retinoic acid, which is a topical retinoid formulation; the patient was also given 3% salicylic acid as a skin cleanser and Vaseline as a moisturizer for dry skin on striae.

4. Conclusions

We conclude from this case report that it highlights the importance of recognizing the diverse clinical presentations of iatrogenic Cushing's syndrome, especially in settings with limited access to specialized diagnostic tests. This case also emphasizes the role of patient education and regulatory measures in preventing iatrogenic Cushing's syndrome. Healthcare providers must be mindful of the potential risks of long-term corticosteroid use and educate patients about the importance of adhering to the prescribed dose and duration of treatment. By increasing awareness of the varied clinical manifestations of this condition, healthcare providers can enhance the diagnosis and initiate appropriate management, ultimately leading to better outcomes for patients.

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Data Availability Statement: The article contains all the necessary data to support the results.

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