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Iatrogenic Cushing's Syndrome at the Regional University Hospital Center of Ouahigouya: A Case Report

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Abstract

Introduction: Iatrogenic Cushing's syndrome encompasses all clinical manifestations induced by chronic exposure to an exogenous excess of glucocorticoids. It is caused by long-term administration of corticosteroids, most often due to failure to adhere to the guidelines for the use of these medications. The clinical manifestations are numerous, some of which are specific. A chronological link between the administration of high doses of corticosteroids and the onset of clinical manifestations is fundamental. Cushing's syndrome is associated with increased mortality from cardiovascular complications, severe morbidity, and a reduced quality of life. **Case Report:** This was a 49-year-old male patient admitted to the hospital for evaluation of severe generalized asthenia, diffuse skin depigmentation, and progressive weight gain. The clinical examination revealed signs of protein hypercatabolism, including skin atrophy, vascular fragility, and wide stretch marks; as well as signs of facial-trunk fat redistribution characteristic of android obesity, with a rounded, erythematous face and a "bison hump". Evidence of prolonged administration of high-dose betamethasone tablets confirmed the drug-induced cause of the Cushing's syndrome presentation. The 8-hour serum cortisol level was significantly reduced to 3.62 µg/dl, confirming suppression of the adrenocorticotropic axis. The course of the illness was marked by the onset of acute pulmonary edema and death on the 16th day of hospitalization. **Conclusion:** Iatrogenic Cushing's syndrome is preventable by adhering to the indications and contraindications for corticosteroids. Hence, there is a need to raise awareness among healthcare providers regarding the importance of increased vigilance when prescribing and dispensing medications, particularly corticosteroids.

Keywords

Cushing's Syndrome, Corticosteroids, Stretch Marks

1. Introduction

Cushing's syndrome encompasses the full range of clinical manifestations caused by chronic exposure to excess glucocorticoids, which can result from a variety of causes [1].

It is considered rare, with an annual incidence of approximately one to six cases per million people per year [1] [2]. Cushing's syndrome can be iatrogenic in origin, resulting from long-term use of glucocorticoids or the administration of a treatment combining a glucocorticoid with medications that alter its pharmacokinetics. Its diagnosis requires both clinical and laboratory evidence. Clinical suspicion is based on the presence of signs of protein hypercatabolism, namely muscle and skin atrophy, vascular and bone fragility, and stretch marks; and a facial-trunk redistribution of fat manifested by android obesity, a moon-shaped, erythematous face, and a "bison hump" appearance. The diagnosis is confirmed by laboratory tests showing excessive cortisol secretion—as evidenced by urinary free cortisol levels more than 3 or even 4 times the normal range—or by evidence of disruption of the circadian rhythm of cortisol secretion, as indicated by a midnight serum cortisol level exceeding 3.6 μg/dl (100 nmol/l) or elevated midnight salivary cortisol levels [2] [3].

A chronological link between corticosteroid administration and the onset of specific signs, as well as discrepancies between the clinical presentation and hormonal testing, are arguments supporting an iatrogenic cause [4] [5].

Cushing's syndrome is associated with a reduced quality of life and excess mortality, primarily of cardiovascular origin; hence, the need for early diagnosis and treatment, along with long-term follow-up of patients.

The aim of this study was to present the consequences of the inappropriate use of high-dose, continuous corticosteroids, which led to the development of Cushing's syndrome in a 49-year-old patient. This is intended to help raise awareness among healthcare professionals and patients regarding the need to adhere to the indications and dosages for corticosteroids.

2. Case Report

2.1. Patient Information

The patient was a 49-year-old man admitted to the Department of Internal Medicine at the Ouahigouya Regional University Hospital for evaluation of severe generalized asthenia, diffuse skin depigmentation, and progressive weight gain.

The patient's medical history includes type 2 diabetes diagnosed three (03) years ago, treated with 1000 mg of metformin every 12 hours. The prescription and follow-up were managed by a nurse at a Health and Social Promotion Center

(CSPS). The patient's mother is reportedly diabetic and is being treated irregularly with metformin tablets.

2.2. Results of the Clinical Examination

The patient reportedly sought care at a CSPS for bothersome cramps. This led a nurse to prescribe betamethasone tablets. This prescription reportedly resulted in a reduction in the severity of the cramps, leading to the continuation of the treatment. The medical history revealed oral administration of high doses of betamethasone (2 mg) at a rate of 2 tablets "whenever he feels pain, or otherwise every 8 hours". The patient and his companions reported that he had been taking the medication orally as soon as he felt cramps—on average 5 to 6 times a day—and had been doing so continuously for 9 months to treat limb cramps. After noticing the gradual onset of skin depigmentation, the patient returned to see the healthcare provider, who insisted that he continue the treatment, especially since his symptoms had improved. The patient did not report using any topical medications.

Faced with progressive weight gain, generalized asthenia, and diffuse depigmentation, the patient sought care at our hospital.

The clinical examination of the patient upon admission revealed severe physical, psychological, and sexual weakness; diffuse skin depigmentation sparing the major skin folds; and facio-trunkal obesity characterized by a rounded face, a "bison hump" (**Figure 1**), and wide vertical stretch marks in the suprapubic, infrapubic, and lumbar regions (**Figure 2**), as well as at the roots of the thighs and on the arms. Numerous bruises and diffuse skin fragility were also noted, particularly on the upper limbs and pelvic region (**Figure 3**).

A gradual increase in body weight was observed, rising from 82 kg before the prescription of corticosteroids to 96 kg upon admission to our department—an increase of 14 kg over 9 months. Grade 1 obesity, corresponding to a body mass index of 31.71 kg/m^2, was also noted.

On admission, blood pressure was 150/95 mmHg, heart rate was 92 beats per minute, and oxygen saturation was 96% in room air. These findings were accompanied by severe epigastric abdominal pain, vomiting, polyuria, and polydipsia.

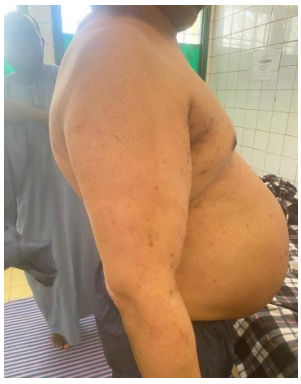
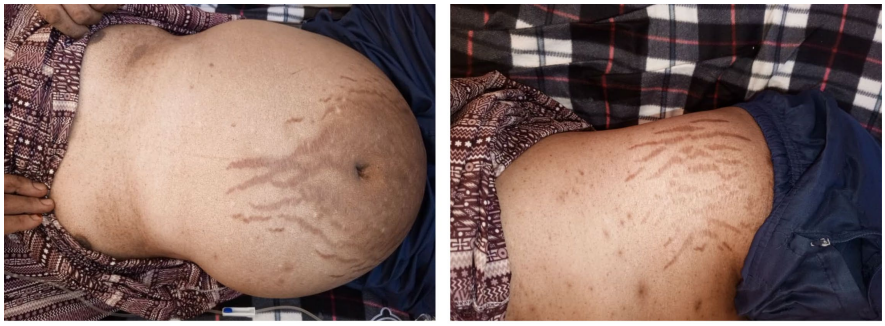
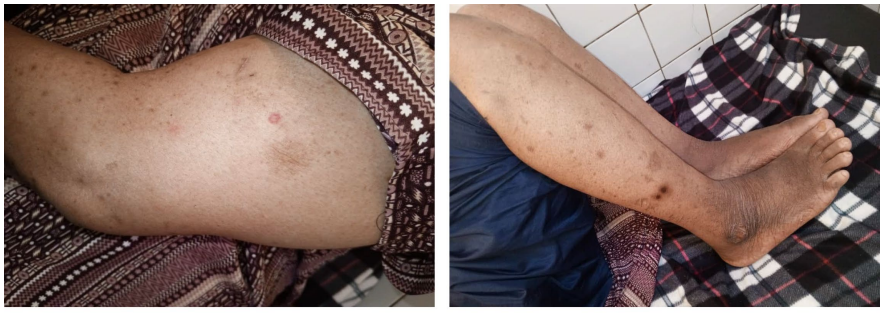


Figure 1. "Bison hump" + filling of the subclavicular depression. Sources: CHUR Ouahigouya.



(a) (b)

Figure 2. Stretch marks in the periumbilical and lumbar regions of patient O.A. (a) Stretch marks in the periumbilical region; (b) Stretch marks in the right lumbar region. Sources: CHUR Ouahigouya.



(a) (b)

Figure 3. Bruises on the limbs. (a) Bruises on the upper extremities; (b) Bruises on the lower extremities. Sources: CHUR Ouahigouya.

2.3. Diagnostic Workup

A blood glucose test performed in the emergency department upon admission showed a level of 22.5 mmol/L. The urine test strip revealed 3 crosses for glucose and traces of ketones. The blood electrolyte panel showed bicarbonate levels of 31.5 mmol/L, normal sodium levels at 141 mmol/L, hypokalemia at 3.12 mmol/L, and hypocalcemia at 1.73 mmol/L. Plasma osmolarity was 308.7 mOsm/L. These results led to an initial diagnosis of decompensated type 2 diabetes in the hyperosmolar state, which was managed with insulin therapy and electrolyte replacement.

Given the history of long-term corticosteroid therapy, the Cushingoid clinical presentation contrasting with severe asthenia, and the abrupt discontinuation of corticosteroid therapy one week prior; a cortisol level was measured, revealing adrenal insufficiency (8-hour cortisol level of 3.62 µg/dl = 100.48 nmol/l), confirming the diagnosis of iatrogenic Cushing's syndrome complicated by adrenal insufficiency in the context of hyperosmolar decompensation of diabetes.

Lipid profile testing revealed dyslipidemia characterized by elevated LDL cholesterol and triglycerides. Liver function tests were normal, specifically: transaminases, alkaline phosphatase, prothrombin time, total bilirubin, and conjugated bilirubin.

2.4. The Patient's Management

- Upon discontinuation of betamethasone, followed by treatment of adrenal insufficiency with injectable hydrocortisone at a dose of 50 mg every 8 hours, switched after 72 hours to hydrocortisone tablets at a dose of 20 mg per day;
- Continued treatment of hyperosmolar decompensation in type 2 diabetes by administering regular insulin intravenously every hour for 6 hours, followed by a switch to intermediate-acting insulin as basal insulin, regular insulin with each meal, and fluid and electrolyte replacement;
- Prevention of thromboembolic diseases using 0.6 ml of injectable enoxaparin every 24 hours.

2.5. Course of the Disease

The course of the illness was marked by a gradual resolution of asthenia with hydrocortisone and glycemic control following high doses of insulin, specifically Insulatard at a dose of 38 international units in the morning and 28 international units in the evening, combined with regular insulin at a dose of 8 units before each of the three meals, all administered subcutaneously.

However, during the second week of hospitalization, the patient developed orthopneic dyspnea and a dry cough; upon examination, clinical signs of global heart failure were observed, necessitating several additional tests. Blood pressure was 105/65 mmHg, pulse rate was 110 beats per minute, and oxygen saturation was 82%.

The electrocardiogram (ECG) was normal except for tachycardia, and the cardiac Doppler ultrasound revealed findings consistent with grade II aortic regurgitation, associated with impaired relaxation. Despite intervention by the cardiology team, with treatment administered according to guidelines, the patient's condition progressively worsened, and he died from acute pulmonary edema (APE) on the 16th day of hospitalization.

3. Discussion

The inability to perform certain tests and our patient's limited financial resources posed limitations. Additionally, the inability to measure cardiac biomarkers and perform an autopsy presented constraints in our clinical case.

This case report highlights certain therapeutic errors that can occur during the prescription process, particularly when prescriptions are issued by personnel not qualified to do so and without regard for the indications and contraindications of the prescribed medications. Given our limited resources, we diagnosed hyperosmolar decompensation based on the following criteria: the absence of Kusmaul's dyspnea and acetone-like breath odor, traces of ketonuria, plasma osmolarity greater than 300 mOsm/L, and plasma bicarbonate levels greater than 18 mmol/L. A ketonuria of 2 crosses or higher would have led us to include a ketotic decompensation in the diagnosis. This is not the case here.

From an epidemiological perspective, Cushing's syndrome—considered a rela-

tively rare but potentially fatal disease—has an annual incidence of approximately one to six cases per million inhabitants per year and is more common in women, according to several authors [4] [6]. This corroborates our data, as we diagnosed two cases of iatrogenic Cushing's syndrome over a two-year period in a region with a population of 1,939,300 in 2022 [7]. Iatrogenic Cushing's syndrome is rarely described in the literature. Inappropriate use of corticosteroids can lead to this condition [2]. This was the case for our patient, who received high-dose corticosteroid therapy for 9 months to treat limb cramps. The maximum dose was exceeded because the medication was taken as needed, and the patient and his caregivers stated that the medication was sometimes administered 5 or 6 times a day as soon as he felt the cramps. This amounts to an administration of 24 mg of betamethasone. However, the maximum dose for our patient should have been no more than 16 mg per day. The duration of treatment was very long, and discontinuation was abrupt. All of this is contrary to the precautions for the use of corticosteroids.

Clinically, there were specific signs and less specific signs. In our case, there were numerous specific signs, namely weight gain, stretch marks, bruising, and a "buffalo hump". Several authors have noted the same specific signs [8]-[10]. However, bone fragility was not objectively observed in our patient.

The classic side effects of corticosteroid therapy—namely, depigmentation, skin fragility, gastric lesions, and diabetic decompensation—were observed in our patient.

The patient's caregivers reported prolonged administration of high-dose corticosteroids, a finding confirmed by the patient himself. Biochemically, the blood electrolyte panel revealed hypokalemia and hypocalcemia, as described in the literature. This hypokalemia is thought to be related to the mineralocorticoid action of cortisol. The suppression of the adrenocorticotropic axis due to exogenous glucocorticoids explains why the patient presented with signs of adrenal insufficiency (notably severe asthenia), which was confirmed by low serum cortisol levels. Additional endocrine tests were not performed because they were not available at our hospital or within a 180-km radius.

The cramps experienced by the patient, which necessitated the prescription of corticosteroids, could be adverse effects of metformin. A thorough understanding of the side effects of antidiabetic medications and adherence to the indications and contraindications for corticosteroids would have allowed us to avoid this situation. The occurrence of iatrogenic Cushing's syndrome in the context of metabolic disorders (diabetes, dyslipidemia) has also been described by Behidj S. in Algeria [10].

From a therapeutic standpoint, betamethasone was discontinued and urgently replaced with injectable hydrocortisone, as the patient was considered to have acute adrenal insufficiency [11]. This hydrocortisone supplementation is recommended in the literature [11] [12].

Blood glucose control was achieved with high doses of insulin, which highlights

the hyperglycemic effect of corticosteroids [11]. Prophylactic anticoagulant therapy was initiated because the patient was bedridden and obese, to reduce the risk of thromboembolic events. This treatment consisted of subcutaneous enoxaparin injections.

The prognosis could have been favorable if diagnosis and treatment had been initiated early. This was not the case for our patient, in whom the diagnosis was made very late. Cushing's syndrome is rare and is usually associated with severe morbidity and excess mortality, particularly of cardiovascular origin. The cardiovascular complications of Cushing's syndrome account for its severity, with a mortality rate four times higher in these patients compared to healthy individuals. These complications are primarily due to a high prevalence of cardiovascular risk factors, namely hypertension, abdominal obesity, diabetes**,** dyslipidemia, and abnormalities in blood viscosity. This was the case with our patient, whose course was unfavorable, as it was marked by cardiovascular complications such as acute pulmonary edema [2] [4]. Our patient died on the 16th day of hospitalization with symptoms of acute pulmonary edema (APE). According to the literature, APE can occur in the context of Cushing's syndrome and is often exacerbated by chronic heart failure [2].

4. Conclusion

Iatrogenic Cushing's syndrome is a rare but serious condition that can lead to the development of cardiovascular complications. The presence of specific clinical signs, along with low serum cortisol levels in the context of prolonged administration of high-dose betamethasone, allowed us to establish the diagnosis. Healthcare professionals must be fully knowledgeable about the adverse effects of corticosteroids, as well as the adjunctive treatment and the pre-treatment assessment for corticosteroid therapy. Corticosteroid therapy should not be prescribed by paramedical staff. Strict adherence to the indications and dosages of corticosteroids is essential to prevent the onset of this condition.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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