Case Report

Dexamethasone-Induced Exogenous Cushing's Syndrome: Unintended Effects of Long-Term Therapy

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ABSTRACT

Glucocorticoids are essential pharmacological agents use to manage autoimmune diseases and other inflammatory conditions due to their strong anti-inflammatory and immunosuppressive properties. However, long term use can result in cushing syndrome. Additionally, sudden discontinuation of glucocorticoids can suppress the hypothalamic pituitary adrenaline axis, leading to adrenal atrophy and insufficient cortisol production. This can trigger Adrenal crisis.

Case report: In our case report, we reported the case of 32 year old male patient who developed the cushing syndrome after administration of Dexamethasone tablet for prolonged period. Due to prolonged use of dexamethasone, increasing the cortisol level in the body and leading to cushing syndrome (moon face, itching all over the body, thin skin, thin arm and legs, poor wound healing). After diagnosis Cushing Syndrome patient discontinue the Dexamethasone. Due to sudden discontinuation of dexamethasone the patient experienced adrenaline crisis (which released insufficient cortisol into the blood). The prominent way to avoid these conditions are to provide a patient counselling before initiating a corticosteroids and warning the patients about sudden withdrawal of corticosteroids.

Conclusion: This is the rare case report study of dexamethasone induced Exogenous Cushing syndrome.

Keywords: Exogenous Cushing syndrome, Dexamethasone, Corticosteroids, Adrenal crisis, cortisol.

INTRODUCTION

Cushing syndrome was diagnosed by the American neurosurgeon harvey cushing in 1912. Cushing syndrome is rare but significant endocrine disorder characterization by prolonged exposure to elevated level of cortisol, a crucial hormone produced by adrenal glands. This condition can arise due to various causes including endogenous factors such as cortisol secreting tumor (adrenal adenoma) or excessive Adrenocorticotropic hormone (ACTH) production, often associated with pitutary adenomas (cushing disease).[1] It may also result from exogenous sources such as prolonged use of glucocorticoids medications.[2] The incidence of CS varies in different studies from 0.7 to 2.4 per million populations per year. A more recent study reported a prevalence of 79 per million and incidence of 1.8 per million per year for CS.[3] Cushing Syndrome is defined as a prolonged increase in plasma cortisol levels that is not due to a physiological etiology. Although the most frequent cause of cushing syndrome is exogenous steroid use.[4] Comorbities include hypertension, type 2 diabeties mellitus, cardiovascular disease, cerebrovascular disease and opportunistic infection.[5]

Long term use of glucocorticoid medications (like Dexamethasone, Prednisone) can lead to Cushing Syndrome. These medication mimic the effect of cortisol, cortisol is a steroid hormone produced by zona fasciculata of the adrenal cortex. After production the cortisol is carried out to different parts of the body by binding "cortisol binding protein" and cause conformational change in the body like moon face, buffalo hump, pendulous (hanging) abdomen, flushed facial skin and poor wound healing.[6]

The aim of the study is to present a rare case of oral administration of corticosteroid induced iatrogenic CS in a 32 years old male patient.

CASE REPORT

A 32 year old male patient was admitted to the dermatology department with complaints of generalised weakness, moon face and insidious-onset, non projectile vomiting (4-5 episodes per day) occurring after food intake, with vomitus containing food particles, all persisting for the past 6 days. He is a known case of type 2 diabetes mellitus since 2 months on regular treatment with metformin 500mg daily.

He had a past history of skin rashes over the body for past 2 months and treatment with Tab Dexamethasone (4mg) twice daily for 2 months.

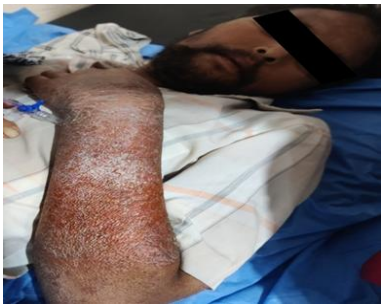

Figure 1: Cushing Syndrome on hand

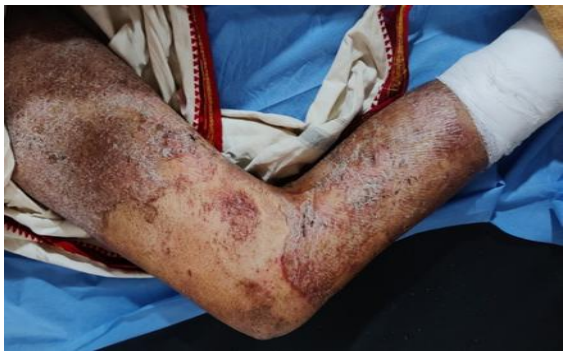

Figure 2: Cushing Syndrome on leg

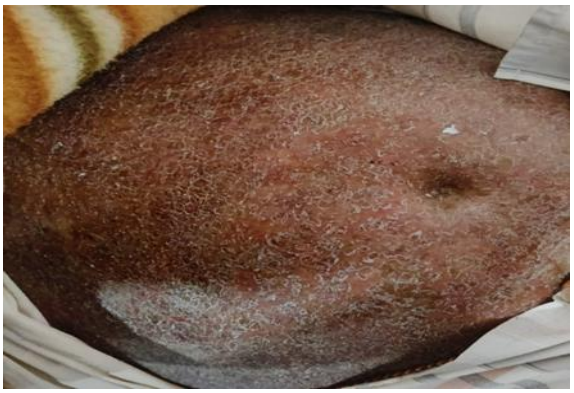

Figure 3: Cushing Syndrome on stomach

On examination, patient Bp was 128/82mmHg, PR: 84bpm and No abnormalities detected in systemic examinations. Physical examinations shows moon face or facial edema, itching all over the body, thin skin, thin arm and legs, poor wound healing (Fig 1,2 and 3). Test like CBC, RFT, LFT, Cortisol levels and serum electrolytes were evaluated and summerized in table 1.

Hormonal evaluation revealed an elevated morning serum cortisol level of 37.5µg/dl (normal value: 5-25µg/dl) and an elevated 24-hour urinary free cortisol level of 133.4µg/dl (normal value: <100µg/dl).

Based on the patients skin findings and hormonal abnormalities, doctors were diagnosed exogenous cushing syndrome with erythroderma secondary to tinea incognito, along with healing ulcers on right lower limb and dexamethasone was discontinued. Patient treated symptomatically with intravenous Inj. Avil, Inj. Pantaprazole, Inj. Ondasetrone, Inj. H.Albumin, Inj, H. Actrapid, Inj. Amicacin, Inj. Metrogly, Cap. Glospor, fudic cream and liquid paraffin. Due to sudden discontinuing of dexamethasone the patient experienced adrenal crisis followed by sever multi-organ failure and finally death.

Table 1: Evaluation parameters:

Investigations		**Observed value**	**Normal value**
Hb	Hb	11.8g/dl	13.5 – 17.5 g/dl
CBC	WBC	6000 cells/ul	4500 -10500 cells/ul
	RBC	4.27 million/ul	4.7 – 6.1 million/ul
	PLT	236000	150000-450000 cells/mm3
RFT	Sr.Urea Sr.Creatinine	18.0mg/dl 0.7mg/dl	7-20mg/dl 0.6-1.2mg/dl
LFT	ALT AST ALP	36.6U/L 40.1U/L 119.1U/L	6-38U/L 6-40U/L 35-149U/L
Cortisol	Morning serum cortisol 24hrUrinary free cortisol	**37.5 µg/dL ↑** **133.4 µg/dL ↑**	(normal: 5–25 µg/dL) (normal: <100 µg/dL).

DISCUSSION

In our case patient prolonged use of dexamethasone lead to cushing syndrome because dexamethasone mimic the effect of cortisol, sortisol is a steroid hormone produced by zona fasciculata of adrenal cortex. After production the cortisol is carried out to different parts of the body by binding “cortisol binding protein” (CBG) and excess of cortisol cause conformational change in the body and cause several effects, including increased gluconeogenesis and glycogenolysis, which lead to elevated glucose level and increased insulin resistance. Cortisol directly affects the transcription and translation of enzyme proteins involved in the metabolism of fats, glycogen, and proteins. Prolonged exposure to high cortisol levels can cause protein catabolism, resulting in purplish striae, osteoporosis, and poor wound healing. Additionally, cortisol promotes fat redistribution, leading to characteristic symptoms such as moon face, buffalo hump, and central obesity. These effects contribute to the development of cushings syndrome and its associated complications.

Treatment is done by tapering the dose of corticosteroids which may take a year. Sudden stoppage of corticosteroids after chronic intake can results in adrenal crisis. Slowly tapering the corticosteroids that is causing syndrome can helps to reverse the effects of adrenal gland atrophy. In our case patient is under adrenal crisis as he stopped the drug suddenly after diagnosed cushing syndrome the patient experienced adrenal crisis followed by sever multi-organ failure and finally death.

CONCLUSION

This case highlights the importance of judicious use of glucocorticoids and careful management of patients receiving prolonged corticosteroid therapy. Prolonged use of dexamethasone can lead to exogenous Cushing Syndrome and sudden discontinuation can result in adrenal crisis. Gradual tapering of corticosteroids is essential to prevent adrenal insufficiency and ensure optimal patient outcomes. Clinicians should be aware of the potential risks and benefits of glucocorticoids therapy and closely monitor patients for signs and symptoms of cushing syndrome and adrenal crisis.

CONSENT TO PARTICIPATE

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that their names and initials will not be published, and due efforts will be made to conceal their identity.

ABBREVATIONS

CS: Cushing Syndrome

ACTH: Adrenocorticotropic hormone

Hb: Hemoglobin

WBC: white blood cells

RBC: Red blood cells

PLT: Platelet

LFT: Liver function test

RFT: Renal function test

AST: Aspartate aminotransferase

ALT: Alanine aminotransferase

ALP: Alkaline phosphatase

Sr: Serum

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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