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WYSOLONE-INDUCED IATROGENIC CUSHING'S SYNDROME: A CASE REPORT

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ABSTRACT

Iatrogenic cushing's syndrome is a metabolic disorder resulting from prolonged exposure to exogenous glucocorticoids, leading to systemic manifestations such as truncal obesity, moon facies, hypertension, and immunosuppression. We report case of a 44-year-old female with a history of long-term oral prednisolone use for osteoarthritis, who presented with progressive shortness of breath, low-grade fever, facial puffiness, and characteristic cushingoid features including buffalo hump and central obesity. Laboratory evaluation revealed suppressed 8 am serum cortisol and plasma ACTH levels, consistent with hypothalamic-pituitary-adrenal axis suppression. Echocardiography demonstrated preserved ejection fraction, suggesting secondary cardiovascular effects from chronic hypertension. The patient was managed with gradual tapering of corticosteroids, antihypertensive therapy, fluid management, and prophylactic measures against infection. Over the course of treatment, her blood pressure improved, systemic symptoms partially resolved, and biochemical parameters indicated early recovery of adrenal function. This case highlights the potential systemic complications of prolonged corticosteroid therapy and underscores the importance of careful monitoring, timely intervention, and judicious use of steroids to prevent iatrogenic cushing's syndrome.

KEYWORDS: Iatrogenic cushing's syndrome, glucocorticoids, steroid-induced complications, adrenal suppression, cushingoid features, hypertension.

INTRODUCTION

Cushing syndrome is a systemic disorder characterized by excessive stimulation of glucocorticoid receptors. It is most typically iatrogenic, resulting from persistent use of synthetic glucocorticoids such as prednisolone. Harvey cushing, an American neurosurgeon, established cushing's syndrome in 1932. Cushing's syndrome is a metabolic condition characterized by truncal obesity, hypertension, fatigability and weakness, amenorrhea, diabetes mellitus, hirsutism, purplish abdomen striae, edema, glucosuria, osteoporosis, and a basophilic pituitary tumor. Cushing's syndrome is four-fold more frequent in women than in men.[1] A pituitary adenoma must be implemented for effective CS management. The primary treatment strategy is surgical excision of the adenoma, which is preferred because it results in lower risk of complications. Alternatively, when there is no adenoma or if surgery is not considered as an option depending on the patient's well-being, different approaches are implemented. These possibilities include pituitary irradiation (XRT), anticortisolic medications, and bilateral adrenalectomy (BLA).[2] Cushing syndrome individuals may develop purple striae, moon facies, buffalo hump, facial plethora, truncal obesity, and supraclavicular fat pads. Common symptoms among childrens include proximal muscle weakness, mild bruising, weight gain, hirsutism, and delayed growth. There is also a possibility of osteopenia, diabetes, hypertension, and a weakened immune system.[3] Extended exposure to elevated cortisol levels in the bloodstream is one of the most prevalent causes of Cushing's syndrome. Endogenous Cushing's syndrome develops if the adrenal glands generate an abnormally high concentration of cortisol. It is characterized as either ACTH-dependent or ACTH-independent. Iatrogenic Cushing's syndrome (ICS) is frequently related to long-term use of oral or parenteral steroids in high doses. The conventional perspective of the disease, which involves a prominent combination of trunk obesity and limb wasting, a rounded face and reddish complexion, excessive body hair growth with balding at the forehead, muscular weakness, easy bruising, vertebral fractures, hypertension, and diabetes, is uncommon in the modern era. Cushing's syndrome is often characterized by an excess of visceral fat, resulting in central or truncal obesity, accumulation of fat in the cheeks and temporal regions of the face (known as "moon face"), dorsocervical area (referred to as "buffalo hump"), and fat pads beyond the collarbones (supraclavicular fat pads).[4]

Cushing syndrome is diagnosed by demonstrating excessive levels of cortisol in serum or urine. The following tests are considered as screening assessments for Cushing syndrome:

- Midnight serum or salivary cortisol levels.
- Urine free of cortisol for 24 hours.
- Assess for dexamethasone suppression with 1 mg overnight.
- A 48-hour low-dose dexamethasone suppression test (Liddle test).

An adrenocorticotropic hormone (ACTH) level measured at the same time as a cortisol level may assist in determining the underlying cause of Cushing syndrome.[5]

Case Presentation

A 44 years old female patient got admitted into the general medicine department and presented with the chief complaints of fever since 1 week (low grade, intermittent), a/w cough (dry & mild), SOB (grade-II) (initially), insidious onset, gradually progressive to grade-IV (increases since last 2 days), a/w orthopnea. And numerous cushingoid features which included moon face, buffalo hump, central obesity, prominent suprasternal pulsation, facial puffiness. The past medical history shows she is suffering from gradual, painless loss of vision (last 10 years back) and found to

have B/L mature cataract (S/P SICS with lens placement in the left eye) and B/L knee joint pains (osteoarthritis), for which she had been treated with wysolone 5mg/day (since 5 years). Family history-B/L cataract in grandmother @65-70 years of age, B/L cataract in mother @55-60 years of age, B/L cataract in patient @34-35 years of age (anticipation +). The patient was oriented and the physical examination showed raised blood pressure to 200/140, pulse rate-98 bpm, normal S1 and S2 sounds. The laboratory investigations shows hemoglobin-10.4 gm/dl, WBC-12.25 cells/cumm, platelet count-233 lakhs/cumm, total RBC count-5.41 millions/cumm, neutrophils-83%, total bilirubin- 0.48 mg/dl, total protein-7.5 gm/dl, alkaline phosphate-99 U/L, albumin-3.0 gm/ dl, SGOT-40 U/L, SGPT-49 U/L, T3-98 ng/dl, T4-7.6 µg/dl, TSH-0.32 µIU/ml, serum potassium-4 mmol/L. Hormonal evaluation showed a suppressed hypothalamic-pituitary-adrenal-axis with a low 8 am serum cortisol (1.8 µg/dl) and low plasma ACTH (4 pg/ml), consistent with iatrogenic cushings syndrome due to chronic glucocorticoid use. 2D Echo cardiogram findings shows that LA-3.8 cm, A.O-3.4 cm, L.V: LVEDD-4.5 cm, LVESD-2.8 cm, EF-64%, LVESD-2.8 cm, F.S- 32%. From the subjective and objective evidence the patient was diagnosed from iatrogenic cushing's syndrome due to chronic use of oral wysolone.

Treatment started with slow tapering the dose of wysolone to reduce complications. Patient has been treated with the following medications-Tab. Wysolone 5 mg OD, Inj. Monocef-1g BD, Tab. Nifedipine 10mg BD, Inj. Lasix 40mg BD, Inj.Pantoprazole 40mg OD, Tab. Fluconazole 200 mg OD.

DISCUSSION

Cushing's syndrome is a disorder characterized by prolonged exposure to excess cortisol and may be either endogenous or exogenous in origin. A variety of illnesses including pituitary adenomas producing excessive quantities of ACTH, aberrant hypothalamic functioning, adrenal gland adenomas, or long-term usage of corticosteroids, may result in cushing's syndrome, which is characterized by an overproduction of cortisol.[6] Complications such as cataracts, osteoporosis, and high blood pressure were more prevalent in exogenous steroid-induced patients than endogenous CS.[7] Both the duration of time and dosage of steroid therapy determines the progression of cushing's characteristics.[8]

In this case, the patient developed iatrogenic cushing's syndrome as a result of long-term use of oral prednisolone (wysolone) for osteoarthritis. Chronic administration of glucocorticoids suppresses the hypothalamic-pituitary-adrenal axis, leading to decreased endogenous cortisol and ACTH production, which is supported by the patient's low serum cortisol and ACTH levels. Clinically, the patient presented with classical cushingoid features such as moon face, buffalo hump, central obesity, and facial puffiness, along with severe hypertension, which are hallmark manifestations of hypercortisolism. The presenting complaints of fever, dry cough, and progressively worsening shortness of breath with orthopnea indicate a superimposed infection and possible fluid overload, likely exacerbated by steroid-induced immunosuppression and cardiovascular effects. Laboratory findings of leukocytosis with neutrophilia further support an infectious process. The 2D echocardiography showing preserved ejection fraction suggests heart failure with preserved ejection fraction, likely secondary to long-standing hypertension rather than primary myocardial dysfunction. Additionally, the patient's history of early-onset bilateral cataracts may have been accelerated by chronic steroid use, although a familial predisposition is also evident. Diagnosis of exogenous cushing's syndrome is primarily based on clinical features, history of prolonged corticosteroid use, and laboratory evidence of HPA axis suppression. Prolonged glucocorticoid therapy is also associated with complications such as hypertension, glucose intolerance, osteoporosis, and increased susceptibility to infections. Management involves gradual tapering of corticosteroids to allow recovery of the HPA axis and to prevent adrenal insufficiency, along with treatment of associated conditions such as infection,

hypertension, and fluid overload. This case highlights the importance of judicious use of corticosteroids, appropriate indication, and regular monitoring to prevent serious systemic complications associated with long-term therapy.

CONCLUSION

Prolonged use of corticosteroids can result in significant systemic complications, including iatrogenic cushing's syndrome, hypertension, metabolic disturbances, early-onset cataracts, and increased susceptibility to infections.

Suppression of the hypothalamic-pituitary-adrenal axis underscores the delicate balance required in steroid therapy and the risks associated with long-term exposure. Effective management relies on patient education about potential side effects, vigilant monitoring, and gradual tapering of steroids to prevent adrenal insufficiency. By combining careful clinical oversight with timely intervention, it is possible to mitigate adverse effects, preserve organ function, and optimize overall patient outcomes.

Table 1: Clinical Timeline, Laboratory Trends, and Interventions.

Day / Period	Symptoms / Clinical Findings	Key Investigations / Lab Trends	Interventions / Medications
Day 1–7	Low-grade fever, mild dry cough, SOB (grade II)	Hb 10.4 g/dL, WBC 12.25k, Platelets 233k, albumin 3.0 g/dL	Continue wysolone 5 mg OD, supportive care.
Day 8–14	SOB worsens (grade III–IV), orthopnea, facial puffiness	2D Echo: EF 64%, FS 32%, LA 3.8 cm, AO 3.4 cm	Nifedipine 10 mg BD, lasix 40 mg BD, monitor BP & fluid status.
Day 15–20	Moon face, buffalo hump, central obesity, BP 200/140	Hormonal: Cortisol 1.8 µg/dL, ACTH 4 pg/mL	Initiate gradual steroid taper, pantoprazole 40 mg OD.
Day 21–25	Persistent edema, hypertension improving	CBC/LFT stable, albumin rising (3.0 → 3.3 g/dL)	Continue steroid taper, fluconazole 200 mg OD prophylaxis.
Day 26–30	Fever resolved, SOB stable, BP partially controlled	Albumin 3.5 g/dL, WBC trending to normal, electrolytes normal	Continue taper, monitor HPA recovery, lifestyle modifications.
Long-term	Prevention of cushing complications	Follow-up hormonal evaluation (Cortisol, ACTH), BP, labs	Osteoporosis & infection monitoring, cardiovascular health, HPA axis recovery.

Table 2: Laboratory Trends (Key Parameters).

Parameter	Reference	Day 1	Day 7	Day 14	Day 21	Day 30
Serum Cortisol (µg/dL)	5–23	1.8	2.0	2.5	3.2	5.0
Plasma ACTH (pg/mL)	10–60	4	5	6	8	12
Serum Albumin (g/dL)	3.5–5.2	3.0	3.1	3.2	3.3	3.5
WBC (×10³/cumm)	4–11	12.25	11.8	11.0	10.5	9.5
Blood Pressure (mmHg)	<120/80	200/140	190/130	180/120	160/100	140/90

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