

EXOGENOUS CUSHING SYNDROME SECONDARY TO CORTICOSTEROID THERAPY: A CASE REPORT WITH METABOLIC AND CLINICAL IMPLICATIONS

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ABSTRACT

Introduction: Cushing syndrome is an endocrine disorder caused by prolonged excess cortisol, most commonly due to extended use of exogenous glucocorticoids. Chronic steroid exposure suppresses the hypothalamic–pituitary–adrenal axis, resulting in decreased endogenous cortisol production and adrenal atrophy. Patients typically present with central obesity, facial rounding, thin skin, violaceous striae, proximal muscle weakness, hypertension, and diabetes. Early diagnosis through clinical assessment and biochemical evaluation is crucial for appropriate management. **Case Presentation:** A 68-year-old female presented with altered sensorium, high-grade fever, and vomiting for three days. On examination, she had mild tachycardia. Laboratory investigations revealed leukocytosis, hyperglycemia, dyslipidemia, hyponatremia, and abnormal serum cortisol levels. She was treated with intravenous

antibiotics, corticosteroids, insulin, antihypertensives, and supportive care. Upon stabilization, she was discharged on oral hydrocortisone, antidiabetic therapy, and other supportive medications. **Case discussion:** This case highlights exogenous Cushing syndrome as a preventable complication of prolonged glucocorticoid therapy. The patient's metabolic abnormalities and altered cortisol levels suggested steroid-induced suppression of the hypothalamic–pituitary–adrenal axis. As recommended by the Endocrine Society guidelines, a detailed medication history is central to diagnosis. Management included gradual hydrocortisone tapering and correction of metabolic disturbances. **Conclusion:** Exogenous

Cushing syndrome remains a common yet preventable cause of hypercortisolism. Rational steroid prescribing, careful monitoring, and gradual tapering are essential to prevent adrenal insufficiency and systemic complications, particularly in elderly patients.

KEYWORDS: Adrenal suppression, Corticosteroids, Hypothalamic–pituitary–adrenal axis suppression, Iatrogenic Cushing syndrome, Steroid-induced metabolic complications.

1. INTRODUCTION

Cushing syndrome is an uncommon endocrine disorder characterized by persistently elevated cortisol (hydrocortisone) levels in the body. The primary etiology of this condition is exogenous Cushing syndrome, which arises from prolonged administration of glucocorticoid medications, commonly known as corticosteroids. These synthetic agents are frequently prescribed for various medical conditions and mimic the physiological actions of endogenous cortisol. Chronic exposure to such drugs suppresses the hypothalamic–pituitary–adrenal axis, resulting in decreased adrenocorticotropic hormone (ACTH) secretion. Consequently, patients may demonstrate altered cortisol concentrations in blood or urine, depending on the type and duration of corticosteroid therapy.^[1]

Cushing's disease is frequently associated with hypertension, diabetes, obesity, osteoporosis, vascular complications, and decreased longevity. Early detection, achieved through thorough clinical evaluation, detailed endocrinological investigations, and appropriate imaging techniques, plays a crucial role in confirming the diagnosis and optimizing the success of surgical treatment.^[2]

1.1 PATHOGENESIS

Exogenous Cushing syndrome develops when persistent administration of synthetic glucocorticoids produces prolonged hypercortisolism, mimicking the effects of elevated endogenous cortisol levels. Chronic exposure to these steroids leads to suppression of the hypothalamic–pituitary–adrenal (HPA) axis through negative feedback on corticotropin-releasing hormone (CRH) and adrenocorticotropic hormone (ACTH), resulting in reduced endogenous cortisol production and adrenal atrophy. The excess glucocorticoid activity on peripheral tissues causes the characteristic Cushingoid features such as central obesity, facial plethora, and easy bruising.^[4]

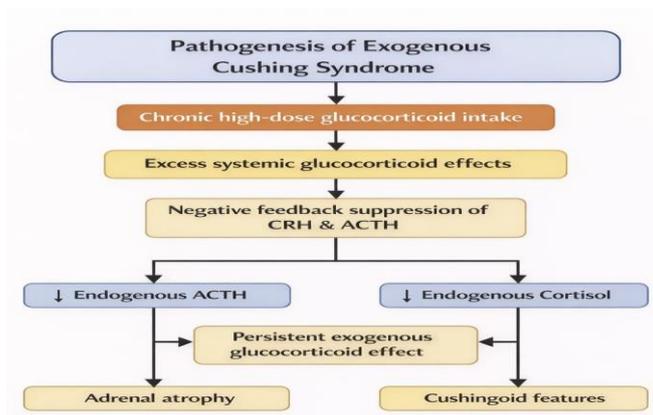


Fig. 1: Pathogenesis of exogenous Cushing syndrome.^[4]

1.2 CLINICAL FINDINGS

Cushing syndrome commonly presents with a distinctive pattern of clinical features. Affected individuals often develop central obesity accompanied by facial rounding with a plethoric appearance, along with excessive fat accumulation in the supraclavicular and dorsocervical regions. The skin typically appears thin and fragile, leading to easy bruising and ecchymoses, and is marked by wide, violaceous striae measuring more than 1 cm in width. Muscular involvement is evident as proximal muscle weakness due to myopathy.^[3]

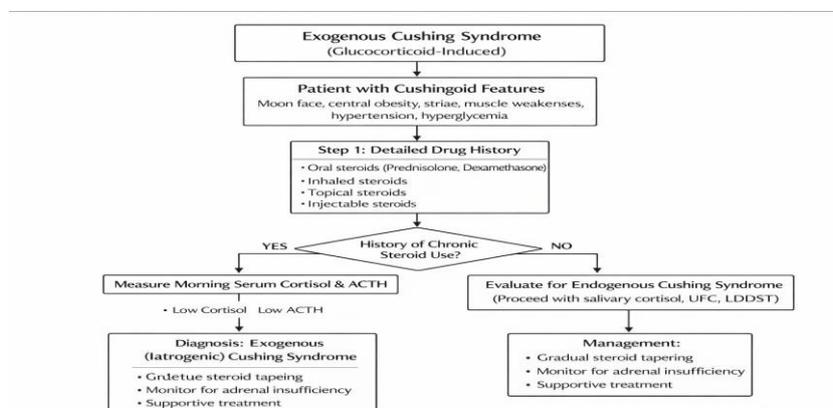


Fig. 2: The flowchart outlines a systematic approach to diagnosing exogenous Cushing syndrome.^[4]

1.3 PHARMACOLOGIC APPROACHES TO CUSHING SYNDROME: A CLINICAL SUMMARY

Drug	Mechanism of action	Recommended dose	Efficacy
Ketoconazole	Inhibits CYP11A1, CYP17A1, CYP11B1, and B2	200 to 1200 mg/day	Normal UFC in 50% to 66%
Levoketoconazole	Steroidogenesis inhibitor	300 to 1200 mg/day	Normal UFC in 50% to 81%
Metyrapone	Inhibits CYP11B1 and B2	500 to 6000 mg/day	Normal UFC in 42% to 47%
Osilodrostat	Inhibits CYP11B1 and B2	2 to 60 mg/day	Normal UFC in 77% to 83%
Etomidate	Inhibits CYP11A1, CYP17A1, CYP11B1	0.1 mg/kg/h	Scarce data
Mitotane	Steroidogenesis inhibitor	500 to 4000 mg/day	Normal UFC in 72%
Pasireotide	SSTR agonist (with SST5 affinity)	1.2 to 1.8 mg/day	Normal UFC in 15% to 26%
Pasireotide LAR	SSTR agonist (with SST5 affinity)	10 to 30 mg/4 weeks	Normal UFC in 41% to 42%
Cabergoline	Dopamine receptor subtype 2 (DRD2) agonist	0.1 to 7 mg/week	Normal UFC in 40%
Mifepristone	Glucocorticoid receptor competitive antagonist	300 to 1200 mg/day	60% improved hyperglycemia 88% improved Global Clinical Response
Cabergoline	Dopamine receptor subtype 2 (DRD2) agonist	0.1 to 7 mg/week	Normal UFC in 40%

Fig. 3: The figure shows the available medical treatments for Cushing syndrome, including different drugs, their mechanism of action, recommended doses, and effectiveness.^[5]

1. CASE PRESENTATION

A 68-year-old female patient was admitted to Ballari Medical College And Research Centre (BMCRC), Ballari (Karnataka) with chief complaints of altered sensorium in the form of reduced responsiveness patient has increased psychomotor activity and increased irritability. Fever which was high grade, continuous not subsiding on medications. Vomiting 3-4 episodes, acute onset 3-4 episodes with food particles since 3 days.

Social history:- Tobacco consumption positive (+)

Past history:- Nothing significant

Family history:- Nothing significant

On Examination :-The patient's blood pressure was recorded at 130/80 mmHg, and the pulse rate was 118 beats per minute. Oxygen saturation was 96% on room air. The random blood glucose level was 108 mg/dL. The patient was drowsy responsive to commands, bilateral pupils equal and reactive to light. On respiratory examination, bilateral normal vesicular breath sounds were present. Cardiovascular examination revealed normal S1 and S2 heart sounds. Per abdominal examination showed a soft and non-tender abdomen.

Table No. 1: Laboratory examination of the patient.

Parameters	Result (24-01-26)	Result (26-01-26)	Reference Range
Hemoglobin	10.7	12.7	12-16g/d
WBC	11410	10350	4500-11,000 cells/cumm

Polymorphs	85	70	40-75%
Lymphocytes	10	23	20-50%
MCHC	32.4	33.5	33-37g/dl
FBS	128	130	< 100mg/dl
PPBS	348	320	< 140mg/dl
Sodium	130		135-145mEq/L
Total cholesterol	329		< 200mg/dl
Triglycerides	278		< 160mg/dl
LDL	152		< 130mg/dl
VLDL	56		< 20mg/dl
LDL/HDL Ratio	3.2		< 2.5
HbA1C	6.8		< 6
MBG	149		
Sr. Cortisol	55.7		133-537nmol/L

2.1 TREATMENT CHART

Table 2: Treatment regimen in hospital.

Name of Medications	Dose	Route	Frequency
INJ. CEFTRIAZONE	2g	IV	BD FOR DAY 1
INJ. DEXAMETHASONE	8mg	IV	TID FOR 2 DAYS
INJ. PANTOPRAZOLE	40mg	IV	OD FOR 5 DAYS
INJ. ONDANSETRON	4mg	IV	BD FOR 5 DAYS
SYP. SUCRALFATE	5ml	PO	BD FOR 5 DAYS
INJ IVF-NS		IV	FOR 3 DAYS
INJ. MEROPENEM	1g	IV	TID FOR 5 DAYS
NEB. IPRAVENT		PN	QD FOR 5 DAYS
INJ. N-ACETYLCYSTEINE	@ 100ml (NS)	IV	BD FOR 5 DAYS
TAB. METHYL PREDNISOLONE	10mg	PO	BD FOR DAY 2-3
TAB. CALCIUM	500mg	PO	BD FOR DAY 2-5
INJ. HYDROCORTISONE	50mg	IV	TID FOR DAY 2-5
INJ INSULIN-R	4UNITS	SC	TID FOR DAY 3-5
TAB. AMLODIPINE	5mg	PO	BD FOR DAY 3-5
INJ. TRAMADOL	500mg in 100ml (NS)	IV	BD FOR DAY 3-5
SYP. AMBROXOL	5ml	PO	TID FOR DAY 4-5

2.3 DISCHARGE MEDICATIONS

Table No. 3: Discharge medications.

Name of Medications	Dose	Route	Frequency
TAB. HYDROCORTISONE	10/5/5	PO	OD FOR 15 DAYS
TAB. METFORMIN	500mg	PO	BID FOR 15 DAYS
TAB. GLIMIPRIDE	1mg	PO	BID FOR 15 DAYS
TAB. FAROPENAM	200mg	PO	BID FOR 15 DAYS
TAB. AMLODIPINE	10mg	PO	OD FOR 15 DAYS
TAB. N-ACETYLCYSTEINE	600mg	PO	BID FOR 15 DAYS
SYP. AMBROXOL	5ml	PO	BID FOR 15 DAYS
TAB. PARACETAMOL	500mg	PO	TID FOR 15 DAYS

2. DISCUSSION

Cushing syndrome is one of the clinical conditions which can occur due to prolonged exposure to glucocorticoids. Out of various etiological factors, prolonged use of corticosteroid is one of the main reason especially for exogenous Cushing syndrome. Whereas endogenous causes such as pituitary adenoma, exogenous cases are preventable in nature.

In our case, a 68 years old female patient presented with altered sensorium, fever, vomiting, irritability along with metabolic abnormalities. Objective examination revealed that PPBS (348mg/dl), triglycerides (278 mg/dl), total cholesterol (329mg/dl), sodium (130mEq/L) and serum cortisol levels were abnormal. Based on this subjective and objective evidences along history of steroid exposure strongly suggests steroid induce hypothalamic pituitary adrenal suppression leading to exogenous Cushing syndrome.

As per Endocrine Society Of Clinical Practice Guidelines, steroid administration is one of the leading cause of Cushing syndrome and diagnosis relies purely on medication history interview for the patient rather than blood testing.

Metabolic abnormalities as noticed in patient, i.e., hyperglycemia and dyslipidaemia are well recognised symptoms of prolonged steroid exposure. Along with this, elevated HbA1C (6.8%) suggest steroid induce worsening and glycemic control. Management usually focuses on gradual tapering of corticosteroid rather than sudden discontinuation.

In our case patient was discharged with tapering dose of hydrocortisone (10/5/5 regimen) which is standard treatment guidelines for HPA axis recovery. Use of oral hyperglycaemic agents (Metformin and Glimpiride) and Antihypertensives reflects need for comprehensive metabolic management.

3. CONCLUSION

Exogenous Cushing syndrome remains most common and preventable cause of hypercortisolism, resulting from prolonged or irrational steroid use. This case demonstrate how prolonged steroid exposure can lead to significant metabolic, neurologic and immunological complications specially in elderly patients. Early recognition to detailed medication history interview, appropriate blood test and regular monitoring of metabolic parameters are essential. Gradually tapering of steroid, rather than abrupt cessation is

important to prevent adrenal insufficiency. This case emphasizes importance of rational steroid prescribing, patient counselling, ADR monitoring and individualized tapering strategies.

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