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Steroid Induced Exogenous Cushing's Syndrome with Biochemical Cortisol Suppression: A Clinical Case Report

P. Sarmas Vali*[1], Mayur P[2], Mohamed Shakeeb[3], G. Harshavardhan[4]
Dr. Syed Mohammed Hussaini[5]

IV Pharm D[1,2,3,4] and Junior Doctor, [5]Sehat Hospital, Ballari.
[1,2,3,4] Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104

Corresponding Author: P. Sarmas Vali
Email: sarmas31aug@gmail.com

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Abstract: Cushing's syndrome is an endocrine disorder caused by prolonged exposure to excess cortisol. Exogenous Cushing's syndrome, the most common form, results from long-term glucocorticoid therapy, leading to suppression of the hypothalamic–pituitary–adrenal axis and reduced endogenous cortisol production. It is associated with significant metabolic, cardiovascular, and infectious complications. Common clinical features include central obesity, moon face, buffalo hump, thin skin, violaceous striae, proximal muscle weakness, hypertension, and hyperglycemia.

***Case Presentation*:** A 70-year-old female with chronic obstructive pulmonary disease and hypothyroidism presented with cough, breathlessness, fever, and bilateral lower limb edema. Examination revealed hypertension, tachycardia, bilateral rhonchi, and Cushingoid features. Laboratory findings showed neutrophilia, altered thyroid profile, hypoproteinemia, and reduced serum cortisol levels, suggestive of hypothalamic–pituitary–adrenal axis suppression secondary to prolonged corticosteroid use, including inhaled beclomethasone. She was managed with antibiotics, systemic corticosteroids, bronchodilators, insulin, antihypertensives, diuretics, and supportive therapy, and improved clinically.

Case Discussion: The patient's presentation was consistent with exogenous Cushing's syndrome complicated by infective exacerbation of COPD and metabolic disturbances. Careful medication review and monitoring were essential for diagnosis and management.

Conclusion: Early recognition, detailed medication history, gradual steroid tapering, and regular monitoring are crucial to prevent complications and ensure safe long-term corticosteroid therapy.

Keywords: Iatrogenic hypercortisolism, Adrenal suppression, Glucocorticoid excess, Chronic steroid therapy.

INTRODUCTION

Cushing's syndrome is an uncommon endocrine disorder characterized by persistently elevated levels of cortisol (hydrocortisone) in the body. Exogenous Cushing's syndrome (ECS) is the most common form and occurs in individuals

who use glucocorticoids, also known as corticosteroids or steroid medications. This condition develops when synthetic glucocorticoids are administered for the treatment of various medical disorders. These drugs mimic the physiological effects of cortisol and, when used in high doses or for prolonged periods, produce features of hypercortisolism.[1]

In exogenous Cushing's syndrome, the external steroid suppresses the hypothalamic–pituitary–adrenal (HPA) axis, leading to reduced adrenocorticotropic hormone (ACTH) secretion. As a result, endogenous cortisol production becomes low. Depending on the type, dose, and duration of corticosteroid therapy, laboratory findings may show decreased ACTH levels and altered cortisol levels in blood or urine. Early recognition through careful clinical evaluation and medication history is essential for appropriate management and safe tapering of steroid therapy.[1]

EPIDEMIOLOGY:-

Cushing's syndrome is a rare endocrine disorder, with an estimated annual incidence of approximately 1–3 cases per million people. The overall prevalence is around 40 cases per million individuals. However, biochemical hypercortisolism is reported more frequently among patients with poorly controlled diabetes mellitus and hypertension, affecting nearly 2–5% of such individuals. The condition shows a marked female predominance, with a ratio of approximately 3:1 compared to males.[2]

Cushing's disease is particularly uncommon in children and is most frequently diagnosed in adults, especially during the third and fourth decades of life. If left untreated, Cushing's disease can be life-threatening. It is associated with significant morbidity and increased mortality, primarily due to cardiovascular complications, recurrent infections, and psychological or psychiatric disturbances.[2]

PATHOGENESIS:-

Cushing's disease (CD) arises from a benign, monoclonal tumor of the pituitary corticotrope cells that produces excessive amounts of adrenocorticotropic hormone (ACTH). The elevated ACTH levels stimulate the adrenal glands to secrete cortisol in amounts far beyond normal physiological requirements. Persistently high circulating cortisol disrupts the normal diurnal rhythm of cortisol secretion and suppresses corticotropin-releasing hormone (CRH) release from the hypothalamus through negative feedback mechanisms. However, the pituitary adenoma remains partially resistant to this feedback inhibition. As a result, despite elevated cortisol levels, the tumor continues to secrete excessive ACTH.[3]

Therefore, Cushing's disease is characterized by reduced CRH secretion, inappropriately high ACTH levels, and sustained overproduction of cortisol.[3]

CLINICAL MANIFESTATIONS:-

The clinical presentation is predominantly characterized by central (truncal) obesity, accompanied by distinct physical changes resulting from chronic cortisol excess.[4]

These include:-

- Facial rounding with facial plethora, giving a "moon face" appearance
- Accumulation of fat in the supraclavicular and dorsocervical regions (commonly described as a "buffalo hump")
- Skin thinning (cutaneous atrophy) with easy bruising and ecchymoses
- Broad, violaceous striae measuring more than 1 cm in width, typically over the abdomen, thighs, or breasts
- Proximal muscle weakness (myopathy), particularly affecting the shoulder and hip girdle muscles
- Increased fine body hair (lanugo hair)
- Recurrent or persistent superficial fungal infections due to immunosuppression
- Growth retardation in children, often accompanied by weight gain.[4]

CASE PRESENTATION:-

A 70-year-old female patient was admitted to Ballari Medical College And Research Centre (BMCRC), Ballari (Karnataka) with chief complaints of cough with expectoration, cough mild-moderate expectoration whitish, non blood tinged. Breathlessness-insidious onset, gradually progressive, breathlessness on minimal activity. Fever low grade, continuous, not subsiding with medications. Bilateral lower limb swelling positive[+], Patient was unable to ambulate the pain and swelling since 4days.

Social history:- Nothing significant

Past history:- COPD on occasional nebulization since 10 years, Hypothyroidism on medications since 5 years.

Past medication:- Tab. Thyroxine 100mg, Beclomethasone dipropionate and lev osalbutamol inhaler

Family history:- Nothing significant

PICCLE:- Edema positive[+]

On Examination :-The patient's blood pressure was recorded at 160/100 mmHg, and the pulse rate was 104 beats per minute. Oxygen saturation was 98% on room air. The random blood glucose level was 144 mg/dL. The patient was conscious and oriented. On respiratory examination, bilateral normal vesicular breath sounds were present, bilateral rhonchi positive[+]. Cardiovascular examination revealed normal S1 and S2 heart sounds. Per abdominal examination showed a soft and non-tender abdomen.

Table 1:- Laboratory examination of the patient

Parameters	Result	Reference Range
Hemoglobin	11.3	12-16g/d
Polymorphs	80	40-75%
Lymphocytes	16	20-50%
MCH	26	27-33pg/cell
RDW-CV	16.3	11.5-14.5%
TSH	0.06	0.27-4.2mcIU/mL
T4	0.5	0.92-1.68ng/L
TOTAL PROTIEN	5.8	6-8.3g/dl
GLOBULIN	2.2	2.5-3.0g/dl
A/G RATIO	1.7	1.2-1.5
SERUM CORTISOL	61.80	133-537nmol/L





Fig. 1:- Clinical image demonstrating facial rounding (moon face) and prominent dorsocervical fat pad ("buffalo hump") in a patient with exogenous cushing syndrome secondary to prolonged corticosteroid use.

TREATMENT:-

Table 2:-Treatment regimen in hospital

Name of Medications	Dose	Route	Frequency
INJ. CEFTRIAXONE	1g	IV	BD FOR 8 DAYS
TAB. DOXYCYCLINE	100mg	PO	BD FOR 6 DAYS
INJ. PANTOPRAZOLE	40mg	IV	OD FOR 7 DAYS
INJ. ONDANSETRON	4mg	IV	BD FOR 7 DAYS
SYP. SALBUTAMOL	5ml	PO	TID FOR 8 DAYS
NEB. BUDESONIDE		PN	TID FOR 6 DAYS
NEB. LEVOSALBUTAMOL AND IPRATROPIUM BROMIDE		PN	TID FOR 8 DAYS
INJ INSULIN-R	6-6-4	SC	TID FOR 8 DAYS
TAB. AMLODIPINE	10mg	PO	OD FOR 8 DAYS
TAB. TELMISARTAN	40mg	PO	OD FOR 6 DAYS

INJ. HYDROCORTISONE	100mg	IV	TID FOR 8 DAYS
TAB. THYROXINE	100mg	PO	OD FOR 8 DAYS
TAB. METAPROLOL	25mg	PO	BD FOR DAY 4-5
SYP. LACTULOSE	20ml	PO	TID FOR DAY 3-8
TAB. ASPIRIN	75mg	PO	OD FOR DAY 3-8
TAB. ATORVASTATIN	40mg	PO	OD FOR DAY 3-8
INJ. FUROSEMIDE	40mg	IV	BD FOR DAY 3-8
TAB. SPIRANOLACTONE	50mg	PO	OD FOR DAY 6-8

DISCHARGE MEDICATIONS:-

Table 3:-Discharge medications

Name of Medications	Dose	Route	Frequency
NEB.BECLOMETHOSONE AND LEVOSALBUTAMOL	50mcg+50mcg	PO	OD
TAB. ASPIRIN	75mg	PO	OD FOR 15 DAYS
TAB. ATORVASTATIN	40mg	PO	OD FOR 15 DAYS
TAB. FUROSEMIDE	40mg	PO	BID FOR 15 DAYS
TAB. SPIRANOLACTONE	25mg	PO	BID FOR 15 DAYS
TAB. AMLODIPINE	10mg	PO	BID FOR 15 DAYS
TAB. THYROXINE	75mg	PO	OD FOR 15 DAYS
SYP. AMBROXOL	5ml	PO	OD FOR 5 DAYS
TAB. CEFEXIME	200mg	PO	BID FOR 5 DAYS

DISCUSSION:-

Cushing syndrome represents a phase of excess of glucocorticoid levels and will be broadly classified into endogenous and exogenous causes. Exogenous cushing syndrome is more common than endogenous etiologist due to prolonged exposure of corticosteroid therapy.

In our case a 70 years old female with history of coronary obstructive pulmonary disease has been on inhaled corticosteroid i,e beclomethasone for long term and received Injection hydrocortisone during hospitalization. Even though inhaled corticosteroids are generally considered safer, In elderly individuals on prolonged use can lead to systemic absorption to cause hypercortisoli sm. Age related physiological changes, Co-morbidis and possible altered drug metabolism may precipitate the risk as well. Clinically our patient exhibited symptoms of cushing syndrome such as moon face, dorsocervical fat pad (buffalo hump) suggesting chronic glucocorticoid exposure. Along with this, hypertension (160/100mmHg), hyperglycaemia (RBS:- 144 mg/dl), proximal weakness and bilateral pleural effusion were consistent with cortisol induced metabolic and cardiovascular effects.

Objective evidences revealed patient serum cortisol levels were reduced (61.80 mmol/L) which indicates exogenous cushing syndrome. In ECS, prolonged steroid administration suppresses ACTH secretion via negative feedback. Patients history of hypothyroidism further adds up as a complication. Glucocorticoids can suppress TSH secretion and alter peripheral thyroid hormone metabolism. Hence follow up of thyroid function tests in patients receiving steroids require careful clinical correction patient presented with cough, Expectoration, Fever and rhonchi suggestive of infective exacerbation of COPD.

This case Emphasises importance of medication reconciliation and review. Polypharmacy was present in this patient including antibiotics, antihypertensives, insulin, diuretics, steroids, and thyroid replacement therapy. Abrupt discontinuation of steroids in ECS is contraindicated due risk of adrenal crisis resulting from suppressed adrenal function. This case also highlights need for periodic monitoring of Blood pressure, Blood glucose, Electrolyte,

Bone mineral density in patients receiving corticosteroids.

CONCLUSION:-

Our case demonstrates a classical presentation of exogenous cushing syndrome secondary to prolonged corticosteroid exposure. Despite of low serum cortisol levels, Presence of characteristics clinical features and history of steroid use strongly supported diagnosis of HPA axis suppression due to exogenous glucocorticoids. This case emphasises on importance of detailed medications history, Careful interpretation of lab findings and regular monitoring in patient receiving long term steroid therapy. Early identification of cushing features can present serious complications such as HTN, DM, Recurrent infections, Osteoporosis and Adrenal crisis.

This case report highlights the important role of Clinical pharmacist in Pharmacovigilance, Patient counselling, Rational steroid use and Designing safe tapering strategies.

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