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CASE STUDY

UNEXPECTED ENDOCRINE CHAOS FEATURING IATROGENIC CUSHING'S: A CASE PRESENTATION

Sumol Ratna*[1], Simran Gangwani[2], Tanishqua[2], Arshita Gupta[2], Ananya Aggarwal[2], Muskan Jain[2] and Divyansh Singh[2]

[1]Assistant professor, Department of General Medicine, Noida International Institute of Medical Sciences, Gautam Budh Nagar, Uttar Pradesh, India
[2]UG MBBS, Noida International Institute of Medical Sciences, Gautam Budh Nagar, Uttar Pradesh, India

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Corresponding author:** ***Sumol Ratna

ABSTRACT

Iatrogenic Cushing Syndrome (ICS) is a condition caused by prolonged exposure to exogenous glucocorticoids, often prescribed for the treatment of various inflammatory and autoimmune diseases such as asthma, arthritis, or dermatological conditions. From this perspective, we are delineating a case report of a38 year-old male diagnosed with the same resulting due to long term prednisolone therapy prescribed for polyarthritis.

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INTRODUCTION

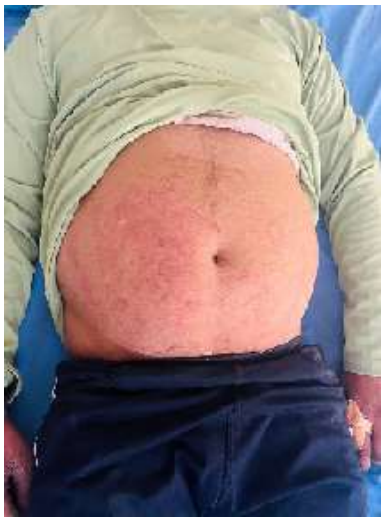
Cushing syndrome is an endocrinological disorder named after an American neurosurgeon Doctor Harvey Cushing. It is an ailment of the adrenal glands reflecting a constellation of clinical manifestations that result from chronic exposure to excess glucocorticoids of any aetiology (Mark, 2010). The disorder can be ACTH- dependent (pituitary corticotrope adenoma, ectopic secretion of ACTH by nonpituitary tumour) or ACTH-independent (adrenocortical adenoma, adrenocortical adenocarcinoma, nodular adrenal hyperplasia) as well as iatrogenic (administration of exogenous glucocorticoids to treat various inflammatory conditions) (Newell-Price, 2006). It occurs with an incidence of 1-2 per 100,000 lakh population per year. It is mostly recognisable by the characteristic physical features of moon facies, enlarged dorsal cervical fat pad, easy bruising and truncal obesity (Nieman et al;., 2008 and Ilias, 2005). However, patients are also at a risk of developing host of systemic disturbances including hypertension, glucose intolerance, decreased bone density and hirsutism along with menstrual irregularities in women (Pivonello, 2010). It has got diverse aetiology, the most common being due to excess use of glucocorticoids (iatrogenic Cushing syndrome) prescribed for various inflammatory conditions (Pivonello, 2010 and Van der Pas, 2013). Therefore, pharmacovigilance of the steroids is a requisite to avoid the detrimental effects it can predispose (Van der Pas, 2013). Mostly it is a clinical diagnosis which is established by increased 24-hour urinary free cortisol excretion, failure to appropriately suppress morning cortisol after overnight exposure to dexamethasone and evidence of loss of diurnal cortisol secretion with high levels at midnight, the time of the physiologically lowest secretion (Cushing, 1932). Treatment for the same involves a tapering regimen with subsequent biochemical testing of the HPA axis once glucocorticoids have been reduced to a physiologic dose. Also, sudden withdrawal of the glucocorticoids should be avoided to prevent adrenal crisis (Lindholm, 2001).

CASE REPORT

A38 year-old male presented to the general medicine department with 2-3 months history of shortness of breath, facial puffiness, and rashes all over his body found progressive in nature. The patient had no history of hypertension, diabetes mellitus, thyroid disorders, or any lung pathologies. However, his past medical history is notable for polyarthritis diagnosed 5-6 years back for which he took prednisone as prescribed by a local practitioner.

Buffalo hump

Moon facies



Violaceous striae

The patient underwent serological and laboratory testing that revealed negative serological markers, Hb - 10gm/dl, RBS - 140mg/dl, ACTH - 23.5pg/ml and cortisol - 33.4 mcg/dl that led to a suspicion of Cushing syndrome. He was advised to undergo dexamethasone suppression test. A brief general examination ofthe patient revealed normal vitals, pallor, moon facies, buffalo hump, supraclavicular fat deposition, violet striae bilateral over his arms, thighs, and abdomen along with centripetal obesity and bilateral pedal oedema. Laboratory findings along with the clinical manifestations culminated into a definitive diagnosis of iatrogenic Cushing syndrome that developed due to prednisone intake prescribed for polyarthritis. Symptomatic and supportive treatment was done after the final diagnosis based on laboratory findings.

DISCUSSION

Cushing syndrome subordinate to prednisone therapy, peculiarly in the context of polyarthritis is a notable clinical issue due to indelible use of corticosteroids in managing the same. In our case, prednisone prescribed for polyarthritis did reduce the inflammation of joints but it's prolonged use suppressed the HPA axis through negative feedback and resulted in reduced endogenous cortisol production. Characteristics clinical features like moon facies, buffalo hump, violet striae and centripetal obesity should alarm the health care professionals for the developing ICS and they should start with the tapering regimen. Higher doses or long-term use of glucocorticoids increase the risk of ICS therefore alternatives to corticosteroids like disease modifying anti-rheumatic drugs (DMARDS) should be handed down. Clinical diagnosis and endocrine evaluation should be employed to look out for the cortisol and ACTH levels for expeditious diagnosis. Diagnosis of Cushing's should be done as soon as the history of long-term glucocorticoids is presented as so to prevent the further complications of the same.

CONCLUSION

In a nutshell, the prevention of ICS grounds on a multifaceted approach that emphasises on vigilant corticosteroids prescriptions, careful patient management along with patient education on the adverse effects steroids can have. Reducing unnecessary exposure to corticosteroids is paramount, requiring health care providers to opt for lowest effective dose and shortest duration of therapy possible. As glucocorticoids are the mainstay of most of the inflammatory and autoimmune disorders therefore regular monitoring of patients undertaking the same, especially those on long term therapy, allows for early detection of symptoms indicative of excessive glucocorticoid exposure. In the long run, prevention of ICS necessitates an alliance between health care providers and the patients to ensure that corticosteroid use is both safe and effective.

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