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Steroid Induced Cushing Syndrome: A Case Report

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ABSTRACT

Glucocorticoids are effective steroid medicines that reduce inflammation. They are often used to treat asthma, allergic disorders such as psoriasis, dermatitis, and rheumatoid arthritis. Long-term corticosteroid use causes symptoms such as moon face, buffalo hump, pink stretch marks, and weight gain. Here we report a 31-year-old female patient developed Cushing syndrome due to continuous prednisolone use. She had rheumatoid arthritis in the last six months and is currently taking prednisolone. She has been experiencing symptoms such as moon face, backache, edema, abdominal distension, muscle weakness, tingling and numbness of both lower limbs till hip region in the last three weeks. Her serum cortisol levels are high.

Keywords: Prednisolone, Cushing syndrome, rheumatoid arthritis, cortisol, moon face, steroids.

INTRODUCTION

A syndrome with purplish abdominal striae, amenorrhea, hirsutism, hypertension, obesity, edema, glucosuria, osteoporosis, and a basophilic tumor of the pituitary were described by American neurosurgeon Dr. Harvey Cushing in 1932.It can also be referred to as hypercortisolism because it is brought on by extended exposure to high levels of cortisol in the blood. Glucocorticoids are significant pharmacological agents that function as immune system stimulants and inflammation reducers to treat a variety of illnesses. On the other hand, if glucocorticoids are used inappropriately, they can cause a number of adverse effects, such as Cushing's syndrome and the suppression of the hypothalamic-pituitary axis[1]. Cushing's syndrome has two primary etiologies: endogenous hypercortisolism and exogenous hypercortisolism. The most frequent cause of Cushing's syndrome, known as exogenous hypercortisolism, is primarily iatrogenic and arises from long-term glucocorticoid use[2]. Endogenous Cushing's syndrome, which can be ACTH-dependent or ACTH-independent, is caused by the adrenal glands producing excessive amounts of cortisol. ACTH-dependent Cushing's syndrome is caused by ectopic ACTH secretion by neoplasms and pituitary adenomas that secrete ACTH; on the other hand, adrenal hyperplasia, adenoma, and carcinoma are the main causes of ACTH-independent Cushing's syndrome[3]. Understanding the pharmacokinetic characteristics, daily dosage, frequency, and variations in each person's steroid metabolism is essential to avoiding side effects associated with glucocorticoid overuse[4].

CASE REPORT

A 31 years old female patient came with chief complaints of moon face, weakness of Both lower limbs since 1-year insidious onset,

progressive in nature. Unable to get up from supine position: 20 days Tingling sensation/ Numbness in both Lower limbs till hip region, cotton wool sensations associated pain, involving both Joints, tingling of both upper limbs up to wrist since 1 year, wasting of both Lower limbs since 2 years. There was no associated fever. Her past Medical History was a known case of seropositive Rheumatoid Arthritis (on steroids), a Known case of CAD, HTN. On the day of admission patients Vitals were found to be PR-99bpm, BP-120/70mmHg, CVS-S1S2+, RS-BAE, P/A - Distended, soft. So based on subjective and objective evidence, the patient is provisionally diagnosed as having

Cushing syndrome due to the chronic use of steroids. For the present complaints & conditions, based on the Vitals she was treated with T. Hydro chloroquine 200mg. BD, I. Opti neuron 1amp in 100ml NS OD, T. Pan 40mg OD, T.MVT OD & prednisolone dose

is tapered. Laboratory studies revealed that Hb-12.1g/dl, WBC-16,670, Na- 116, K-4.11, Cl-82, Sr. cortisol – 30.4mcg/dl, ENMG findings- Mild motor Axonal neuropathy of bilateral lower limbs. After all the investigations patient was treated with. following drugs such T. Cat Vit D 500mg Hydro chloroquine -200mg Bo. OD, T. Gaba NT 100/10 OD, T. Lasix 40mgBD, T. Enalapril 2.5mg BD, T. Met-XL 50mg BD, T. Dapagliflozin 10 ug OD, T. Methotrexate. 7.5mg OD, T. Leflunomide song BD.

DISCUSSION

Pituitary tumors and ectopic production of the hormone ADTH are examples of endogenous causes of Cushing syndrome, while exogenous causes include external delivery of corticosteroids. Exogenous Cushing's syndrome is a disorder that arises from extended exposure to the therapeutic use of corticosteroids. Another name for them is steroid-induced

Iatrogenic Cushing syndrome, also known as Cushing's syndrome[1]. Cushing’s disease patients typically exhibit one or more symptoms that are a result of the presence of too much ACTH or cortisol[5]. Unless the patient is taking a corticosteroid, serum cortisol levels are low in exogenous Cushing’s syndrome.

A round, red, full moon face, growth retardation in children, skin infections, purple marks (striae) on the skin of the breast, abdomen, and thighs, thin skin that is easily bruised, back pain during routine activities, fat deposit between the shoulders and above the collar bone, hip and shoulder muscle weakness, and fractures of the ribs and spine due to thinning of the bones are the most common symptoms of Cushing syndrome patients. Common laboratory findings associated with Cushing syndrome include low ACTH, low ACTH stimulation, elevated fasting blood sugar, decreased serum potassium, elevated blood cholesterol, and decreased bone density.

The corticosteroid dose is tapered off as part of the treatment, which could take a year. Adrenal crisis may occur from abrupt withdrawal of corticosteroids following a prolonged period of use. One way to counteract the effects of adrenal gland atrophy is to gradually reduce the amount of corticosteroid causing Cushing syndrome[6].

In clinical practice, glucocorticoids are frequently used to treat autoimmune, inflammatory, and allergic diseases. The most frequent use of glucocorticoids is irrational, especially when used in long-term treatments. This can have a variety of negative effects, including suppression of the hypothalamic-pituitary-adrenal axis, Cushing's syndrome, an increased risk of infections, and mental abnormalities. The factors affecting the therapeutic and side effects of glucocorticoids are the pharmacokinetic characteristics of the drug, individual differences in steroid metabolism, daily dosage, and length of treatment[7].

Before initiating steroid therapy, patients should be well informed about the possible side effects of steroids. Otherwise, it may lead to severe systemic side effects including Cushing’s syndrome, hypertension, dyslipidemia, suppression of hypothalamic-pituitary-adrenal axis, striae, glaucoma, skin

atrophy, cataract and predisposition to life-threatening infections[8].

CONCLUSION

The most common cause of iatrogenic Cushing's syndrome is prolonged corticosteroid use. Many clinicians prescribe these drugs, and they are used to treat a wide range of diseases. As a result, when patients are prescribed corticosteroids, all clinicians must educate them about the potential side effects, as well as the importance of close monitoring and dose tapering. Patients diagnosed with Cushing's syndrome require long-term follow-up until the condition is resolved. Finally, clinicians, nurses, and pharmacists should work together to prevent unsupervised and inappropriate glucocorticoid use. Patients should be educated on the risk of adrenal crisis when abruptly discontinuing corticosteroids. Effective management of Cushing's syndrome requires collaboration among multiple professionals. If the diagnosis is missed, there is a significant risk of morbidity.

Declaration by Authors

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