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CASE REPORTS

Steroid Induced Cushing's Syndrome in an Asthmatic patient: a Case Report

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Abstract

Glucocorticoid therapies are used widely in arrange of medical conditions including respiratory, allergic, inflammatory and autoimmune diseases. All forms of glucocorticoid delivery, even at therapeutic doses, have the potential to cause Cushing's syndrome. Here, we present a case of exogenous Cushing's syndrome resulting from recurrent use of intermittent high dose oral prednisone to treat asthma exacerbation.

Keywords: Cushing's syndrome, glucocorticoids, asthma, hypercortisolism.

INTRODUCTION

In 1932, an American Neurosurgeon Dr. Harvey Cushing's described a syndrome characterized by truncal obesity, hypertension, fatigability and weakness, amenorrhea, hirsutism, purplish abdominal striae, edema, glucosuria, osteoporosis, and a basophilic tumor of the pituitary. It is caused by prolonged exposure to high levels of circulating cortisol; thus, it can be interchangeably called hypercortisolism. Glucocorticoids are important pharmacological agents that reduce inflammation and work with the immune system to treat wide range of medical conditions.

However inappropriate use of glucocorticoids can lead to many adverse effects including but not limited to suppression of hypothalamic-pituitary axis and endocrinal disorders like Cushing's syndrome.[1] There are two main etiologies of Cushing's syndrome: exogenous hypercortisolism and endogenous hypercortisolism. Exogenous hypercortisolism, the most common cause of Cushing's syndrome, is mostly iatrogenic and results from the prolonged use of glucocorticoids.[2] Endogenous Cushing's syndrome results from excessive production of cortisol by adrenal glands and can be Adrenocorticotrophic Hormone (ACTH)-dependent and ACTH-independent. ACTH-secreting

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pituitary adenomas (Cushing's disease) and ectopic ACTH secretion by neoplasms are responsible for ACTH-dependent Cushing's syndrome; while adrenal hyperplasia, adenoma, and carcinoma are major causes of ACTH-independent Cushing's syndrome.[3] The knowledge of pharmacokinetic properties, daily dosage, frequency and differences in individual steroid metabolism is crucial in preventing adverse effects related to excessive use of glucocorticoids. Pharmacovigilance of steroid is necessary which is concerned with identifying the hazards associated with pharmaceutical products and minimizing the risk of any harm that may come to patients.[4] ACTH-dependent cortisol excess due to a pituitary adenoma is called Cushing's disease, and is responsible for 80% of endogenous Cushing's syndrome.[5]

CASE REPORT

A 34-year-old obese male, chronic smoker with a history of asthma, diagnosed two years ago, presented to the emergency department with intermittent breathlessness for the last two days. Other symptoms included chest tightness, dry cough, puffiness of face, intermittent headaches and generalized weakness. There was no associated fever or chills. Heals gave a history of recent weight gain of about eight kilograms in the last three months.

The patient's history suggested that he was on irregular treatment with multiple local healthcare facilities from the last five to six months for breathlessness. Some outside medical records that he showed contained prescriptions of prednisone for different duration each time he was attended for acute asthma. On most visits he was given 60mg prednisone and on two occasions he was given 80 mg daily doses. He did not follow up with the same treating facility.

During this episode of presentation to our emergency department, his vital signs were: Heart Rate - 120 beats per minute, respiratory rate - 24/min, afebrile, blood pressure - 136/94 mmHg and oxygen saturation approximately - 93–95% on room air. On examination, the patient was an obese male, in mild-moderate respiratory distress with bilateral expiratory wheeze, thin extremities, central obesity, facial plethora, moon facies, facial acne (Fig. 1), buffalo hump (Fig. 2), abdominal striae (Fig. 3), thinned out and fungal infected skin (Fig. 4) and swollen digits in the upper and lower extremities. No other significant findings were noted on physical examination.

Figure 1. Thin Extremities, Central Obesity, Facial Plethora, Moon Facies, Facial Acne

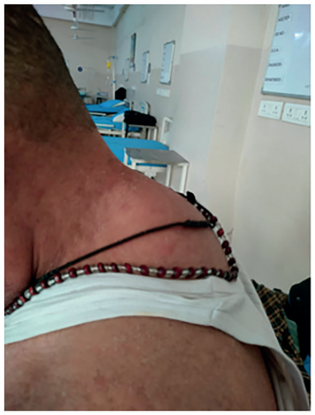

Figure 2. "Buffalo Hump"

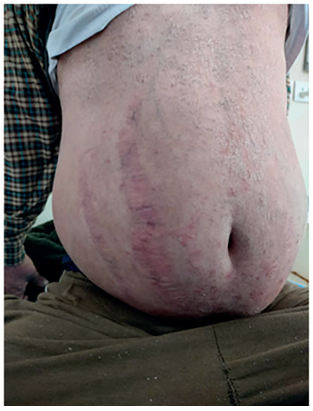

Figure 3. Pendulous abdomen with wide violaceous striae

Figure 4. Thinned out skin, Fungal Infection over skin

His initial work up in the emergency room included:

- Electrocardiogram was unremarkable,
- Complete blood count showed a Total Leucocyte Count (TLC) of 10300/mm^3,
- Random blood sugar level of 272.5 mg/dL (suggesting Diabetes Mellitus),
- Ultrasound whole abdomen showing hepatomegaly grade 2 fatty liver,
- and chest x-ray showing prominent broncho-vascular markings.

He was admitted to hospital for acute bronchial asthma and treated with levosalbutamol – ipratropium bromide nebulization thrice daily and 100 mg of intravenous hydrocortisone. Further investigations revealed: Deranged Lipid Profile showing hypercholesterolemia (314 mg/dL), hypertriglyceridemia (506.4 mg/dL); Gamma-glutamyl transferase – 44.3 IU/L, Serum Alkaline-phosphatase – 159 IU/L, Skin scrapings KOH mount exam showed thin, hyaline, septate & branched hyphae, suggestive of dermatophytosis.

On the second day of admission his chest findings improved; bilateral rhonchi decreased and the patient was less dyspneic. Vital signs were- heart rate: 114 beats per minute, respiratory rate: 20/min, blood pressure: 140/98 mmHg and oxygen saturation: 97% on room air. Since his clinical features were suggestive of steroid induced iatrogenic Cushing's syndrome; therefore, dexamethasone suppression test was advised. Interestingly, laboratory evaluation revealed decreased serum cortisol level (<1.0 microgram/dL) suggesting suppression of endogenous hormones by excessive exogenous steroids. Eventually, he was treated with inhaled bronchodilators, inhaled corticosteroids, oral hypoglycemics, diuretics, expectorants and other supportive medications. The dose of oral steroids was appropriately tapered and eventually stopped. He was discharged in stable condition with advice to follow up in the out-patient department.

DISCUSSION

Patients with Cushing's syndrome usually present with a history of weight gain, fatigue, weakness, delayed wound healing, easy bruising, back pain, bone pain, loss of height, depression, mood swings, emotional reactivity, loss of libido, erectile dysfunction, irregular menstrual cycles, infertility, hyperhidrosis, hirsutism, biparietal visual loss (if there is a large pituitary adenoma), recurrent fungal & bacterial infections due to impaired immunity.[5] Psychological problems such as cognitive dysfunction and depression are not uncommon. Some patients may develop severe osteopenia and bone fractures. Patients may also have a history of hypertension, peptic ulcer disease, and diabetes.[6] Physical examination of the patients also reveal increased fat deposits in the upper half of the body leading to "Buffalo torso", characteristic moon facies (earlobes are not visible when viewed from the anterior aspect of the face), thin arms and legs, acne, hirsutism, proximal muscle weakness of the shoulder & hip girdle muscles, paper-thin skin, abdominal pain due to gut perforation in rare cases, and wide vertical purplish abdominal striae.[7] Physical examination of the patient reveals increased fat deposits in the upper half of the body leading to "Buffalo torso", characteristic moon facies (earlobes are not visible when viewed from the anterior aspect of the face), thin arms and legs, acne, hirsutism, proximal muscle weakness of the shoulder & hip girdle muscles, paper-thin skin, abdominal pain due to gut perforation in rare cases, and wide vertical purplish abdominal striae.[8]

Many of the findings described above were found in our patient. Moreover, his history of repeated steroid intake in view of asthma exacerbations correlated with his physical features of Cushing's syndrome.

CONCLUSION

The most common cause of iatrogenic Cushing's syndrome is the prolonged use of corticosteroids. These drugs are prescribed by many clinicians they have a widespread use in all spectrums of diseases. Therefore, when patients are prescribed corticosteroids, all clinicians must educate them about the side effects and the need for close follow-up and dose tapering.

Patients diagnosed with Cushing's syndrome need long-term follow-up until the syndrome has been cured. Finally, clinicians, nurses and pharmacists should all work together in stopping unsupervised and inappropriate use of glucocorticoids. They must also educate patients about the potential adrenal crisis if the corticosteroid is stopped abruptly. Hence, it is important to manage Cushing's syndrome with an inter-professional team. For, if the diagnosis is missed, there is significant risk of morbidities and mortality.

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