

Case Report



Case Report on Dexamethasone induced Iatrogenic Cushing Syndrome

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ABSTRACT

Introduction: Corticosteroids are the steroid hormones, which are mainly used in the treatment of rheumatoid arthritis, osteoarthritis, rheumatic fever, gout, allergic reactions, renal disease, hematological disorders and shock. The use of glucocorticoids in supra physiological doses for more than 2-3 weeks causes a number of undesirable effects. Most of the adverse effects are extension of pharmacological actions such as hyperglycemia, Cushing syndrome, edema, hypertension, CCF, steroid myopathy, glaucoma, various fungal infections etc. Cushing syndrome can be iatrogenic or endogenous cortisol secretion, due to the either an adrenal tumor or hyper secretion of adrenocorticotropic hormone by the pituitary gland. Cushing's syndrome was discovered by Harvey Cushing in 1912. It is characterized by high levels of cortisol in blood or glucocorticoid substance. **Case Presentation:** Here we report a 55 year old female patient with moon face, weight gain, elevated blood glucose levels and increased blood pressure due to prolonged use of dexamethasone since 2 years regularly. **Conclusion:** The patient condition was improved after withdrawal of dexamethasone. Health awareness should be created among general public to avoid the unnecessary use of over the counter medications.

Keywords: Corticosteroids, Dexamethasone, Cushing syndrome.

INTRODUCTION

Cushing's syndrome was discovered by the American neurosurgeon Harvey Cushing in 1912.¹ Cushing syndrome, a systemic disorder, is the result of prolonged administration of glucocorticoids in large amounts.² It can occur due to endogenous over production of cortisol or exogenous corticosteroid administration and some other causes are obesity, depression, alcohol and food.³

Thyroid is an endocrine gland, located at just inferior to the larynx (voice box). It secretes hormones T₃(tri-iodothyronine) and T₄(thyroxine). It mainly involved in the maintenance of normal basal metabolic rate in the body. Decreased production of thyroid hormone by thyroid gland results in hypothyroidism. Clinical features of hypothyroidism are hypertension, constipation, hypothermia, bradycardia, dry skin, peripheral neuropathy, goiter, loss of hair in the outer 1/3rd of eyebrow, hoarse voice, macroglossia, xanthelasma, peripheral cyanosis, pseudo- myotonia, physical sluggishness, pericardial effusion, cerebral ataxia, pallor, small volume pulse.

Hypothyroidism is diagnosed by measuring the levels of thyroid –stimulating hormone and thyroxine levels in blood.⁴

In this case report we describe a patient with iatrogenic type of Cushing syndrome due to chronic use of dexamethasone.

CASE HISTORY

A 55 year old female patient was admitted in the General Medicine Department of Rajeev Gandhi Institute of Medical science, Kadapa with the chief complaints of facial puffiness, breathlessness, abdominal distension, anuria and constipation since 10 days. Patient reported that she had rapid weight gain. She is a known case of hypothyroidism; type -2 diabetes mellitus since 4 years and on regular treatment with thyroxine 50mcg once daily, metformin 500mg and glibenclamide 5mg daily.

During her past medication history interview she reported a daily intake of tablets (OTC) Dexamethasone (Dexa 4mg) OD and chlorzoxazone 500mg, Diclofenac sodium 50mg and paracetamol 325 mg OD (Diclotal MR) since 2 years for body pains.

On general examination the patient was conscious and coherent and her vitals were as follows BP-160/100 mm of Hg, PR-84bpm, CVS-S1,S2+ ,RS-Wheezing+, CNS- no abnormality present, P/A- distension+.

Investigations

Her laboratory investigations were as follows

Hb-10 gm%, TC-9000cells/mm³, Differential count-polymorphs-54%, Lymphocytes- 40%, Eosinophils-2%. Random Blood Sugar-176mgs/dl, Liver Function test: serum creatinine-0.5mgs/dl, Total Bilirubin-0.4mgs/dl, Direct Bilirubin-0.1 mg/dl, Indirect Bilirubin-0.3mg/dl, alkaline phosphatase-87IU/L, SGOT-58U/L, SGPT-53U/L, Total Proteins-6.4g/dl. TSH-5.42micro IU/ml,T₃-139.88



ng/dl, T₄-11.9 micro g/dl. Electrolytes: potassium-3.0 mEq/L. USG Abdomen-Hepatomegaly with fatty changes.



Figure 1: Patient with Cushing Syndrome

Chest x-ray-Mild cardiomegaly.

So based on subjective and objective evaluation patient have experienced Cushing's syndrome due to the prolonged usage of corticosteroids. Clinical evaluation was done and patient was treated symptomatically with (Tab. Telmisartan 40 mg OD, Tab. Metoprolol 50 mg OD, Tab Paracetamol 500 mg TD, Syp. Lactulose 20 ml, Tab. Pantoprazole 40 mg OD, Inj. Ceftriaxone 1 gm BD, Tab. Amlodipine 5 mg OD, thyroxine 50mcg daily, metformin 500mg and glibenclamide 5mg daily OD).

DISCUSSION

Cushing's syndrome can be iatrogenic or the result of endogenous cortisol secretion, due either to an adrenal tumor or to hyper secretion of corticotrophin (adrenocorticotrophic hormone) by the pituitary (Cushing disease) or by a tumor. It is characterized by moon face, buffalo hump, pendulous (hanging) abdomen, Facial skin is flushed, and poor wound healing. The elevated level of cortisol causes hyperglycemia, osteoporosis, weakness, hypertension, increased susceptibility to infection, decreased resistance to stress, and mood swings.

The elevated level of cortisol causes hyperglycemia, osteoporosis, weakness, hypertension, increased susceptibility to infection, decreased resistance to stress, and mood swings.⁵⁻⁷ In our case, patient had developed the Cushing syndrome after 2 years intake of dexamethasone as over the counter medication; a similar case was reported by Gibin George et.al describes glucocorticoid induced cushing syndrome due to chronic use of dexamethasone.⁸

The patient condition was improved after withdrawal of dexamethasone by the physician.

Patient symptoms facial puffiness, breathlessness, constipation reduced. Blood pressure came to normal (130/80 mm Hg) and potassium normalized (4.4 meq/lit).

CONCLUSION

Iatrogenic Cushing's Syndrome developed in the patient following long term use of the dexamethasone tablets. The patient condition was improved after withdrawal of dexamethasone. Tapering of dexamethasone dose is suggestive to prevent adrenal crisis. Health awareness should be created among general public to avoid the unnecessary use of over the counter medications.

Abbreviation Used

BP: Blood Pressure; CNS: Central Nervous System; CVS: Cardio Vascular System; P/A: Per abdomen; T₃: tri-iodothyronine; T₄: thyroxine.

REFERENCES

1. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, Harrison principles of internal medicine.16th ed, New York, McGraw Hill, 2005, 2134-2135.
2. V C Medvei, The history of Cushing's disease: a controversial tale. *Journal of the Royal Society of Medicine*, 1991 June, 84, 363-366.
3. A J Razenberg, J W F Elte, A P Rietveld, H C T van Zaanen and M Castro Cabezas, A 'smart' type of Cushing's syndrome. *European Journal of Endocrinology*, 2007, 157, 779-781.
4. R Alangappan, Manual practice of medicine.4th ed, Jaypee brother medical publishers, 2011, 621-622.
5. Finkel, Richard, Clark, Michelle A, Cubeddu, Luigi X, Lippincott's Illustrated Reviews: Pharmacology, 4th ed, Lippincott Williams & Wilkins; 2009, 312-316.
6. Chiang MY-M, Sarkar M, Koppens JM, Milles J, Shah P, Exogenous Cushing's syndrome and topical ocular steroids. *Eye*, 2005, 20, 725-727.
7. Dipiro TL, Talbert RL, Yee GC, Matzke GR, Wells BG, Pharmacotherapy A Pathophysiologic Approach.7th ed, New York. McGraw Hill, 2008, 1267-1268.
8. Gibin George, Nikky Jain Thomas, Satish Kumar bp. A case report on glucocorticoid induced Cushing's syndrome. *World Journal of Pharmaceutical Research*, 2015, 4(8), 1503-1506.

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