



Dexamethasone Induced Cushing Syndrome

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Abstract

Corticosteroid therapies have been utilized to treat plethora of medical conditions such as allergic, inflammatory, autoimmune and respiratory diseases. Long-term exposure to steroids can cause a wide range of serious adverse effects such as hyperglycemia, cushing syndrome, osteoporosis, weight gain, hypertension, immune suppression. Here we are reporting a case of a 38 year old female patient who is suffering from Exogenous Cushing syndrome characterized by moon face, central obesity, edema in right limbs and bullous eruption on right hand with long term use of steroids.

Keywords

Cushing syndrome, steroids, cortisol, ACTH, HPA suppression

Introduction

Cushing syndrome is a disease that happens

while your body makes an excessive amount of the hormone cortisol over an extended duration of time. Cortisol is every so often referred to as the strain hormone as it facilitates the body to respond to strain. Cortisol additionally facilitates to keep blood pressure, adjust blood glucose, lessen infection and flip the meals we devour into energy. Cushing syndrome most usually influences adults generally 30-50, however also can arise in children. Cushing syndrome influences approximately 3 instances as many ladies as men. Signs and symptoms of Cushing syndrome consist of weight gain, skinny fingers and legs, spherical face, multiplied fats across the base of neck, a fatty hump among the shoulders, smooth bruising, huge red stretch marks & vulnerable muscle. The fundamental hormonal defect in Cushing syndrome is the overproduction and secretion of cortisol. The overproduction of cortisol results from

pathological processes that relate to the adrenal cortex.

Glucocorticoids are utilized in each endocrine and non-endocrine disorders. Pharmacologic doses of glucocorticoid are used to deal with infection, hypersensitivity and immunological disease. But this has many damaging results starting from HPA suppression and Cushing syndrome to infections and extrade in intellectual status. Both endogenous and exogenous glucocorticoid exerts the bad comments managed at the HPA with the aid of suppressing hypothalamic CRH manufacturing and pituitary corticotropin secretion. This results in adrenal atrophy and lack of cortisol secretory capability. The improvement of Cushing syndrome relies upon the dose, timing and period of glucocorticoid administration. In this we give a case record of Dexamethasone caused Cushing syndrome.

Case Report

A 38 year old female patient presented to the general medicine department with complaints of moon face, central obesity, edema in right limbs and bullous eruption on right hand (index and middle finger) and also had complaints of right shoulder pain. She is allergic to the drug Eptoin. She is a recognised case of CVA- proper hemiparesis, later recognized as AVM ruptured underwent burr holing +EVD shunt and became in ICU for about 30 days and had health center obtained bacterial meningitis dealt with with meropenem, hypovitaminosis D and dyslipidemia. Her past medical history shows right upper limb weakness and hence MRI taken shows hydrocephalus (on steroids from 2021). Her medication history shows she was on T. Frisium 10mg and T. Dexa 4mg 1-1-1-1 for 1 week and thereafter tapering the dose, T. Atorva 10 mg HS, T. Cipcal OD, T. Vitamin D3 Once a week, T. Zincovit, T. Pantop 40 mg OD.

On general examination the patient was conscious and oriented. Her vitals were, Blood Pressure : 110/70mmHg, Pulse Rate: 104beats/min, Respiratory Rate: 16/min, Per Abdomen: distended, nontender, Central Nervous System: Right sided weakness S1 (+). Her laboratory research have been as follows:

Total leukocyte count: 10400/ μ l, differential count: polymorph; 54%, lymphocyte: 43% monocyte: 3%, Hemoglobin: 11.5g/dl, RBC: 3.49 million, PCV: 39.4%. Liver function test: direct bilirubin: 0.06mg/dl, indirect bilirubin: 0.29mg/dl, total bilirubin: 0.35mg/dl, total protein: 4.91g/dl [low], albumin: 3.23g/dl [low], globulin; 1.7g/dl [low], SGOT; 20U/L , SGPT: 46U/L [high] , alkaline phosphatase; 43U/L, calcium: 8.28mg/dl [low]. Serum cortisol: >600 ng/l [high], TSH: 0.89 μ IU/ml, T3: 72ng/l, T4: 4.52ng/l [low], vitamin D: 16ng/l [low], Low Density Lipoprotein: 161[high], triglyceride: 222[high]. Her serum potassium level: 3.2mmol/l. Her laboratory research suggests that she has hypokalemia and multiplied cortisol levels. So based on subjective and objective assessment the person became affected by Cushing syndrome because of extended use of corticosteroids.

Discussion

Cushing syndrome (Hypercortisolism) is an endocrinological disorder resulting from high glucocorticoid levels in the blood (1). Patients with Cushing syndrome usually present with one or more signs and symptoms secondary to the presence of excess cortisol or ACTH (2). It is characterized by moonface (Figure.1.1), buffalo hump, pendulous abdomen, facial puffiness, growth retardation, central obesity, poor wound healing which is accompanied by muscle pain and joint pain(2). Systemic administration of glucocorticoids causes HPA axis suppression by reducing ACTH production which reduces cortisol

secretion by the adrenal gland. The development of Cushing Syndrome is due to exogenous (exogenous administration of steroids) and endogenous causes (such as ectopic ACTH production, pituitary tumor) (4). The features of Cushing Syndrome is based on the

duration and dose of steroid administration. Cushing Syndrome caused by steroids is not uncommon (3). The serum cortisol levels are low in exogenous Cushing Syndrome unless the patient is taking a corticosteroid (4).



Figure 1.1

The aftereffects of Cushing syndrome consist of hypertension, diabetes mellitus, osteoporosis, weight gain, cataract, glaucoma, improved danger of stroke, extra clotting of blood, improved or uncommon infection(2). The common laboratory investigation of Cushing syndrome is low ACTH degree, extended fasting blood sugar degree, excessive cortisol degree, decreased bone density, improved blood low density lipoprotein cholesterol degree, reduced serum potassium degree etc.

In this report, routine blood investigation revealed that the patient has hypokalemia, elevated serum cortisol level, vitamin- D deficiency and elevated cholesterol level. In view of elevated cortisol level and cushing syndrome T. Dexamethasone was stopped and INJ. Hydrocortisone was started and tapered off. She was given potassium correction for decreased serum potassium level. Vitamin -D 60k units once a week for

vitamin - D deficiency. T. Atorvastatin 10mg at bed time for elevated cholesterol level. Orthopedic consultation was sought in view of right shoulder pain and they advised MRI shoulder. X-Ray right shoulder reported normal.

Treatment is done by tapering the dose of steroid which may take a year. Sudden discontinuation after chronic intake may result in adrenal crisis.

The prognosis of Cushing syndrome depends on the cause of disease. Many individuals with Cushing syndrome show improvement with treatment. If Cushing syndrome is untreated it may potentially become fatal.

Conclusion

This case report highlights the importance of educating the patients about long term side effects of using steroids. It is important to be alert to the fact that if Cushing syndrome is not treated promptly, it may

lead to more complications. Early detection of this disease will decrease the morbidity and mortality rates.

9.	TSH -	Thyroid Stimulating Hormone
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Abbreviations

1.	HPA SUPPRESSION-	Hypothalamic Pituitary - Adrenal Suppression
2.	ACTH -	Adrenocorticotropic Hormone
3.	CRH -	Corticotropin Releasing Hormone
4.	CVA -	Cerebrovascular Accident
5.	AVM -	Arteriovenous Malformation
6.	EVD SHUNT -	External Ventricular Drain Shunt
7.	RBC -	Red Blood Cell
8.	PCV -	Packed Cell Volume