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A CASE REPORT ON DEXAMETHASONE INDUCED DIABETES

K. Manasa*[1], P. Khamrunisa Begam[1] and Dr. K. Thirumala Naik[2]

[1]Pharm. D Intern, Pharmacy Practice, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh.
[2]Associate Professor, Pharmacy Practice, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh.

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***Corresponding Author**
K. Manasa
Pharm. D Intern, Pharmacy Practice, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh.

ABSTRACT
A steroid is an organic molecule with biological activity that suppresses the immune system in living things. In the pharmaceutical sector, steroids can be classified in various ways. The majority of the time, doctors will advise their patients to take steroids to lessen immune system-induced inflammation. The blood sugar level of the patient may rise as a result of steroids. Long-term steroid users are more likely to acquire diabetes mellitus than shorter-term users. These medications decrease the sensitivity of cells to insulin while increasing glucose synthesis in the liver. Patient blood sugar level increases as a result of the medication's administration. It is a common instance of diabetes mellitus brought on by steroids. Steroid use has negative side effects, such as hyperglycemia, which can worsen underlying diabetes or cause new, "steroid-induced" diabetes, as well as gastritis, glaucoma, and hypertension.

KEYWORDS: Diabetes mellitus, glucocorticoids, hyperglycemia, dexamethasone.

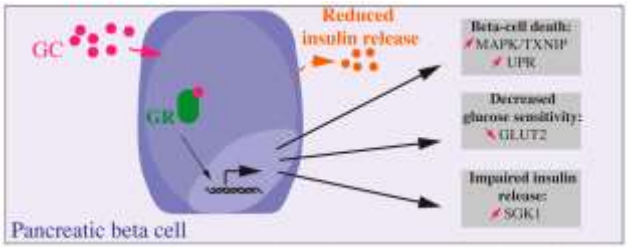
INTRODUCTION
Numerous acute and chronic disorders are often treated with glucocorticoids. It has significant impact on the metabolism of carbohydrates, encouraging the liver to produce glucose from amino acids and glycerol[1]. Steroid use has negative side effects, such as hyperglycemia, which can worsen underlying diabetes or cause new, "steroid-induced" diabetes, as well as gastritis, glaucoma, and hypertension. The wide range of negative effects, which can be categorised into three categories, limits utilisation. categories include idiosyncratic, immediate, and gradual. Retention of fluids is among the immediate effects. visual impairment, mood swings, sleeplessness, weight gain, and reaction modification.[4] Additionally, gradual onset is thought to apply to dyspepsia, skin thinning, and acne. Avascular necrosis, cataracts, chronic glaucoma, and insanity are a some of the unusual outcomes.[4]

In humans, glucocorticoid-induced hyperglycemia has long been documented.[1,2] A long-acting glucocorticoid with greater potency and a longer half-life than prednisolone is dexamethasone.[2]

By boosting hepatic glucose synthesis and reducing muscular glucose uptake, steroids raise blood sugar levels. Additionally, they have a complicated impact on beta cell function.[1]

An 8-hour fasting blood glucose level of less than 7.0 mmol/L (126 mg/dL), a 2-hour post-75-gram oral glucose tolerance test (OGTT) level of less than 11.1 mmol/L (200 mg/dL), a hemoglobin A1c level of less than 6.5%, or, in patients with symptoms of hyperglycemia, a random plasma glucose level of less than 11.1 mmol/L (200 mg/dL).[1]

Dexamethasone has been demonstrated to raise blood sugar levels even after delivery of a single dose. Blood glucose levels significantly rose over time and peaked 120 minutes after 10 mg of dexamethasone. The impact of glucocorticoids on glucose metabolism most likely results from the malfunctioning of beta cells among other routes. On the other hand, when given in divided dosages, they result in persistent hyperglycemia.[4]



With a steroid hyperglycemia that lasts for longer than 24 hours and a small reduction with an overnight fast, dexamethasone belongs to the class of long-acting GCs. Steroid effects are often temporary and reversible.[5]

CASE STUDY
A 46 year female patient was admitted in the General Medicine Department of sri Balaji medical college and hospital institute, Renigunta with the chief complaints of

headache, episode of giddiness of fall. with difficulty in holding objects in right arm, and bed sores since 8 months. Patient reported that she had rapid weight gain. She is a known case of brain tumour (glibastoma multiforme); since 8 months and on regular treatment for maintaining condition and also HTN denovo. past history since 1 month for brain tumour.

On general examination the patient was semi-conscious and her vitals were as follows BP-140/100 mm of Hg, PR-114bpm, RR-26/min, CNS- abnormality present, spo2- 97%, CVS-S1,S2+ ,RS-B/L AE+,

INVESTIGATIONS

Her laboratory investigations were as follows
glycolated random blood sugar -286mg/dl, temp-98f, hb-12.6, serum creatinine-0.8,tlc- 11200 cells/, plc-3.8l/mm^3,bun-26, rr-26bpm, plt-95, wbc-9.99, rbc-8.90, neutrophils%-89.8, lymphocytes%-8.6,monocytes-1.4,hct-32, serum electrolytes like sodium-133.7, potassium-3.46

So based on subjective and objective evaluation patient have experienced diabetes due to the prolonged usage of corticosteroids. Clinical evaluation was done and patient was treated symptomatically with INJ.MONOCEF 1GM IV BD, INJ.MANNITOL 100ML IV OD, INJ.LEVIPIL 750MG IV BD, INJ.PANTOP 40MG IV OD, INJ. DEXONA 4MG IV BD, INJ. TRAMADOL 1 AMP IV IN 100ML NS SOS, MM3.

INJ EMESET 4MG IV BD, INJ.MVI IN 100ML NS OD, TAB. DIAMOX 250MG P/O BD, TAB.REJUNEX- CO3 P/O OD, TAB. FLUCAN 150MG BD, TAB.AMLODIPINE 10MG P/O OD, INJ.H-ACTRAPID 8UNITS, TAB.ZOLPIDEM 5MG P/O OD, TAB.MAHACEF – CV 200/125 BD, TAB. DOLO 650 MG SOS, SYP. POTKLOR 15ML TID.

DISCUSSION

An abnormal rise in blood sugar triggered by the use of glucocorticoids in a patient with or without a history of diabetes mellitus is referred to as SIDM. Drugs called steroids have been used widely for a number of ailments. However widely Glucocorticoids are recommended for their immune-suppressing and anti-inflammatory effects. Hyperglycemia being one of the most common adverse effects common and exemplary.[1]

Increasing LDL cholesterol, endothelial dysfunction, activation of the coagulation cascade, increased production of pro-inflammatory cytokines, and oxidative stress that lead to the progression of macrovascular disease have all been linked to increased cardiovascular mortality.

Transient elevations in blood glucose have been linked in a number of studies to acute inflammatory reactions and endothelial dysfunction in both diabetic and non-diabetic patients.

Before beginning chronic medication, it is important to identify patients who are at risk for developing steroid-induced diabetes. Patients who are considered at risk should have an oral glucose tolerance test (OGTT) as soon as possible.[2,3,1]

Lifestyle changes, like as exercise and dietary counselling, are the first stages in improving glycemic control. These changes may help to reduce post-prandial hyperglycemia.[1]

CONCLUSION

Diabetes is a documented side effect of steroid therapy, whether used alone or in conjunction with other medications. According to certain theories, the dosage, length of administration, and type of steroid have an impact on how diabetogenic glucocorticoids are. Consequently, the doctor must modify the dose in accordance with the patient's pharmacokinetic parameters.

GCs are medications that have been used extensively for a number of medical disorders. Despite their therapeutic value, steroid-induced hyperglycemia continues to be a frequent and possibly dangerous issue that must be taken into account while using any type or dose of GC. Despite its frequent occurrence, little is known about how steroid-related hyperglycemia affects clinical co morbidity and mortality.

Since early detection and efficient treatment of these patients' conditions will be made possible by a thorough understanding of the mechanisms underlying steroid hyperglycemia, this understanding is essential. To avoid all the difficulties linked to the hyperglycemic state, appropriate guidelines that specify the recommendations for the diagnosis and treatment of steroid diabetes are required.

In most cases, insulin must be the first line of treatment, particularly when the serum glucose level is greater than 200 mg/dl. However, each patient must be treated individually in order to take oral hypoglycemic medications and lifestyle changes into consideration.

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