



International Journal of Research In Pharmacovigilance and Pharmacotherapeutics



A case report on steroid induced diabetes mellitus

S. Priyanka*¹, S. Sujitha¹, Y. Srujana¹, Robin George², S Sirisha²

¹Pharm D, Department of Pharmacy Practice, Sri Padmavathi Medical College Hospital (SVIMS), Tirupati, Andhra Pradesh, India.

²Department of Pharmacy Practice, Seven Hills College of Pharmacy, Tirupati, Andhra Pradesh, India.

ABSTRACT

A steroid is a biologically active organic compound having immunosuppressive action in living organism. Different classification of steroids is available in pharmaceutical industry. Steroids are mostly suggested by clinician to the patients for the reduction of inflammation caused by immune system. Steroid can cause elevation of patient blood sugar level. Those who take steroids for a longer period of time are more susceptible to develop diabetes mellitus. The most commonly used steroids are prednisone and cortisone. These drugs promote glucose production in liver and reduce the sensitivity of cells to insulin. In this case report a 45 year old women suffering from nephrotic syndrome was advised to take Inj. Methyl prednisolone. The administration of this drug leads to a rise in her blood sugar level. It is typical case of steroid induced diabetes mellitus.

Keywords: Diabetes Mellitus; Steroids.

ISSN: Awaiting

Case Reports

Corresponding Author

Name: Dr. S Sirisha

Email: sirishajessy57@gmail.com

Article Info

Received on: 05-09-2019

Revised on: 10-09-2019

Accepted on: 14-09-2019

DOI: <https://doi.org/10.33974/ijrhcp.v2i1.147>



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INTRODUCTION

Glucocorticoids are commonly used to treat a wide variety of both acute and chronic illnesses. It has profound effects on carbohydrate metabolism: stimulating liver to form glucose from aminoacids and glycerol. Adverse effects associated with use of steroids includes hyperglycaemia and can worsen preexisting diabetes or precipitate new "steroid-induced" diabetes, gastritis, glaucoma and hypertension. glucocorticoid-induced hyperglycaemia has long been noted in humans.^[1-5] Steroids elevate blood glucose levels by increasing hepatic glucose production and inhibiting

glucose uptake into muscles. They also have a complex effect on beta cell function.^[6-8] The criteria for diagnosing diabetes by the American Diabetes Association, is an 8 hour fasting blood glucose ≥ 7.0 mmol/L (126 mg/dL), 2 hour post 75g oral glucose tolerance test (OGTT) ≥ 11.1 mmol/L (200 mg/dL), HbA1c $\geq 6.5\%$, or in patients with symptoms of hyperglycemic, a random plasma glucose of ≥ 11.1 mmol/L (200 mg/dL).^[9] The effect of glucocorticoids on glucose metabolism is likely the result of impairment of multiple pathways including beta cell dysfunction. risk factors for steroid-induced diabetes beyond cumulative dose and longer duration of steroid course include traditional risk factors for type 2 diabetes-older age, family history, high BMI and impaired glucose tolerance.^[10]

CASE REPORT

A 45 year old women was admitted to a hospital presented with bilateral lower limb edema from 1 month, and h/o hematuria, frothy urine. She was diagnosed with diabetes mellitus, (blood glucose level was 209mg/dl) who had a history of nephrotic syndrome, hypertension, hypothyroidism. Patient undergone renal biopsy showed IgA nephropathy with 2 fibrocellular crescent patient given 3 doses of IV methylprednisolone followed by 5 doses of IV cyclophosphamide for every one month and along with oral wysolone of 40mg.

On examination the patient was conscious and coherent and other systemic examinations were BP of 134/80 mmHg, pulse rate of 90 beats/min, respira-

tory rate of 22 breaths/min. To treat nephrotic syndrome the patient was treated with IV methylprednisolone of 3 doses along with oral wysolone of 40mg. On adhering to the treatment continuously patient suddenly developed raise in blood glucose levels. Suspected to be having steroid induced diabetes mellitus. We can understand that the increased blood glucose levels was due to prednisolone and the patient was treated with metformin of 500mg and then shifted to glipizide 5mg and later the dose of prednisolone was tapered blood glucose levels stabilized to normal.

Table 1: Lab Reports

Lab parameter	Observed value	Normal value
Hemoglobin	12.2 g/dl	12-16g/dl
RBC	4.7 million/cumm	4.5-5 million/cumm
WBC	13300 cells/cumm	5000-11000 cells/cumm
HbA1c	12.9%	4-6%
FBS	106 mg/dl	70-110mg/dl
RBS	127mg/dl	60-140mg/dl
PPBS	209 mg/dl	160mg/dl
Serum creatinine	1.41mg/dl	0.30-1.30mg/dl
Serum potassium	3.3mmol/L	3.50-5.0mmol/L
Serum sodium	139mmol/L	135-145mmol/L
Serum urea	31 mg/dl	10-40 mg/dl
Serum total protein	7.1g/dl	6-8 g/dl

DISCUSSION

SIDM is defined as an abnormal increase in blood glucose associated with the use of glucocorticoids in a patient with or without a prior history of diabetes mellitus. Steroids are drugs that have been used extensively in a variety of conditions. Although widely prescribed for their anti-inflammatory and immunosuppressive properties, glucocorticoids have several side effects, being hyperglycemia one of the most common and representative.

Fluctuations in serum glucose levels have been associated with increased cardiovascular mortality associated with increased LDL cholesterol, endothelial dysfunction, activation of the coagulation cascade, increased pro-inflammatory cytokine production, and oxidative stress resulting in macrovascular disease progression. Several studies have reported that transient increases in serum glucose are associated with acute inflammatory processes and endothelial dys-

function in both diabetic and non-diabetic patients.^[11] There is a need to detect those at risk for developing steroid-induced diabetes before starting chronic therapy. Oral glucose tolerance testing (OGTT) should be done as early as possible in patients who are deemed to be at risk.

Most patients given glucocorticoids at a dose at least equivalent to 40mg/day for more than two days develop hyperglycemia it is well known that glucocorticoid therapy may provoke new onset type II diabetes mellitus.^[12] Initial steps to improve glycemic control include lifestyle modification which includes exercise and dietary counseling to provide options which can perhaps lessen post-prandial hyperglycemia.

CONCLUSION

Diabetes is a known complication of steroid therapy whether as a single agent or in combination with other drugs. The diabetogenic effect of glucocorticoids is said to be determined by dose, duration of administration and type of steroid. Hence physician has to adjust the dose based upon patients pharmacokinetic parameters. GCs are drugs that have been widely used in a variety of medical conditions. Despite their medical efficacy, steroid induced hyperglycemia remains as a common potentially harmful problem that must be considered when using any type a dose of GC. Despite its frequency, little is known about the impact of hyperglycemia associated with steroid use on clinical co morbidity and mortality. A proper understanding of the mechanisms involved in steroid hyperglycemia is needed, since this will allow early detection and effective treatment in these patients. Appropriate guidelines that establish the recommendations for the diagnosis and treatment of steroid diabetes are needed in order to prevent all de complications associated with the hyperglycemic state. In most cases insulin must be the treatment of choice, especially in cases of serum glucose > 200 mg/dl. Nevertheless an individualized approach must be taken in each patient in order to consider lifestyle modifications and oral hypoglycemic drugs as alternative therapeutic

Cancer describes the disease that result when cellular changes cause uncontrolled growth and division of cells, the term 'neoplastic' a synonym of cancer which means 'new growth'. The meaningful definition of a neoplasm or tumour is 'a mass of tissue formed as a result of abnormal, excessive, uncontrolled, autonomous and purposeless proliferation of cells even after the cessation of the stimulus to growth which caused it^[1]. In the most basic term, cancer refers to calls that grow out of control and invade other tissue. Cells are capable of detect and repair DNA damage, if cells is severely damaged and cannot repair itself, it usually undergoes programmed cell death so called apoptosis, cancer occurs when damaged cells grows, divide and spread abnormally instead of self-destructing as they should.^[2]

In United States, an estimate of 15.5 million people with a history as of Jan 1, 2016, according to the 2018 report from the American cancer society.^[3] There are many causes of cancer and over genetics' most of them are preventable. Cancer is the 2nd leading cause of death in United States.^[4]

There are many causes of cancer and some of them are preventable smoking, heavy alcoholism, obesity, physical activity, poor nutrition etc. Genetics influence the cell production of protein, and proteins that carry many of the instruction for cellular growth and division. Cancer is more curable when detected early, although some cancers develop completely without symptoms, or the symptoms are non-specific. Cancer treatment advances every year and early detection has made many cancers treatable. Cancer symptoms and signs are also depend on the size and location of the cancer as well as the presence or absence of metastasis.

REFERENCE

1. Hesketh PJ (2008) Chemotherapy-induced nausea and vomiting. *N Engl J Med* 358:2482–2494
2. Berger MJ, Ettinger DS, Aston J, Barbour S, Bergsbaken J, Bierman PJ, Brandt D, Dolan DE, Ellis G, Kim EJ et al (2017) NCCN Guidelines Insights: Antiemesis, Version 2.2017. *J Natl Compr Canc Netw* 15:883–893
3. Albany C, Brames MJ, Fausel C, Johnson CS, Picus J, Einhorn LH (2012) Randomized, double-blind, placebo-controlled, phase III cross-over study evaluating the oral neurokinin-1 antagonist aprepitant in combination with a 5HT3 receptor antagonist and dexamethasone in patients with germ cell tumors receiving 5-day cisplatin combination chemotherapy regimens: a hoosier oncology group study. *J Clin Oncol* 30:3998–4003
4. Basch E, Hesketh PJ, Kris MG, et al. (2011). Antiemetics: American society of clinical oncology clinical practice guideline update. *J Oncol Pract*;7:395–8
5. Herrington JD, Jaskiewicz AD, Song J (2008) Randomized, placebo-controlled, pilot study evaluating aprepitant single dose plus palonosetron and dexamethasone for the prevention of acute and delayed chemotherapy-induced nausea and vomiting. *Cancer* 112:2080–2087
6. Griffin AM, Butow PN, Coates AS et al (1996) On the receiving end. V: Patient perceptions of the side effects of cancer chemotherapy in 1993. *Ann Oncol* 7:189–195
7. Schmoll HJ, Apro MS, Poli-Bigelli S, Kim HK, Park K, Jordan K, von Pawel J, Giezek H, Ahmed T, Chan CY (2006) Comparison of an aprepitant regimen with a multiple-day ondansetron regimen, both with dexamethasone, for antiemetic efficacy in high-dose cisplatin treatment. *Ann Oncol* 17(6):1000–1006
8. Hesketh PJ, Grunberg SM, Gralla RJ et al (2003) The oral neurokinin-1 antagonist aprepitant for the prevention of chemotherapy-induced nausea and vomiting: a multinational, randomized, double-blind, placebo-controlled trial in patients receiving high-dose cisplatin—The Aprepitant Protocol 052 Study Group. *J Clin Oncol* 21:4112–4119