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STEROID-INDUCED GLYCEMIC ALTERATIONS IN DIABETIC PATIENTS: A SYSTEMIC LITERATURE REVIEW

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ABSTRACT

Glucocorticoids are commonly prescribed for the management of inflammatory, autoimmune, and respiratory diseases because of their potent anti-inflammatory and immunosuppressive properties. Despite their therapeutic benefits, corticosteroids are associated with significant metabolic adverse effects, particularly steroid-induced hyperglycaemia and glucocorticoid- induced diabetes mellitus. Corticosteroids impair glucose homeostasis by increasing hepatic gluconeogenesis, reducing peripheral glucose uptake, inducing insulin resistance in skeletal muscle and adipose tissue, and suppressing pancreatic β-cell insulin secretion. These effects predominantly cause postprandial hyperglycaemia, especially during the afternoon and evening after morning glucocorticoid administration. This systematic literature review evaluates the glycaemic status of diabetic patients with respiratory infections undergoing corticosteroid therapy. Respiratory conditions such as community-acquired pneumonia, chronic obstructive pulmonary disease, asthma, and interstitial lung disease frequently require glucocorticoid treatment, thereby increasing the risk of hyperglycaemia. Regular monitoring of fasting and postprandial blood glucose levels is essential for early detection and prevention of complications. Risk factors include advanced age, obesity, family history of diabetes, higher steroid doses, and prolonged duration of therapy. Management strategies involve lifestyle modifications, oral antidiabetic agents, and insulin therapy, particularly NPH insulin for intermediate-acting glucocorticoids. Early assessment and proper glycaemic management are important to reduce morbidity, hospital stay, and infection-related complications in diabetic patients receiving corticosteroid therapy.

KEYWORDS: Glucocorticoids – Steroid -Induced Hyperglycaemia -Diabetes Mellitus – Blood Glucose Monitoring.

1.1 INTRODUCTION

Glucocorticoids are used to treat a wide range of conditions, such as inflammatory diseases, immunosuppression after transplantation, antiallergic reactions, and improving the prognosis of shock in critically ill patients.[2] They offer a powerful therapeutic response with few alternatives..It is important to consider glucocorticoids can worsen hyperglycemia in people with diabetes mellitus, reveal undiscovered diabetes mellitus, or hasten the onset of glucocorticoid-induced diabetes mellitus.[1] Glucocorticoids have a number of common metabolic adverse effects, such as diabetes, osteoporosis, and hypertension, while being frequently given for their anti-inflammatory and immunosuppressive qualities.[2] For more than 60 years, glucocorticoid-induced diabetes mellitus (GIDM) has been identified as a side effect of glucocorticoid therapy .In individuals with diabetes mellitus, glucocorticoids

worsen hyperglycemia.[12]

A thorough description of glycaemic variations was made possible by the greater accessibility of CGM. Hyperglycemia after more than three days of daily dexamethasone administration was examined in a number of trials[20] .

1.2 PATHOPHYSIOLOGY OF STEROID INDUCED-HYPERGLYCEMIA

We still don't fully understand the exact mechanisms underlying GC's effects on glucose homeostasis.[6] Corticosteroid directly harm insulin secretion and cause insulin resistance in the liver, adipocytes, and skeletal muscle. A familial history of type 2 diabetes is, therefore, a major risk.

After entering the cell nucleus, the receptorglucocorticoid complex dimerizes and attaches its self to glucocorticoid response elements.[4]

The genes that produce inducible nitric oxide synthase, cyclooxygenase-2, and pro- inflammatory cytokines such tumor necrosis factor alpha and other interleukins are inhibited by corticosteroids. corticosteroids initiate upregulation of lipocortin and of annexin A1, a protein that reduces prostaglandin and leukotriene synthesis and that also inhibits cyclooxygenase-2 activity and reduces neutrophil migration to inflammatory sites.[4]

β-cell failure follows severe glucose intolerance brought on by insulin resistance. Subtle increases in blood glucose levels result from healthy β-cells secreting more insulin, which balances the suppressive effect of acute glucocorticoid treatment on β-cells within a few hours.[2]

The liver is essential for preserving glucose homeostasis, which glucocorticoids can upset[2] Glucocorticoids directly increase hepatic glucose production by altering the molecular regulation of gluconeogenic enzyme expression, including phosphoenolpyruvate carboxykinase and glucose-6-phosphatase.[2] Glucocorticoids help move metabolites across the mitochondrial membrane and increase the availability of substrates for hepatic gluconeogenesis.[3] Glucocorticoids directly increase hepatic glucose production by altering the molecular regulation of gluconeogenic enzyme expression, including phosphoenolpyruvate carboxykinase and glucose-6-phosphatase.[2]

Glucocorticoids help move metabolites across the mitochondrial membrane and increase the availability of substrates for hepatic gluconeogenesis.[3] By reducing insulin-mediated inhibition, glucocorticoids promote gluconeogenesis by opposing the metabolic actions of insulin.[2] Numerous phases of the insulin-signaling cascade exhibit the metabolic effects of glucocorticoids on glucose metabolism. Glycogenolysis and gluconeogenesis are stimulated, leading to increased hepatic glucose synthesis. Hyperglycemia is further exacerbated by reduced insulin synthesis and secretion, as well as inhibition of muscle and fat glucose absorption. Instead of exaggerated fasting glucose levels, which peak 8–12 hours after glucocorticoid ingestion, the normal profile is a marked post-prandial glucose spike[13] .which is showed in figure 1.1.

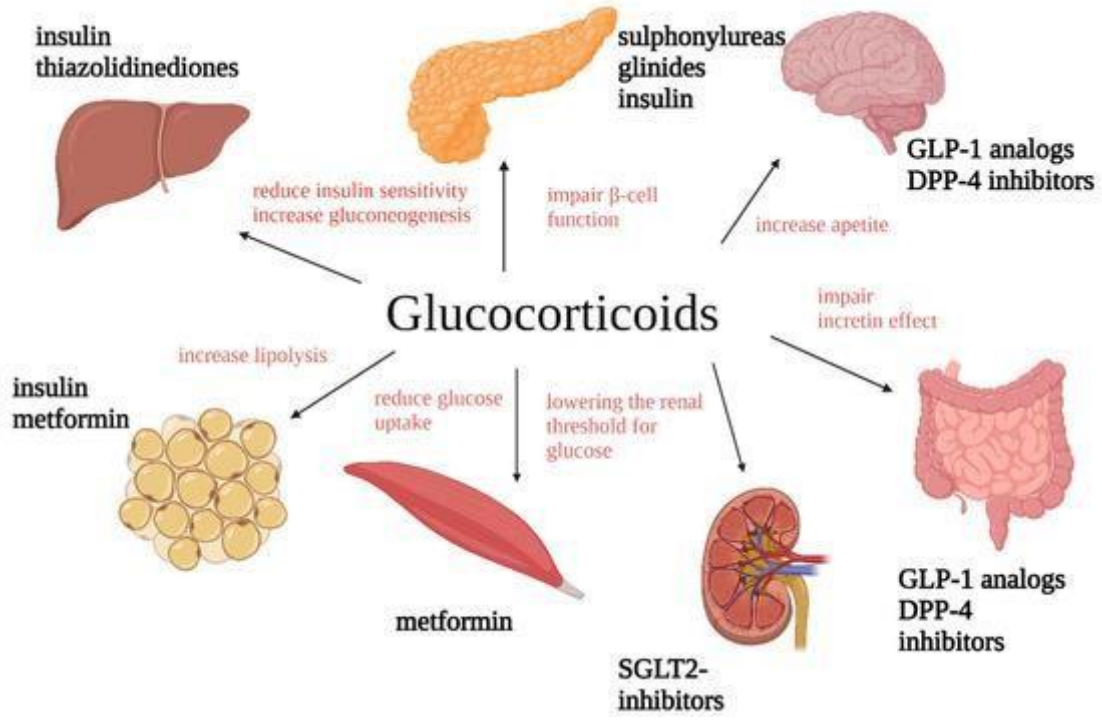

Figure 1.1: Pathophysiology.

1.3 PHARMACODYNAMICS AND PHARMACOKINETICS OF STEROID INDUCED HYPERGLYCEMIA

Adrenal- derived steroids are produced from cholesterol and are secreted in a pulsatile ultradian rhythm and circadian pattern. Normal secretion is between 8 and 15 mg/d, of which 10% circulates freely and the remainder is bound to carrier proteins, namely albumin and cortisol binding globulin. Depending on the type of GC employed, the plasma half-life varies from 80 to 270 minutes, and the effect in tissues lasts for 8 to 12 hours. The liver breaks them down, and the kidneys are primarily responsible for eliminating their conjugated metabolites.[18]

1.4 TYPES OF RESPIRATORY INFECTION STUDIED

- Community-acquired pneumonias (CAPs) are frequent and dangerous acute illnesses that are frequently caused by bacteria.
- Persistent airflow blockage and persistent respiratory symptoms are the hallmarks of the diverse lung disease known as chronic obstructive pulmonary disease (COPD). Because systemic CSs effectively accelerate recovery times and lower the likelihood of early relapse and treatment failure, they are frequently utilized in acute exacerbations of COPD.
- Chronic airway inflammation, fluctuating respiratory symptoms, and restricted expiratory airflow are the hallmarks of asthma, a very common illness.
- A diverse group of lung conditions with diffuse lung fibrosing lesions are known as interstitial lung diseases (ILDs).[15]

1.5 NEED FOR THE STUDY

Patients with type 2 diabetes face significant clinical challenges due to infections, but little is known about the effects of glycemic control.[7] Since hyperglycemia is linked to poor clinical outcomes, including as infection, impairment upon hospital discharge, extended hospital stay, and death, GC-induced hyperglycemia should be managed. Therefore, in order to lessen severity, careful monitoring of glucose levels is advised.[1]

1.6 METHODS AND MATERIALS

Westmead Hospital, a tertiary referral hospital, developed a procedure for routine fingerprick blood glucose (BG) measurement among patients started on high dose steroid medication. The protocol addressed the respiratory, immunology, rheumatology, and oncology units—all of which frequently employ steroid therapy. Prednisone 25 mg/day or more, dexamethasone 4 mg/day or more, and hydrocortisone 100 mg/day or more were considered high dose steroid therapy.[7]

1.7 ASSESSMENT OF GLYCEMIC STATUS

The kind, dosage, and schedule of GC administration all affect the pattern of glycemic excursions that are observed. Patients using corticosteroids are treated for a range of conditions using various corticosteroid formulations in terms of dosage, frequency, duration of action, and timing of administration.[10]

Over 50% of hospitalized patients receiving corticosteroid treatment without a known history of diabetes had hyperglycemia, which is defined as blood glucose >200 mg/dL . The fact that not all glucocorticoid users experience GIDM suggests that GIDM exclusively affects susceptible groups. Numerous indicators of GIDM onset have been found. A family history of diabetes, age, weight, prior glucose intolerance, decreased sensitivity to insulin or impaired insulin production induced by glucose, the amount and duration of glucocorticoid medication.[12]

Although replacement doses result in the least level of hyperglycemia, people on moderate to high doses of prednisolone, such as 7.530 and 30 mg daily, respectively, may experience noti ceable hyperglycemia.

These problems have made it challenging to research corticosteroid-induced hyperglycemia, and the present approach of modifying regular insulin regimens is frequently unsuccessful.[10]

1.8 CORTICOSTEROID PHARMACODYNAMICS IN DIABETES

Among the systemic corticosteroids, prednisone is arguably the most commonly utilized. It is typically utilized as an immunosuppressive and anti-inflammatory drug due to its high GC activity in comparison to mineralocorticoid action. Methylprednisolone may be chosen when mineralocorticoid effects (such as water retention) are especially undesired because it has even less mineralocorticoid action than prednisone and prednisolone.

Compared to prednisone and prednisolone, dexamethasone is far more effective and has a longer duration of action, but it also has very little mineralocorticoid activity. Dexamethasone is typically saved for short-term usage in extremely severe, acute situations because of its great potency, which is linked to severe hypothalamic-pituitary-adrenal (HPA) axis suppression. Additionally, it is not appropriate for alternate-day therapy due to its prolonged duration of action. The least effective GCs are cortisone and hydrocortisone. These medications are typically chosen for usage in individuals with adrenal insufficiency because they have both mineralocorticoid and GC action.[9]

1.9 BASELINE GLYCEMIC STATUS IN DIABETIC PATIENTS [Pre-Steroid]

It is important to take note of the patient's family history, concurrent drugs, and clinical status, including the severity of the illness, changes in the trajectory of

glucose measurements, concurrent medications, and nutritional status, which may have an impact on glucose levels. When a patient is admitted to the hospital, random CBG should be carried out.[1] Whenever possible, the HBA1c test should be carried out at the time of diagnosis.

In addition to providing information regarding the previous glycemic control in individuals w ith recognized diabetes, this would aid in the detection of pre-existing undiagnosed diabete.[1] High-dose steroid therapy is known to cause hyperglycemia in both individuals with and without a diagnosis of diabetes. The use of steroids increases the risk of infection and poor wound healing. Hyperglycemia in steroid-treated patients is a serious issue since it may make these problems worse.[7]

2 GLYCEMIC STATUS AFTER CORTICOSTEROID ADMINISTRATION

GCs are given in the morning, and their metabolic effects peak in the evening. Prednisolone- treated participants exhibit interstitial glucose excursion in the afternoon, according to studies employing continuous glucose monitoring systems. In order to screen for glucocortico id- induced hyperglycemia and diabetes, it is crucial to monitor glycemia between 4 and 5 p.m.[14] High-dose treatment promotes insulin resistance in patients with pre-existing and newly diagnosed diabetes, and exogenous corticosteroid use is linked to hyperglycemia . Within hours of steroid treatment, the effects of GC injection on glucose levels are seen, and they seem to be dose-dependent.

The odds ratios (ORs) for hyperglycemia were 1.77, 3.02, 5.82, and 10.34 for 1–39 mg/day, 40–79 mg/day, 80–119 mg/day, and ≥120 mg/day of hydrocortisone-equivalent, respectively, according to a population-based study of over 11,000 patients. Additionally, postprandial glucose levels seem to be more affected by GCs than fasting glucose levels.

In the absence of GC medication, individuals with pre-existing diabetes or glucose intolerance often have the same glycemic goals and management techniques as those with GC-induced hyperglycemia or diabetes.

It is estimated that between 12.4% and 26% of hospitalized patients have diabetes.[13] Efforts to adjust inpatient diabetes regimens to mitigate the rise in plasma glucose related to corticosteroids have been challenging.[10]

Systemic Glucocorticoid Drug / Dose categpry Low dose GC	**Hyperglycemia Range**	**Duration & onset of Hyperglycemia**	**Glycemic status / patterns**
Hydrocortisone	1-39 mg/day	Glucose elevation may begin within hours after administration; effects more evidence in afternoon/evening	Mild increase in glucose levels; postprandial glucose affected more than fasting glucose
Moderate dose GC	40-80 mg/day	Hyperglycaemia typically appears within hours and may persist through the day depending on steroid therapy	Increased insulin resistance; afternoon /evening glucose excursions common
High dose GC	80-120 mg/day	Rapid onset within hours; prolonged elevation in glucose	Significant postprandial hyperglycaemia ;may unmask latent disease
Very high dose GC	>120 mg/dl	Early onset with sustained hyperglycaemia throughout steroid activity period	Severe insulin resistance; marked hyperglycaemia in patients with and without pre-existing diabetes
Prednisolone	Afternoon interstitial glucose excursions documented	Metabolic effect peaks in the evening; glucose monitoring recommended	Predominantly postprandial hyperglycaemia
Exogenous corticosteroid Therapy [general]	Dose – dependent increase in glucose levels	Effects seen within hours of therapy initiation	Hyperglycaemia more pronounced after meals than in fasting state

2.1 MONITORING GLYCEMIC STATUS AFTER CORTICOSTEROID ADMINISTRATION

Every CBG reading that was acquired was utilized.[11] A glucometer was used to measure CBG four times a day (fasting, prelunch, predinner, and sleep).[19] CBG might have been measured in accordance with the primary team's judgment or the nurse-initiated hypoglycemia protocol, which suggests repeated CBG checks every 15 minutes until CBG recovers to >70 mg/dL, in addition to the four pre-meal and bedtime readings per day. Hospital meal distribution timing was used to categorize CBG readings: pre-breakfast from 5:00 am to 9:30 am, pre-

lunch from 9:31 am to 4:00 pm, pre-dinner from 4:01 pm to 8:00 pm, and nighttime from 8:01 pm to 4:59 am. CBG <40 mg/dL and >400 mg/dL were recorded in our glucose database as <40 mg/dL and >400 mg/dL, respectively, and were represented in our analysis as 39 mg/dL and 401 mg/dL, respectively. We classified hypoglycemia as CBG <70 mg/dL.[11]

2.2 PATTERNS OF GLYCEMIA VARIABILITY POST PRANDIAL BLOOD GLUCOSE LEVEL

Most people believe that the primary effect of glucocorticoids is a rise in postprandial blood glucose levels. The use of a continuous blood glucose monitor in patients with prednisolone- treated chronic obstructive pulmonary disease showed that hyperglycemia mostly happened in the afternoon and evening, suggesting that this would be the best time to screen for GIDM and to start certain treatments.[12]

If any of the test values are high (Random CBG ≥ 180 mg/dL [10 mmol/L], Pre-meal ≥ 140 mg/dL [7.8 mmol/L], Post-meal ≥ 180 mg/dL [10 mmol/L], FPG ≥ 110 mg/dL [6.1 mmol/L],

DRUGS	DOSE	CLASSIFICATION	ONSET OF ACTION	DURATION
Hydrocortisone	20	Short Acting	1 hr	8
prednisolone	5	Intermediate -Acting	1-2 hr	16-36
Methylprednisolone	4	Intermediate Actng	1-12 hr IV;48 mints	18-40
Dexamethazone	0.75	Long-Acting	1-2 hr IV 5-10 nits	36-54

HbA1c ≥6.0%), CBG should be checked for underlying hyperglycemia for at least two days [1]

2.3 RISK FACTORS FOR STEROID INDUCED HYPERGLYCEMIA

As previously noted, patients with diabetes mellitus and carbohydrate intolerance frequently experience worsened and uncontrolled hyperglycemia.[18] The following are the primary risk factors that have been found to be predictive of acquiring diabetes.

Drug type and dosage Treatment duration

Posology (e.g., bolus versus continual treatment)

≥65 years of age Male sex

BMI* > 25 kg/m2

The ethnic group of African Americans eGFR° <40 mL/min/1.73 m2

HbA1c$ ≥6.0%

Gestational diabetes history Diabetes mellitus in the family

Using calcineurin inhibitors and mycophenolate mofetil simultaneously.[17]

2.4 MANAGEMENT STRATAGIES

Depending on the type of steroid used—intermediate-acting or long-acting GCs—insulin resistance develops primarily after meals. Intermediate-acting GCs include prednisone and methylprednisolone, which peak four to six hours after dosing. When given in a single dose, they mostly affect glucose levels in the afternoon and at night and have little effect on fasting glucose.[18]

Current glucose levels for hospitalized, non-critically sick patients are typically accepted in relation to the diagnostic criteria28. From a practical Thus, when the pre-prandial and postprandial blood glucose levels are greater than 140 mg/dL and 200 mg/dL, respectively, therapy for SIDM and SIH should begin.

The proposed control target serves as the basis for both the control objectives and the necessity of treatment in the case of chronic GC treatment. As a result, selecting insulin or oral antidiabetic drugs (OADs) is an important decision that should also take the length of GC therapy with account.

2.5 DIABETES MELLITUS

The morning dosage should be raised for people on insulin secretagogues. Within the JBDS criteria, a Gliclazide dosages should be increased by 40 mg to a maximum of 240 mg30. One option is to temporarily add insulin NPH in the morning or adjust the dosage of metformin.

If the patient is solely receiving basal insulin, think about moving to morning administration and increasing the dosage in 10% increments every 24 to 48 hours based on the BG monitoring results. Every 24 to 48 hours, people on a twice-daily pre-mixed insulin regimen should think about increasing their morning insulin dosage by 10%. An increase in the short-acting insulin dosage between lunch and the evening may be necessary for people on an MDI regimen.

2.6 ORAL AGENTS

The effectiveness of oral medications in treating steroid - induced hyperglycemia is not well understood. Exercise, nutrition therapy, and oral antidiabetic medications should be the main focus of treatment for patients with fasting glucose levels below 200 mg/dL, no history of diabetes, and low-dose GCs. Because metformin directly improves insulin sensitivity, it may be a useful treatment choice. Patients with steroid-induced hyperglycemia were treated over an extended period of time with thiazolidinediones (TZDs). They have minimal impact on insulin secretion but function as ligands for PPAR-γ receptors, increasing insulin action in skeletal muscle and adipose tissue.[18] Selective inhibitors of glucagon-like peptide-1 and dipeptidyl peptidase 4 (DPP-4) have demonstrated efficacy in managing hyperglycemia.[18]

2.7 INSULIN TREATMENT

NPH insulin's peak and duration of effect are comparable to those of traditional intermediate- steroids (prednisone and prednisolone), it is advised for patients who take a single daily steroid dose, usually in the morning.[35] According to Clore et al.[14], a weight and steroid dose- based plan should be used, with an initial dose of 0.4 U/kg of NPH and subsequent modifications based on response.[18] If steroid dose is given throughout a day NPH insulin was not enough to moderate hyperglycemia then the dose was divided into 30% divided Basal insulin.

The best insulin treatment for GIH patients has been the subject of numerous studies. Insulin is thought to be the best treatment for GIH, which is a prevalent problem during hospital stays. The goal of insulin is to keep blood glucose levels at or below 180 mg/dL in order to enhance the clinical results for inpatients.[44] Insulin therapy permits high doses to be given via subcutaneous or intravenous infusion without a maximum dose limit, which is essential for treating extreme hyperglycaemia . It also provides the flexibility to modify dosages in response to the quickly changing patterns of GIH.[2]

2.8 PATIENT EDUCATION\

Reinforces Northwestern Medicine's dedication to providing safe, evidence-based care while educating patients about the dangers of utilizing steroids for common illnesses like colds and influenza.[16]

CONCLUSION

In individuals with diabetes mellitus, glucocorticoid-induced hyperglycemia has important clinical ramifications. Patients undergoing glucocorticoid treatment should benefit from early detection and appropriate proactive management of glucocorticoid-induced hyperglycemia.

Although SIDM and SIH are frequently encountered issues in clinical practice, there is a depth of scientific research and insufficient information in the literature. Information about the clinical implications of GC-induced hyperglycemia and methods for diagnosing and treating it. It is recommended that all patients receiving medium to high doses of GCs undergo screening for SIDM.

The primary warning is to the early and accurate diagnosis of SIDM and/or SIH, which depends on 2-hour postprandial glycemia. Insulin should always be chosen over OADs, especially for individuals who are hospitalized. A weight-based NPH insulin regimen is recommended as the initial treatment for SIDM/SIH in these patients based on the available data. They concluded that screening blood glucose levels in patients receiving steroid therapy and in those with known Diabetes Mellitus was very important. The management of steroid-induced hyperglycemia through insulin was recommended, and postprandial blood glucose levels were screened beforehand to minimize complications.

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