

“Prescription Patterns of Insulin in Steroid-Induced Hyperglycemia Among Type 2 Diabetic Patients with Respiratory Infections: A Systematic Literature Review”

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ABSTRACT

Corticosteroids are widely used in the management of respiratory infections such as Chronic Obstructive Pulmonary Disease, Bronchial Asthma, Pneumonia and Covid19 due to their anti-inflammatory and immunosuppressive effects. Although corticosteroids improve respiratory outcomes, their use frequently leads to Steroid -Induced Hyperglycemia, especially in patients with pre-existing Diabetes Mellitus. Steroid induced hyperglycemia is a common yet often underdiagnosed clinical condition in hospitalized patients. Poor glycemic control during hospitalization may increase the severity of infections, delayed recovery, prolonged hospital stay and increase the risk of other complications. Therefore, respiratory patients receiving corticosteroid therapy require careful blood glucose monitoring and appropriate management. Insulin therapy is considered the preferred treatment for managing steroid induced hyperglycemia in hospitalized patients because of its rapid onset of action, effectiveness and flexibility in dose adjustment. Thus appropriate insulin therapy becomes essential in the management of steroid-induced hyperglycemia. Various insulin regimens such as sliding scale insulin, basal insulin, premixed insulin and basal-bolus therapy are used in clinical practice, and thus the prescribing patterns vary widely. Evaluating these patterns helps optimizing treatment strategies and improve patient outcomes.

KEYWORDS: Steroid induced hyperglycemia, Respiratory infections, Insulin, Diabetes mellitus

I. INTRODUCTION

The term "diabetes mellitus" (DM) describes a collection of common metabolic diseases that are characterized by hyperglycemia. Genetics and environmental factors interact in a complex way to generate various kinds of diabetes mellitus. Reduced insulin secretion, decreased glucose utilization, and increased glucose production are factors that contribute to hyperglycemia.^[1] Insulin is a key anabolic hormone that influences how proteins, fats, and carbs are metabolized. Due to insulin resistance, the metabolic problems linked to diabetes primarily impact tissues like the liver, skeletal muscles, and adipose tissue. The type and duration of diabetes can affect how severe the symptoms are. Increased appetite, polydipsia, dysuria, weight loss, and eyesight issues are among the symptoms that people with high blood sugar levels, especially those with a total lack of insulin, like children, may encounter. Some diabetics, particularly those with type 2 diabetes in their early stages, may not exhibit any symptoms.^[2] The pathogenic process that causes hyperglycemia is used to categorize diabetes mellitus. Type 1 and type 2 DM are the two main types of DM. Autoimmunity against insulin-producing beta cells causes type 1 diabetes, which is characterized by insulin insufficiency. A diverse range of conditions known as type 2 diabetes are typified by varying degrees of insulin resistance, decreased insulin secretion, and elevated hepatic glucose production.^[1]

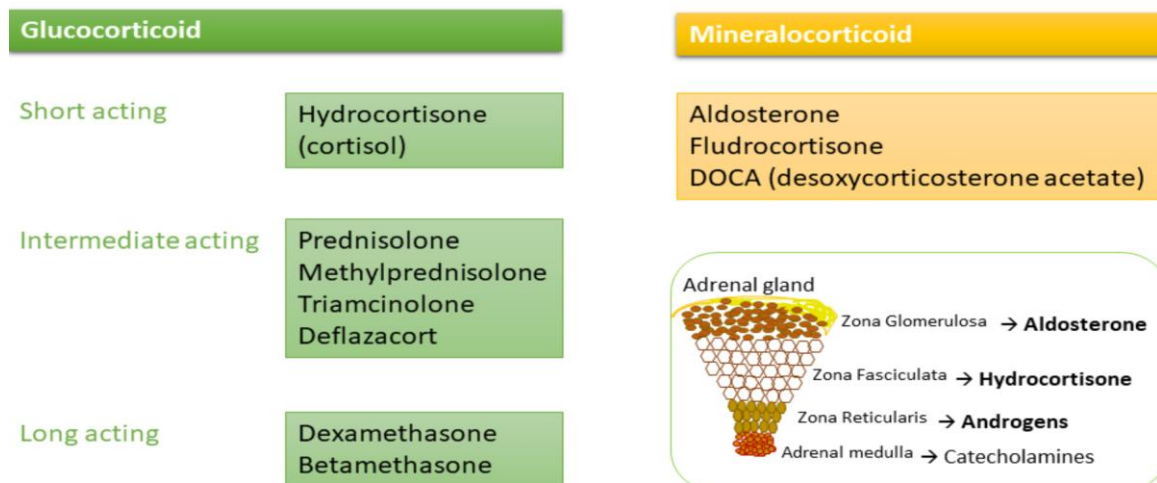
CORTICOSTEROIDS

Corticosteroids are the synthetic derivatives of the natural steroid hormones made by the adrenal cortex and it is classified as glucocorticoids and mineralocorticoids. The synthetic hormones exhibit various degrees of mineralocorticoid and glucocorticoid characteristics. In addition to its immunosuppressive, anti-inflammatory, and vasoconstrictive properties, glucocorticoids are primarily involved in metabolism. While mineralocorticoids influence ion transport in the epithelial cells of the renal tubules to regulate electrolyte and water balance^[3]

For around 50 years, systemic corticosteroids have been used to treat a wide range of illnesses. The least effective products are those

with short half-lives, such hydrocortisone. The intermediate-acting drugs prednisone and methylprednisolone have four to five times the potency of hydrocortisone. The potency of dexamethasone, a long-acting systemic corticosteroid, is roughly 25 times higher than that of short-acting medications. When treating individuals with AIDS who have *Pneumocystis carinii* pneumonia, corticosteroids lower morbidity and the incidence of respiratory failure as well as the requirement for hospitalization in croup patients. Patients with meningitis brought on by *Mycobacterium tuberculosis* or *Haemophilus influenzae* may experience fewer problems if they take corticosteroids.^[4]

Corticosteroid classification



INDICATIONS

Pulmonary: asthma flare-ups, COPD flare-ups, anaphylaxis, urticaria and angioedema, rhinitis, pneumonitis, sarcoidosis and interstitial lung disease.

Dermatology: pemphigus vulgaris, contact dermatitis.

Endocrinology: congenital adrenal hyperplasia, adrenal insufficiency.

Gastroenterology: autoimmune hepatitis, inflammatory bowel disease.

Haematology: idiopathic thrombocytopenic purpura, leukemia, lymphoma and hemolytic anemia.

Rheumatology: polymyositis, dermatomyositis, polymyalgia

rheumatica, systemic lupus erythematosus and rheumatoid arthritis.

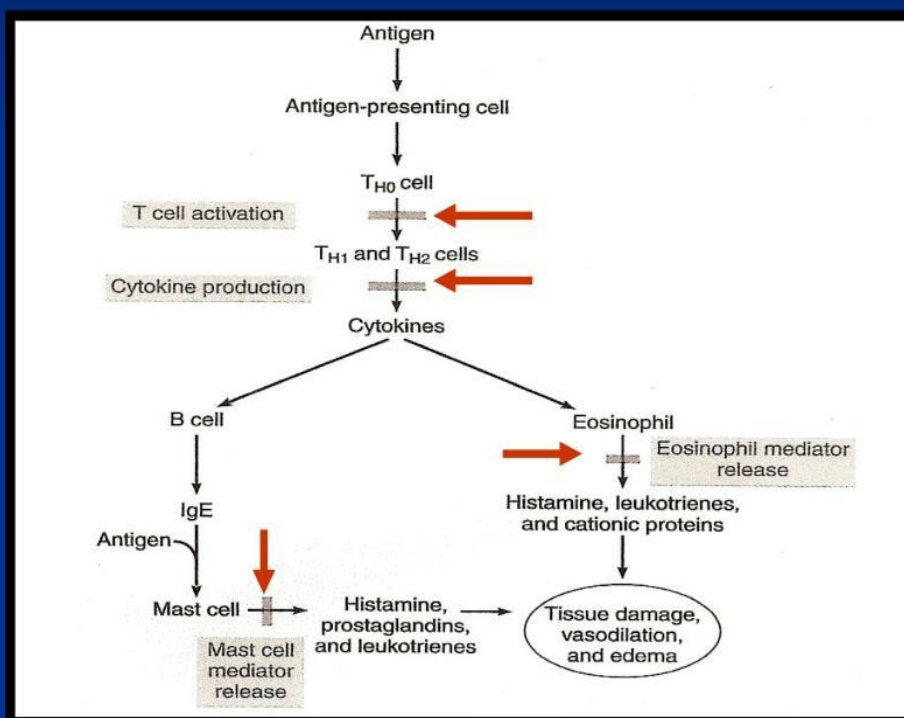
Ophthalmology: keratoconjunctivitis and uveitis
Other conditions include multiple sclerosis, nephrotic syndrome, cerebral edema, organ transplantation and prenatal lung maturation.

MECHANISM OF ACTION

The glucocorticoid receptor is found intracellularly in the cytoplasm. When it binds, it quickly translocates into the nucleus, where it influences gene transcription and inhibits the expression and translation of genes in inflammatory leukocytes and structural cells like epithelium. Proinflammatory cytokines, chemokines,

cell adhesion molecules, and other inflammatory response-related enzymes decrease as a result^[3]

Mechanism of Action of Anti-Inflammatory Steroids



ADVERSE EFFECTS OF CORTICOSTEROIDS

Osteoporosis, fractures and osteonecrosis
 Adrenal suppression
 Diabetes and Hyperglycemia
 Cushingoid features
 Myopathy
 Glaucoma and cataracts^[3]

ROLE OF CORTICOSTEROIDS IN RESPIRATORY INFECTIONS

Since their introduction in the 1950s, glucocorticoids have been essential in the management of a number of inflammatory conditions, including respiratory disorders. Glucocorticoids have been widely utilized to treat idiopathic interstitial pneumonia, chronic obstructive pulmonary disorders, endobronchial tuberculosis, sarcoidosis, hypersensitivity pneumonitis, and other respiratory conditions because they reduce

inflammation and minimize tissue damage^[5] Patients with wheeze and dyspnea are frequently administered systemic glucocorticosteroids (steroids). Since both acute asthma and COPD are becoming more widely acknowledged as inflammatory disorders, research supports the use of steroids to treat their flare-ups. Short courses of systemic steroids have been demonstrated to enhance spirometric and clinical outcomes in patients with COPD exacerbations.

A common wintertime virus that affects people of all ages, respiratory syncytial virus (RSV) is linked to wheezing, particularly in young children. Systemic corticosteroids have been shown to provide no desirable therapeutic benefit in children with RSV-related bronchiolitis and wheeze. Although the virus is rarely specifically recognized in clinical practice, it has been linked to COPD and asthma flare-ups in adults. Systemic corticosteroids are frequently recommended since wheezing is linked to the illness.

However, it has never been investigated how short-term high-dose steroid treatment affects viral load and adaptive immunity to a particular virus during acute infection.^[6]

Nowadays, one of the recognized treatments for SARS-CoV2 (COVID-19) pneumonia is the use of glucocorticoids (GC), which are commonly used for immunosuppressive and anti-inflammatory purposes. Dexamethasone is used for severe infections because the damage the illness causes is closely linked to the hyperactive inflammatory response that is set off.

However, GCs suffer from a number of metabolic side effects, such as osteoporosis, diabetes, and hypertension.^[7]

STEROID INDUCED HYPERGLYCEMIA

Steroid induced hyperglycemia is defined as the abnormally elevated blood glucose associated with the use of Glucocorticoids in patients with or without pre-existing DM. A confirmed fasting blood glucose level of ≥ 7 mmol/L (≥ 126 mg/dL), a glucose level of ≥ 11.1 mmol/L (≥ 200 mg/dL) at 2 hours after ingesting 75 g glucose in an oral glucose tolerance test (OGTT), a HbA1c of $\geq 6.5\%$ (≥ 48 mmol/mol), or a random blood glucose level of ≥ 11.1 mmol/L (≥ 200 mg/dL) defines steroid induced hyperglycemia. However, diagnosing SIHG patients might be more difficult because fasting blood glucose levels may be normal, particularly when short-acting or intermediate-acting GCs are given in single morning doses. In addition to the challenges associated with administering an oGTT to hospitalized patients, hyperglycemia may not occur following glucose exposure in an oGTT, particularly if the test is conducted in the morning when the GCs' diabetogenic impact is still developing. Since HbA1c reflects the glycaemic state in the weeks before the time point of measurement, it may not be noticeable, particularly in patients receiving GC medication for the first time. The accuracy of HbA1c readings is also impacted by a number of illnesses, including hemoglobinopathies and chronic renal disease, which are common in patients who need steroids. However, measuring HbA1c can be helpful in assessing glycaemic control in patients receiving long-term GC therapy or in differentiating between pre-existing DM and new-onset diabetes in cases of hyperglycemia following GC initiation. For individuals receiving high doses of GCs (defined as >20 mg prednisolone or equivalent), regular (capillary) glucose monitoring is advised due to the limitations of the standard diagnostic technique to detect SIHG. This strategy is especially advised for

those who have a high risk of developing SIHG (e.g., advanced age, higher BMI, reduced glucose tolerance in the past, prediabetes, or a family history of diabetes). The diagnosis of SIHG can then be established using a random glucose measurement of ≥ 11.1 mmol/L (≥ 200 mg/dL)^[8]

EPIDEMIOLOGY

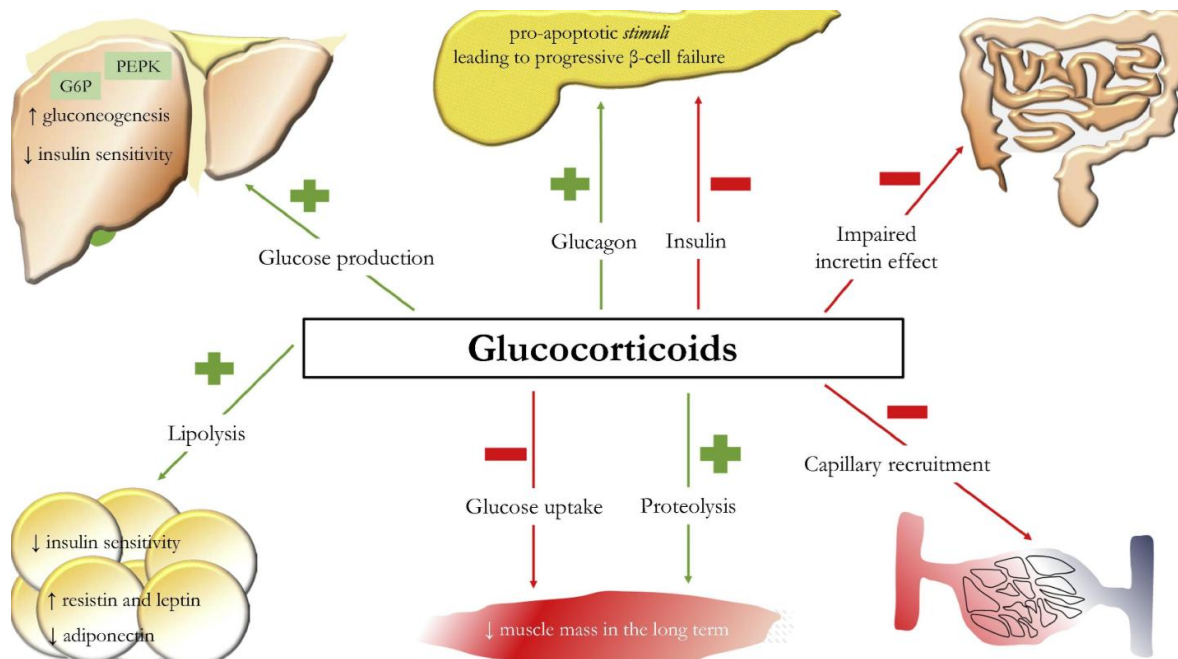
In the hospital context, there is evidence that more than half of the patients receiving high-dose steroids develop hyperglycemia, with a frequency of 86% of at least one episode of hyperglycemia and 48% of patients presenting a mean blood glucose ≥ 140 mg/dL. The main factors that related to inpatient hyperglycemia are previous history of DM, a higher prevalence of comorbidities, prolonged treatment with steroids and older age.^[9] In 2% of cases, oral GCs are linked to diabetes, but topical treatments, inhalers, eye drops, or injections containing GC have a negligible correlation with diabetes. High-dose prednisolone-induced SIDM in hospitals has a quick onset and peaks in the late afternoon, necessitating early blood glucose monitoring.^[7]

RISK FACTORS

Higher doses of steroids (prednisolone >20 mg, hydrocortisone >50 mg, dexamethasone >4 mg) and longer duration of steroid use are risk factors for GC-induced diabetes. Additional risk factors include being older, having a higher BMI, having previously experienced glucose intolerance or impaired glucose tolerance, having a personal history of gestational diabetes or GC-induced hyperglycemia, having a family history of DM, and having a HbA1C of $\geq 6\%$.^[10]

PATHOPHYSIOLOGY

The GLUT-4 transporter-related signaling pathways can be impacted by steroids, which can result in a 30–50% reduction in muscle cell glucose uptake. Increases in blood free fatty acids and amino acids brought on by steroid-induced protein and lipid catabolism may also have an impact on this. Induction of nuclear Peroxisome Proliferator-activated Receptor alpha (PPAR- α) also directly increases IR. Lipid buildup in the muscles, which is encouraged by steroids, also decreases intramuscular glucose entry and storage. Through gluconeogenesis, steroids can amplify the effects of glucagon and adrenaline. Additionally, they reduce GLUT-2 and glucokinase receptor expression, which inhibits insulin production and secretion through lipotoxicity.^[7]



The binding of glucocorticoid receptors in hepatic, adipose, skeletal, and pancreatic tissue mediates glucocorticoid-induced hyperglycemia. Gluconeogenic enzymes are upregulated in the liver by glucocorticoids. To further speed up gluconeogenesis, the liver receives gluconeogenic precursors such fatty acids from lipolysis and amino acids from protein catabolism. Another important mechanism is hepatic insulin resistance. Insulin normally inhibits gluconeogenesis, but when glucocorticoids are present, gluconeogenesis is permitted to proceed. Glucocorticoids reduce the absorption of glucose into skeletal muscle via inhibiting the recruitment of glucose transporter type 4 (GLUT4). Steroid-induced hyperglycemia and/or diabetes are caused by the effects of glucocorticoids on increased central adiposity, increased hepatic steatosis as a result of enhanced lipolysis, and increased appetite stimulation.^[11]

DIAGNOSTIC CRITERIA

When a patient is admitted to the hospital, random CBG should be carried out. The first significant meal in the hospital should then be followed by a pre-meal and two-hour post-meal

CBG. Blood samples should be sent for glycosylated hemoglobin (HbA1c) and fasting plasma glucose (FPG) testing the next morning if lab facilities are available. 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], HbA1c $\geq 6.0\%$), CBG should be checked for underlying hyperglycemia for at least two days. If any of the test results (Random CBG ≥ 250 mg/dL [13.9 mmol/L], Pre-meal ≥ 150 mg/dL [8.3 mmol/L], 2 hours Post-meal ≥ 200 mg/dL [11.1 mmol/L]) satisfy the glycemic criteria, pharmacotherapy should be started. HbA1c test should be performed at the time of diagnosis wherever possible. In addition to providing information regarding the previous glycemic control in individuals with recognized diabetes, this would aid in the detection of pre-existing undiagnosed diabetes. Additionally, it helps with risk assessment and patient counseling. All hospitalized patients with diabetes or hyperglycemia (blood glucose >140 mg/dL [7.8 mmol/L]) should have an A1C test if it hasn't been done in the previous three months, according to ADA recommendations.

INSULIN TYPES ACCORDING TO MODE OF ACTION

Insulin Type	Examples	Onset	Peak	Duration	Clinical use
Rapid-acting	Lispro(Humalog), Aspart(Novolog), Glulisine(Apidra)	10-30 min	30min-3hr	3-5hr	Taken before meals to control post-meal spikes

Short-acting	Regular insulin (Humulin R, Novolin R)	30-60min	2-5hr	5-8hr	Mealtime control
Intermediate-acting	NPH (Humulin N, Novolin N)	1-2hr	4-12hr	12-18hr	Basal coverage
Long acting	Glargine (Lantus, Basaglar), Detemir (Levemir)	1-2hr	Minimal/none	Upto 24hr	Steady background insulin
Ultra-long acting	Degludec (Tresiba)	30-90min	No significant peak	Over 42hr	Extended coverage
Premixed	Humulin 70/30, Novolin 70/30, Humalog Mix 75/25	5-60min	Varies	10-16hr	Short+intermediate insulin for simplified dosing

[12]

INSULIN THERAPY IN STEROID INDUCED HYPERGLYCEMIA

It is feasible to transfer the hyperglycemic effect of several GCs to their pharmacokinetic characteristics. As a result, the insulin treatment selected for SIHG. The glucose-lowering therapy is started when pre- or post-prandial glucose levels consistently surpass 7.8 (140 mg/dL) or 11.1 mmol/L (200 mg/dL), respectively. Stepwise intensification of antihyperglycemic medication and regular reevaluation should be carried out in SIHG, much like the management strategies to lower glucose in patients with type 2 diabetes (T2DM).^[5] Insulin dosage can be changed by half the percentage of the GC dose variation in a practical manner. For instance, it is recommended to increase or decrease the insulin dosage by 25% when GCs are increased or decreased by 50%.^[9]

Short-Acting Glucocorticoids (Hydrocortisone)

If greater subsequent glucose levels or persistent post-prandial hyperglycemia occur, insulin therapy can be escalated by adding insulin corrections, provided that the intensification necessitates pre/post-prandial glucose measurements. In these situations, the planned insulin dose can be increased by stepwise increments of 0.04 IU/kg for pre-prandial levels between 11.1 and 16.7 mmol/L (200 and 300 mg/dL) or 0.08 IU/kg for values greater than 16.7 mmol/L (300 mg/dL). It is crucial to note that insulin requirements are GC dose-dependent; hence, a decrease in GC is typically associated with an improvement in glycemia. Rapid-acting insulin should be reduced in proportion to the reduction in GC dosage; conversely, when GC dosages are advised to be raised, rapid-acting insulin dosage can be increased.

Intermediate-Acting Glucocorticoids (Prednisolone and Methylprednisolone)

The most often prescribed steroids are glucocorticoids with an intermediate half-life. They are helpful for long-term anti-inflammatory and

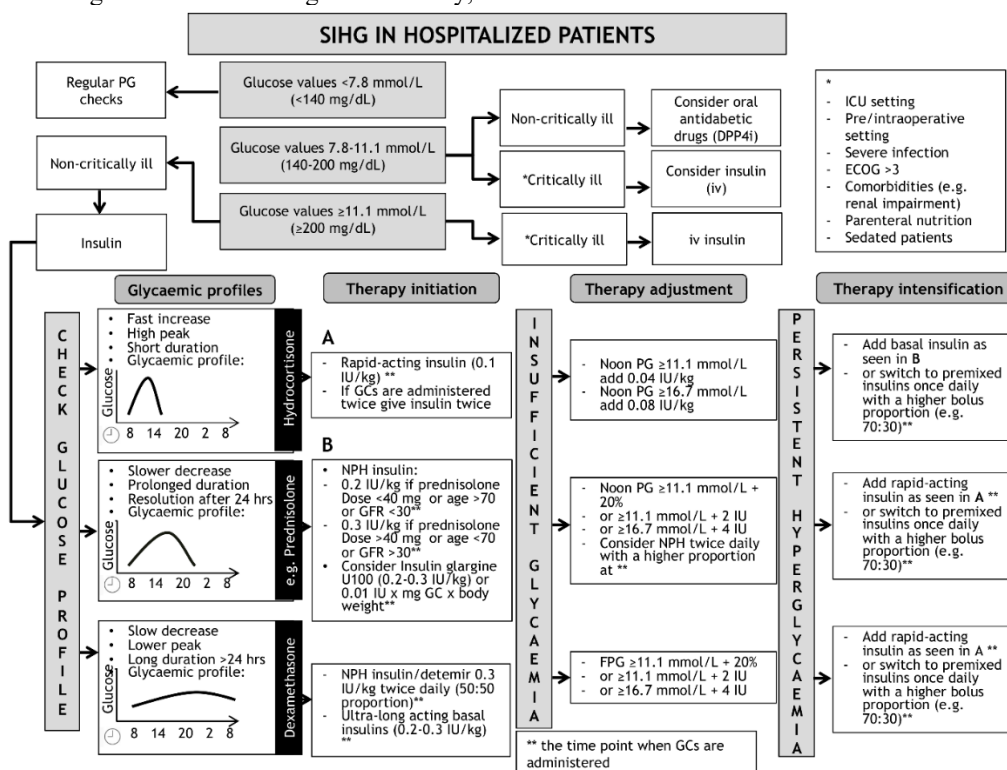
immunosuppressive treatment because of their high glucocorticoid activity, particularly in patients with COPD and solid organ transplant recipients. When a single dose is administered in the morning, as is the case with most prescriptions, hyperglycemia develops gradually but continuously, mostly lasts until the evening, and then gradually recovers until the following morning concurrently with the steroid agent's peak and duration of action. Short- or intermediate-acting basal insulins, such as insulin detemir or NPH (neutral protamine Hagedorn) insulin, are suggested to best fit this glucose pattern. Clore et al. recommended using 0.4 IU/kg of NPH insulin to start a weight-dependent regimen as part of a clinical guideline. clinical efficacy whether or not patients were fasting and when smaller doses of NPH (0.2–0.3 IU/kg) were given, depending on the GC dose. While two randomized studies using insulin glargine U100 at a fixed starting dose of 0.5 IU/kg or initiated according to admission glucose (0.3 or 0.4 IU/kg) showed non-inferiority compared to NPH insulin in terms of efficacy and safety, including nocturnal hypoglycemia, the kinetics of intermediate-acting glucocorticoids seem to fit the glucose-lowering property of NPH insulin. A study that used insulin glargine U100 integrated into a clinical decision support system for the treatment of SIHG in hospitals also revealed adequate performance. Starting basal insulin at a GC dosage-dependent dose—0.1 IU/kg BW if patients get 10 mg of prednisone or similar, 0.2 IU/kg BW if GC dose is 20 mg, 0.3 IU/kg BW if dose is set at 30 mg, and so on—is probably a realistic and straightforward strategy. It has been suggested that starting doses of insulin should be lower in patients with impaired renal function (eGFR < 30 mL/min/1.73 m²) or older than 70 years. Given that the GC is taken in the morning, subsequent dose modifications should be based on the accomplishment of glycaemic objectives as determined by glucose measures obtained the following morning. Because hyperglycemia may overlap and persist, multiple daily administrations of

intermediate-acting GCs are more complicated NPH insulin once a day won't work in this situation; either NPH insulin twice a day or a switch to longer-acting insulin (such as glargine) is needed. Additional fast-acting insulin boluses may be added as needed. This can be determined by either switching to premixed insulin, which is a combination of 70% rapid-acting and 30% basal insulin given in a manner similar to the GC consumption.

Long-Acting Glucocorticoids (Dexamethasone)

The most effective GC medication, dexamethasone, has an extended duration of action that lasts longer than 24 hours. It is used clinically in a number of situations, including inflammatory disorders, as an analgesic, and to lower brain pressure in cases of cerebral malignancy or cerebral edema. Regardless of diabetes status, dexamethasone has been advised for people with impaired gas exchange due to viral pneumonia during the recent severe acute respiratory syndrome coronavirus type 2 (COVID-19) pandemic. Since worsening glycaemic control and new-onset hyperglycemia were linked to worse outcomes in COVID-19 patients, this strategy warrants more research in diabetics. When glucose levels above a threshold of 12 mmol/L (~216 mg/dL), begin administering NPH insulin at a dose of 0.3 IU/kg/day; two thirds should be given in the morning and the remaining third in the evening. Additionally,

they suggest lowering the dosage to 0.15 IU/kg if the patient is older than 70 or has an eGFR of less than 30 mL/min. Insulin dosages should be adjusted based on morning or evening glucose levels, with a 20% reduction if glucose is less than 4.1 mmol/L (~70 mg/dL) and a 10% reduction if glucose is between 4.1 and 6.0 mmol/L (~70–110 mg/dL). Conversely, if glucose levels are higher than 18 mmol/L (~320 mg/dL), the insulin dose should be increased by 20%, and if they are between 12.1 and 18 mmol/L (~220–320 mg/dL), it should be increased by 10%. Generally speaking, hyperglycemia associated with long-acting GCs—which are typically taken in the morning—develops gradually, peaks at several times during the day, and persists for 24 hours following ingestion. Therefore, it is recommended to administer intermediate-acting basal insulins (NPH insulin, insulin detemir) twice a day at an initial dose of 0.3 IU/kg BW. Alternatively, the best insulin to treat hyperglycemia in this case may be long- or ultralong-acting basal insulin analogues (insulin glargine U100/U300 or insulin degludec; initial dose 0.2 IU/kg BW). Insulin dosage should be modified based on blood glucose levels 24 hours following GC consumption and the start of hyperglycemia. To the best of our knowledge, no research has been done to date to test new generation ultra-long-acting basal insulin analogues for SIHG treatment.^[8]



COMMON PRESCRIPTION PATTERN

Basal-bolus insulin regimen was the most commonly prescribed insulin strategy because it improves glycemic control in patients receiving steroid than sliding scale. Rapid acting bolus insulin was given prior to meals to reduce postprandial hyperglycemia brought on by corticosteroids, whereas basal insulin was used to maintain background glucose control. The pharmacokinetic profile of NPH insulin closely matched the pattern of glucose rise brought on by intermediate acting corticosteroids. Thereby patients on Prednisolone were often prescribed with intermediate acting insulin, such as NPH insulin. For long term glucose management, basal bolus regimens frequently included long acting basal insulin analogues such as insulin glargine.^[13]

PATIENT RELATED FACTORS

To avoid hyperglycemia, patients receiving glucocorticoid medication need to maintain a whole lifestyle. This comprises careful management without adverse effects, education to prevent hypoglycemia, and routine glucose monitoring for early identification. Until levels return to the normoglycemic range that existed prior to the start of glucocorticoid treatment, glucose monitoring

should be continued. In order to check for the onset of GIH, a HbA1c test is also advised three months after beginning glucocorticoids. Patients should be made aware of the signs of acute hyperglycemia, which include weight loss, polyuria, and polydipsia. For patients to understand the possible adverse effects of different glucose-lowering medications, education is also crucial. People who use insulin or insulin secretagogues and are susceptible to hypoglycemia should be aware of the significance of routine fasting blood glucose testing and know how to modify their medication to avoid hypoglycemia, especially if they intend to taper or stop glucocorticoid therapy.^[14]

CHALLENGES IN MANAGEMENT

Nursing staff may find it challenging to execute sophisticated insulin regimens, adding to their workload and sometimes compromising patient safety. However, from a patient perspective, a once-daily morning NPH regimen may be easier to learn than multiple daily injections, particularly for patients who will be discharged home on GC therapy. Patients receiving any type of supraphysiologic GC who are treated with a basal-bolus regimen require a higher percentage of nutritional insulin to achieve normoglycemia.

Establishing procedures and guidelines that describe the best ways to achieve and maintain glycemic control, such as administering NPH concurrently with intermediate-acting GCs like prednisone or methylprednisolone, may help overcome obstacles to treating GC-associated hyperglycemia. Establishing these standards may result in higher expenses related to teaching nurses and healthcare professionals how to administer complicated insulin regimens that need for closer observation. For patients with GC-associated hyperglycemia in the hospital, the recommendation was based on low-certainty evidence showing comparable glycemic outcomes for mean BG, hyperglycemia, hypoglycemia, and hospital LOS with regimens based on NPH and BBI. For the glycemic control of GC-associated hyperglycemia, either NPH- or BBI-based regimens. Cost, viability, acceptability, and equity benefits were not shown by either regimen.^[15] Patients with SIH/SIDM have no indications for post-discharge care. Believe that the current recommendations for managing diabetic patients after discharge can be taken into consideration. Specifically, each patient should have a defined discharge plan based on the discharge environment (home with or without visiting nurse services, assisted living, rehabilitation, skilled nursing institutions, etc.). When it comes to the type and length of glucocorticoid therapy, self-monitoring blood glucose levels and antidiabetic medication will play a significant role in the management of SIH/SIDM in self-sufficient patients who are returned home. Within a month, all patients should schedule an outpatient follow-up visit with a doctor (primary care physician, endocrinologist, or diabetes educator).^[16]

II. CONCLUSION

Steroid – induced hyperglycemia is a significant clinical challenge among Type 2 diabetic patients receiving corticosteroid therapy for respiratory infections. The reviewed literatures indicates that insulin is the treatment for managing steroid induced hyperglycemia in hospitalized patients. Among the various insulin regimens, basal-bolus insulin therapy was found to provide better glycemic control compared to sliding scale insulin, as it more closely mimics normal physiological insulin secretion and helps maintain stable blood glucose levels throughout the day. Sliding scale insulin alone was associated with fluctuating glucose levels and less effective glycemic management.

Insulin prescription patterns were influenced by factors such as steroid dose and

duration, severity of hyperglycemia, patient comorbidities and hospital treatment protocols. Early initiation of insulin therapy, individualized dose adjustment and regular blood glucose monitoring were identified as important strategies for achieving optimal therapeutic outcomes.

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