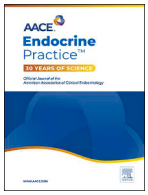
Endocrine Practice xxx (xxxx) xxx

Endocrine Practice™
www.endocrinepractice.org



Review Article

Glucocorticoid-Induced Hyperglycemia in Patients With Cancer: Mechanisms, Clinical Implications, and Management Strategies

Luana Lury Morikawa, Medical Intern [1], Marcia Nery, MD, PhD [2], Marcos Tadashi Kakitani Toyoshima, MD, PhD [3, 4, *]

[1] *Faculty of Medicine, Universidade Nove de Julho - Campus Guarulhos, São Paulo, Brazil*
[2] *Division of Endocrinology and Metabolism, Hospital das Clinicas da Faculdade de Medicina da Universidade de Sao Paulo (HCFMUSP), São Paulo, Brazil*
[3] *Endocrine Oncology Service, Instituto do Cancer do Estado de São Paulo Octávio Frias de Oliveira, Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo (HCFMUSP), Sao Paulo, Brazil*
[4] *Laboratório de Carboidratos e Radioimunoensaio (LIM18), Hospital das Clinicas da Faculdade de Medicina da Universidade de Sao Paulo (HCFMUSP), Sao Paulo, Brazil*

ARTICLE INFO

Article history:
Received 19 October 2025
Received in revised form
29 December 2025
Accepted 10 January 2026
Available online xxx

Key words:
cancer
chemotherapy
diabetes
glucocorticoids
hyperglycemia

ABSTRACT

Objective: To review the pathophysiology, risk factors, clinical implications, monitoring strategies, and therapeutic approaches for glucocorticoid-induced hyperglycemia (GCIH), with a focus on patients with cancer.
Methods: This narrative review integrates findings from clinical studies, expert guidelines, and recent advances in glucose monitoring and pharmacologic therapy, particularly in oncologic settings where glucocorticoid use is common.
Results: GCIH is a frequent and often underrecognized complication, even in individuals without pre-existing diabetes. In patients with cancer, GCIH is associated with increased risk of infections, chemotherapy delays, longer hospital stays, and higher mortality. Key mechanisms include enhanced insulin resistance, increased hepatic gluconeogenesis, and β-cell dysfunction. Afternoon and postprandial hyperglycemia are typical due to the pharmacodynamics of once-daily morning glucocorticoids. Self-monitoring of blood glucose (SMBG) and continuous glucose monitoring (CGM) are essential tools. HbA1c may assist in baseline assessment, but fructosamine better reflects short-term glycemic changes. Insulin is the treatment of choice for moderate to severe GCIH, with basal-bolus regimens, especially using NPH insulin aligned with glucocorticoid timing, providing effective control. Selected non-insulin agents may be considered in stable outpatients with mild hyperglycemia. However, standardized definitions, evidence-based algorithms, and randomized trials remain limited.
Conclusion: Optimal GCIH management requires proactive monitoring and individualized treatment strategies tailored to glucocorticoid type, dose, and clinical setting. Further research should aim to refine diagnostic criteria, validate therapeutic protocols, and assess emerging technologies such as automated insulin delivery systems and selective glucocorticoid receptor modulators.

Abbreviations: 11β-HSD1, 11β-hydroxysteroid dehydrogenase type 1; ADA, American Diabetes Association; AGRP, agouti-related peptide; AI, artificial intelligence; AID, automated insulin delivery; Akt, serine/threonine kinase Akt or protein kinase B; BBI, basal–bolus insulin; CART, chimeric antigen receptor T-cell; CGM, continuous glucose monitoring; DKA, diabetic ketoacidosis; DPP-4, dipeptidyl peptidase-4; eGFR, estimated glomerular filtration rate; EGFR, epidermal growth factor receptor; ER, endoplasmic reticulum; GCIH, glucocorticoid-induced hyperglycemia; GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide-1; GLUT2, glucose transporter type 2; GLUT4, glucose transporter type 4; G6Pase, glucose-6-phosphatase; GRE, glucocorticoid response element; HbA1c, glycated hemoglobin; HHS, hyperosmolar hyperglycemic state; ICI, immune checkpoint inhibitors; ICU, intensive care unit; IRS1, insulin receptor substrate 1; JBDS, Joint British Diabetes Societies; MAPK, mitogen-activated protein kinase; mTOR, mammalian target of rapamycin; NPH, neutral protamine hagedorn (insulin); NPY, neuropeptide Y; PD-1, programmed cell death protein 1; PD-L1, programmed death-ligand 1; PEPCK, phosphoenolpyruvate carboxykinase; PI3K, phosphoinositide 3-kinase; RCT, randomized controlled trial; SGLT2, sodium–glucose cotransporter 2; SMBG, self-monitoring of blood glucose; SNP, single-nucleotide polymorphism.

* Address correspondence to Dr Marcos Tadashi Kakitani Toyoshima, Endocrine Oncology Service, Instituto do Câncer do Estado de São Paulo Octávio Frias de Oliveira, Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, Av. Dr. Arnaldo, 251 - 1o andar - Cerqueira César, Sao Paulo, Brazil 01246-000.
E-mail address: marcos.tadashi@hc.fm.usp.br (M.T.K. Toyoshima).

https://doi.org/10.1016/j.eprac.2026.01.024
1530-891X/

Please cite this article as: L.L. Morikawa, M. Nery and M.T.K. Toyoshima, Glucocorticoid-Induced Hyperglycemia in Patients With Cancer: Mechanisms, Clinical Implications, and Management Strategies, Endocrine Practice, https://doi.org/10.1016/j.eprac.2026.01.024

Introduction

Glucocorticoid-induced hyperglycemia (GCIH) is a frequent yet often underrecognized complication of glucocorticoid therapy. Even individuals without preexisting diabetes may experience transient or persistent elevations in blood glucose, particularly when exposed to high-dose or prolonged glucocorticoid regimens.[1-8]

Glucocorticoids are widely prescribed in oncology, not only as antiemetics, components of chemotherapy protocols, but also for the management of cerebral edema, immune-related adverse events, and graft-versus-host disease in hematopoietic stem-cell transplantation.[1,9] Additionally, they are frequently used in supportive and palliative care for specific indications such as spinal cord compression, lymphangitic carcinomatosis, bowel obstruction, and raised intracranial pressure. Glucocorticoids may also be employed for refractory symptoms including pain, anorexia, fatigue, and dyspnea, although the evidence supporting these nonspecific uses is less robust.[10]

In this setting, GCIH has been associated with increased risks of infection, chemotherapy delays, prolonged hospital stays, and poorer overall outcomes.[1,4,11,12] In oncology, glucocorticoids such as prednisone, dexamethasone, and methylprednisolone are commonly prescribed with variable schedules and potencies. These pharmacologic differences influence the timing and magnitude of hyperglycemia and must be considered when selecting insulin types and schedules (see Supplementary Tables 1 and 2 for pharmacokinetic profiles of glucocorticoids and insulins commonly used in oncology).[13-15]

Despite its clinical relevance, there is currently no standardized definition of GCIH and no universally accepted guidelines for its monitoring and management.[16] Most recommendations are extrapolated from general diabetes care or inpatient hyperglycemia protocols, which may not fully reflect the unique metabolic and therapeutic complexities complexities of patients receiving glucocorticoids.

This narrative review summarizes current evidence on the pathophysiology, risk factors, diagnosis, monitoring, and treatment of GCIH, with a special focus on patients with cancer. We present practical strategies for glucose monitoring, pharmacologic treatment, and we discuss emerging technologies that may enhance the detection and management of GCIH in this high-risk population.

The level of evidence cited in this review was classified as follows.[17]

- Level I: evidence from at least one properly randomized controlled trial (RCT);
- Level II-1: evidence from well-designed cohort or case-control analytic studies;
- Level II-2: evidence from comparisons between times or places with or without intervention;
- Level III: expert opinion based on clinical experience, descriptive studies, or reports of expert committees.

For clarity, we use 'glucocorticoid' to refer to the subset of corticosteroids with clinically relevant anti-inflammatory and metabolic effects.

Highlights

- Glucocorticoid-induced hyperglycemia (GCIH) is common, underdiagnosed, and associated with worse outcomes in individuals with cancer, including higher rates of infection, chemotherapy delays, and increased mortality
- Afternoon and postprandial hyperglycemia are characteristic of GCIH, particularly with once-daily morning corticosteroids, highlighting the importance of appropriately timed glucose monitoring
- Insulin remains the mainstay of GCIH management, particularly in moderate to severe cases. Weight-based NPH insulin timed to glucocorticoid administration is a practical and effective strategy
- Non-insulin therapies may be cautiously considered for mild GCIH in stable outpatients, although clinical evidence remains limited, especially in oncology settings
- Gaps in clinical practice include the absence of standardized diagnostic criteria, insufficient screening of individuals without known diabetes, and limited clinical trial data—emphasizing the need for personalized management and further research

Clinical Relevance

Glucocorticoid-induced hyperglycemia is highly prevalent in oncology but often overlooked. This review provides practical strategies for monitoring and treatment, with emphasis on insulin regimens tailored to glucocorticoid type and timing, aiming to reduce complications and improve outcomes in individuals with cancer.

Mechanisms of Glucocorticoid-Induced Hyperglycemia

GCIH results from a combination of increased hepatic glucose production, reduced peripheral glucose uptake, and impaired pancreatic β-cell function. These effects are dose-dependent and can occur rapidly, even in individuals without preexisting diabetes.[18,19]

Increased Insulin Resistance

Glucocorticoids, especially at moderate to high doses, lead to insulin resistance by reducing glucose uptake in peripheral tissues such as skeletal muscle and adipose tissue. This effect is mediated in part by inhibition of GLUT4 translocation to the cell membrane, rather than direct downregulation of total GLUT4 expression, thereby reducing insulin-stimulated glucose utilization. In adipose tissue, glucocorticoids stimulate lipolysis, increasing circulating free fatty acids, which further impair insulin signaling via inhibition of the PI3K/Akt pathway.[18-21] These molecular alterations contribute to the characteristic postprandial and afternoon hyperglycemia seen in GCIH. Notably, once-daily morning administration of intermediate-acting glucocorticoids such as prednisone leads to peak pharmacologic effects in the afternoon, coinciding with enhanced hepatic gluconeogenesis and maximal peripheral insulin resistance. This mechanistic alignment explains the temporal pattern of hyperglycemia typically observed in GCIH patients, particularly those without preexisting diabetes.[3,21,22]

Hepatic Gluconeogenesis

A hallmark of glucocorticoid action is the stimulation of hepatic gluconeogenesis. Glucocorticoids upregulate key gluconeogenic enzymes, such as phosphoenolpyruvate carboxykinase (PEPCK) and glucose-6-phosphatase (G6Pase), leading to excessive hepatic glucose output, particularly in fasting or postabsorptive states.[18-21]

This is facilitated by glucocorticoid binding to glucocorticoid-response elements (GREs) in hepatocytes, where co-regulators like FOXO1 and PGC1α further enhance gluconeogenic gene transcription. Additionally, glucocorticoid-induced proteolysis in skeletal muscle and lipolysis in adipose tissue increase the availability of amino acids and glycerol as substrates for gluconeogenesis, amplifying hepatic glucose production.[15-17,20]

Pancreatic β-Cell Dysfunction

Acute glucocorticoid exposure can initially lead to compensatory hyperinsulinemia, but prolonged or high-dose exposure impairs β-cell function and promotes apoptosis. Chronic glucocorticoid exposure activates inflammatory and stress-related signaling pathways, notably the p38 MAPK pathway, contributing to oxidative stress and endoplasmic reticulum stress. This ultimately leads to β-cell exhaustion and apoptosis, particularly in susceptible individuals.[5,18,19] Furthermore, glucocorticoids downregulate GLUT2 expression in pancreatic β-cells, compromising glucose sensing and impairing insulin secretion.[5,19]

Epigenetic mechanisms also appear to modulate β-cell susceptibility to glucocorticoids. Methylation of the *NR3C1* gene (encoding the glucocorticoid receptor) and altered chromatin accessibility may affect receptor signaling in β-cells and hepatocytes, influencing interindividual variability in glycemic response.[19,23-25]

Glucocorticoid-Induced Appetite and Weight Gain

Glucocorticoids increase appetite and promote central adiposity through both hypothalamic and peripheral mechanisms. In the central nervous system, glucocorticoids upregulate neuropeptide Y (NPY) and agouti-related peptide (AgRP) in the arcuate nucleus, enhancing food intake. Simultaneously, they induce leptin resistance and shift dietary preference toward calorie-dense foods, contributing to weight gain and worsening insulin resistance.[18,19] These effects are particularly relevant in patients with cancer, where appetite stimulation may initially appear beneficial but may later exacerbate metabolic dysregulation.

Epidemiology and Risk Factors of Glucocorticoid-Induced Hyperglycemia

Hyperglycemia is common among patients receiving glucocorticoid therapy. The incidence of new-onset glucocorticoid-induced diabetes mellitus has been reported to range from 15% to 40% in patients treated for respiratory diseases, renal disorders, cancer, solid organ transplantation, or autoimmune diseases such as rheumatoid arthritis.[18] However, specific prevalence data in populations with cancer remain limited, with rates varying according to cancer type, glucocorticoid regimen (dose, duration, and potency), and patient-related factors such as age, nutritional status, and baseline glycemic profile. The development of GCIH is multifactorial, influenced by patient characteristics, pharmacologic exposures, and genetic predisposition. Table 1 summarizes the key risk factors associated with GCIH, along with supporting evidence from the literature.

Clinical and demographic risk factors include: advanced age, obesity, family history of type 2 diabetes, baseline HbA1c $\geq$ 6%, and reduced renal function.[18,24,26]

Pharmacologic risk factors also play a central role in the development of GCIH. The dose, potency, and duration of glucocorticoid therapy are directly associated with the risk of hyperglycemia. Higher-potency and longer-acting agents, such as dexamethasone, are more likely to induce sustained glycemic elevations than shorter-acting glucocorticoids like hydrocortisone. Additionally, systemic exposure depends on the dose, formulation, and duration, with oral regimens often leading to sustained exposure in outpatient settings.[4,18,26,27]

Genetic and molecular factors may further explain the interindividual variability in glycemic response to glucocorticoids. Variations in epidermal growth factor receptor (EGFR) expression and glucocorticoid receptor sensitivity have been linked to differing degrees of insulin resistance and glucose intolerance following glucocorticoid exposure, highlighting the future potential for personalized risk prediction.[19,23,25]

Clinical Presentation and Diagnosis

GCIH often presents subtly and may be underrecognized, particularly in patients without a history of diabetes. Early identification relies on proactive glucose monitoring rather than symptom recognition alone, especially in high-risk populations receiving moderate to high-dose glucocorticoid therapy.

Hyperglycemia is defined by the American Diabetes Association (ADA) as a fasting plasma glucose $\geq$126 mg/dL or a random glucose $\geq$200 mg/dL.[11,28] However, these thresholds may not fully capture the circadian glycemic fluctuations typical of GCIH. Specifically, GCIH often peaks in the postprandial and afternoon periods, particularly when glucocorticoids are administered in the morning. Consequently, fasting glucose levels may appear normal, potentially delaying diagnosis if not supplemented by targeted postprandial monitoring.[6,7,13,29,30]

The clinical presentation of GCIH varies depending on the degree and duration of hyperglycemia.

- Asymptomatic hyperglycemia is common, particularly in patients without preexisting diabetes and in those with glucose levels <200 mg/dL.

Table 1
Risk Factors for Glucocorticoid-Induced Hyperglycemia (GCIH)

Risk factor	Description	Supporting evidence
Glucocorticoid Dose, Duration, and Route	Higher doses, prolonged use, and oral administration increase GCIH risk.	4,15,23,24
Type of Glucocorticoid	Dexamethasone and methylprednisolone induce greater hyperglycemia than prednisolone or hydrocortisone.	4,23
Age	Older adults (>60 y) are at increased risk due to insulin resistance and decreased β-cell function.	15,21,23
Pre-existing Metabolic Conditions	Obesity, prediabetes, prior GDM, HbA1c $\geq$ 6%, and reduced eGFR (<40 mL/min/1.73 m^2).	15,21,23
Concomitant Medications	Calcineurin inhibitors and loop diuretics may worsen hyperglycemia.	15,23
Genetic and Epigenetic Factors	EGFR downregulation, SNPs, chromatin changes, and polygenic diabetes risk scores contribute to GCIH.	16,20-22
Family History of Type 2 Diabetes	Glucocorticoid-induced diabetes is more common in individuals with a family history of diabetes.	15,23

Abbreviations: eGFR = estimated glomerular filtration rate; EGFR = epidermal growth factor receptor; GCIH = glucocorticoid-induced hyperglycemia; GDM = gestational diabetes; SNP = single nucleotide polymorphism.

- Mild symptoms, such as polyuria, polydipsia, fatigue, polyphagia, and blurred vision, may emerge when glucose levels exceed 200 mg/dL.
- Severe hyperglycemia, particularly with glucose levels >300 mg/dL, increases the risk for acute complications such as diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS) in susceptible individuals.

In individuals undergoing chemotherapy, symptoms like fatigue, nausea, or malaise may be misattributed to treatment side effects, further complicating the recognition of GCIH.[3,28] Uncontrolled hyperglycemia in hospitalized patients is associated with immune dysfunction, oxidative stress, increased risk of infections, thrombotic events, and longer hospital stays. These effects are particularly detrimental in oncology settings, where GCIH may compromise cancer treatment delivery and overall prognosis.[1,4,11,31]

Glucose Monitoring

Glycemic monitoring in individuals with cancer presents unique challenges. GCIH may be underdiagnosed due to overlapping symptoms with chemotherapy side effects (eg, fatigue, nausea, polyuria) or due to irregular monitoring practices in patients without a prior diagnosis of diabetes (Level of Evidence III).[32]

Effective glucose monitoring is essential for the timely detection and management of GCIH. Because GCIH often presents with postprandial and afternoon glycemic excursions, especially following morning glucocorticoid administration, reliance on fasting glucose or HbA1c alone may fail to detect early dysglycemia (Level of Evidence II-1).[3,25]

Capillary Glucose Monitoring

- Self-monitoring of blood glucose (SMBG) or point-of-care (POC) capillary glucose may be considered at the initiation of glucocorticoid therapy, even in individuals without diabetes, especially in those at high-risk (eg, older adults, elevated HbA1c, history of prediabetes), or those receiving moderate to high doses of glucocorticoids (Level of Evidence II-2).[3,4,11,32-34]
- Two-hour post-lunch and pre-dinner blood glucose measurements are particularly informative, as the glycemic peak typically occurs 6-12 h after morning glucocorticoid administration[3,4,11,33,34] (Level of Evidence II-1).
- In hospitalized patients, pre-meal and bedtime glucose monitoring is recommended as part of standard inpatient hyperglycemia protocols (Level of Evidence II-1).[22]
- In outpatient settings, targeted postprandial monitoring (eg, 2 h after lunch) or late afternoon (pre-dinner) may be sufficient for individuals at lower risk. While universal glucose monitoring in all outpatient scenarios is not required, selective afternoon glucose measurements can improve early detection of GCIH and facilitate prompt intervention (Level of Evidence III).[35]

Continuous Glucose Monitoring (CGM)

CGM provides real-time glucose trends and enables the identification of circadian glycemic patterns, making it particularly useful in oncology settings (Level of Evidence II-1).

In a pilot study involving individuals with hematologic malignancies and no prior diagnosis of diabetes, 60% exhibited interstitial glucose >180 mg/dL for ≥1 hour during glucocorticoid therapy, with prominent glycemic peaks observed in the afternoon and evening hours.[3]

CGM may be especially valuable in patients with cancer undergoing chemotherapy or in those with variable oral intake or irregular treatment schedules[3,29,30,36] (Level of Evidence II-2).

Although not yet routinely adopted in inpatient or oncology care, the use of CGM is expanding in research and select clinical scenarios. Further studies are warranted to assess its cost-effectiveness and clinical utility in this high-risk population.

HbA1c and Fructosamine Testing

Baseline HbA1c should be considered prior to initiating glucocorticoids to evaluate underlying glycemic status (Level of Evidence III)[11,30,32,37,38] While still useful for longitudinal monitoring, HbA1c may not reliably reflect short-term glycemic excursions, commonly seen with glucocorticoid therapy, particularly in low-dose or short-term regimens (Level of Evidence II-1). Clinically meaningful changes in HbA1c typically require sustained hyperglycemia, as observed with prolonged or high-dose glucocorticoid exposure.[11,32,33,39,40]

Importantly, an HbA1c ≥ 6.0% has been identified as an independent risk factor for the development of GCIH, highlighting the need for enhanced glucose monitoring in these individuals[24] (Level of Evidence II-2).

In oncology settings, interpretation of HbA1c must be approached with caution due to several confounding factors, including anemia, frequent blood transfusions, chemotherapy-induced bone marrow suppression, and renal dysfunction. These conditions may shorten erythrocyte lifespan or affect hemoglobin glycation kinetics, potentially leading to falsely low or unreliable HbA1c values. Additionally, hemoglobinopathies such as sickle cell disease or thalassemia may interfere with specific HbA1c assay methodologies (Level of Evidence II-2)[28,32,35] Nevertheless, findings from the GlicoOnco study found strong correlations between HbA1c, fructosamine, and 3-month SMBG, supporting the use of HbA1c even in patients with mild anemia[41] (Level of Evidence II-1).

Fructosamine, which reflects average glycemia over the preceding 2 to 3 weeks, is more sensitive to short-term changes.[41] In the same study, fructosamine showed good correlation with 1-month SMBG and consistent performance in patients receiving chemotherapy, glucocorticoids, or presenting with mild hypoproteinemia[41] (Level of Evidence II-1).

Taken together, HbA1c and fructosamine provide complementary information and should be selected based on clinical context and patient-specific factors.

Management Strategies

The management of GCIH should be individualized according to the degree of hyperglycemia, the glucocorticoid type and regimen, and the patient's clinical context and baseline glycemic status. Treatment typically integrates lifestyle modifications, pharmacologic interventions, and frequent glucose monitoring to achieve safe and effective glycemic control.

Insulin remains the mainstay of treatment for moderate to severe GCIH, particularly in hospitalized or acutely ill patients. However, non-insulin agents may be appropriate in select outpatient settings or for individuals with milder forms of hyperglycemia. Regardless of chosen therapy, close glucose monitoring and timely dose adjustments are critical to avoid both hyperglycemia and hypoglycemia throughout the course of glucocorticoid treatment (Level of Evidence III).

Non-Insulin Therapies

Non-insulin agents may be considered in stable outpatient settings, especially in cases of mild hyperglycemia (blood glucose <180-200 mg/dL) and in individuals without preexisting diabetes (Level of Evidence III). Their use should be individualized based on renal function, weight, comorbidities, cancer status, and risk of adverse effects.[1,5,18,26]

- Metformin improves insulin sensitivity and reduces hepatic glucose production. It may help lower postprandial glucose levels. However, its delayed onset limits its utility in acute settings. It should be avoided in patients with renal dysfunction or increased risk of lactic acidosis[16,18,26,37,38,42] (Level of Evidence II-1: observational data showing effectiveness in mild GCIH; limited RCT data in cancer settings).
- Sulfonylureas and glinides: Short-acting sulfonylureas such as gliclazide have shown efficacy in dexamethasone-induced hyperglycemia.[18,38,43-45] However, gliclazide is not FDA-approved in the United States; therefore, U.S. clinicians may consider glipizide as an alternative. Caution is warranted in older adults due to the risk of hypoglycemia. Glinides (eg, repaglinide) offer rapid postprandial glucose control with lower hypoglycemia risk, but require multiple daily doses and may be limited by cost and access[18,37,44] (Level of Evidence II-2).
- Thiazolidinediones or Glitazones have shown efficacy in managing GCIH in early studies.[46,47] However, safety concerns, particularly related to fluid retention, weight gain, and risk of heart failure, limit their use, especially in individuals with cancer or cardiovascular comorbidities. Despite a low risk of hypoglycemia, they are rarely used in this context due to their slow onset and adverse effect profile[18,37,43,46,48] (Level of Evidence II-1: RCT evidence for efficacy in GCIH, but safety concerns).
- α-Glucosidase inhibitors delay intestinal carbohydrate absorption and modestly reduce postprandial glycemia. However, there is scarce evidence supporting the use in GCIH, and gastrointestinal side effects often limit tolerability[18] (Level of Evidence III: limited general diabetes data; little/no data in GCIH).
- DPP-4 inhibitors are weight-neutral, generally well tolerated, and may provide modest improvements in postprandial glycemia. Several studies support their use in mild GCIH (eg, glucose <180 mg/dL).[49-51] However, results are inconsistent, especially under high-dose glucocorticoid regimens, where efficacy in preventing postprandial hyperglycemia was not observed.[52] Therefore, DPP-4 inhibitors are not routinely recommended as monotherapy for GCIH but may be considered in stable outpatients with low to moderate hyperglycemia[16,18,37,38,44] (Level of Evidence II-1).
- GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists may benefit patients with overweight or glucocorticoid-induced hyperphagia, but may worsen anorexia or cachexia in patients with cancer (Level of Evidence III). The role of GLP-1 agonists in GCIH remains exploratory, and current evidence is limited.[18,37,53] No studies have evaluated dual GLP-1/GIP receptor agonist (eg tirzepatide) in GCIH specifically (Level of Evidence II-2: extrapolated from type 2 diabetes studies).
- Sodium-glucose cotransporter 2 (SGLT2) inhibitors may reduce insulin requirements in select patients with preexisting diabetes, but concerns about euglycemic diabetic ketoacidosis (DKA), dehydration, and genitourinary infections limit their use during acute illness or fasting[16,18,44,54] (Level of Evidence III: expert opinion; safety concerns; minimal GCIH data).

Evidence Summary for Non-insulin Therapies

Clinical data supporting the use of non-insulin therapies in GCIH remain limited.[16,37] Among the available options, metformin has shown modest benefits in reducing postprandial glucose levels, and DPP-4 inhibitors have demonstrated variable efficacy, with some studies suggesting utility in mild GCIH.[16,18,37] For other non-insulin agents, including SGLT2 inhibitors, GLP-1 receptor agonists, dual GLP-1/GIP receptor agonist, α-glucosidase inhibitors, and thiazolidinediones, evidence is either scarce or extrapolated from studies in individuals with type 2 diabetes, without specific evaluation in GCIH. In particular, their safety profiles in individuals with cancer raise concerns (eg, risk of dehydration, weight loss, or heart failure), further limiting their widespread use in oncology.

Table 2 summarizes the mechanism of action, clinical applicability, advantages, and limitations of these agents in GCIH.

Insulin-Based Therapies

Insulin is the preferred treatment for GCIH when blood glucose exceeds 200-250 mg/dL, particularly in individuals with preexisting diabetes or those hospitalized with moderate to severe hyperglycemia. This threshold is supported by inpatient hyperglycemia management guidelines and expert consensus, although randomized trials specifically evaluating optimal glucose cutoffs for GCIH are lacking[5,43,55] (Level of Evidence III). Treatment regimens should consider the type, dose, and timing of glucocorticoids, as well as the patient's baseline glycemic profile and clinical status.[43] Individualized assessment is essential. Some patients may tolerate higher glucose levels without acute complications, whereas others, such as those with infections, undergoing chemotherapy, or admitted to the intensive care unit (ICU), may benefit from earlier intervention.

In hospitalized patients, insulin regimen choice should be guided by illness severity.

- Critically ill patients (eg, those in intensive care units or with hyperglycemic emergencies) should receive intravenous insulin infusions, allowing rapid titration and tighter control in the setting of hemodynamic instability, sepsis, or enteral feeding interruptions (Level of Evidence I: extrapolated from ICU hyperglycemia trials).[56,57]
- Non-critically ill inpatients should be managed with subcutaneous insulin regimens, preferably basal-bolus strategies tailored to glucocorticoid pharmacokinetics and nutritional intake (Level of Evidence I).[55]

For patients receiving once-daily prednisone or prednisolone, NPH insulin administered at the same time as the glucocorticoid (usually in the morning) is preferred to match the expected afternoon glycemic peak. For long-acting glucocorticoids (eg, dexamethasone), long-acting basal analogs such as glargine or degludec are more appropriate.

Prandial and Correction Insulin Strategies

Prandial insulin, with or without correctional doses, is appropriate for patients receiving short-acting glucocorticoids (eg, hydrocortisone). A common starting dose is 0.1 U/kg per meal, titrated based on postprandial glucose monitoring. If hyperglycemia persists despite bolus insulin, basal insulin should be added[5,16,18] (Level of Evidence II-2).

In patients with impaired oral intake, such as those experiencing nausea or vomiting, scheduled prandial insulin

Table 2
Non-Insulin Therapies for Glucocorticoid-Induced Hyperglycemia (GCIH)

Class/agents	Mechanism of action	Use in GCIH	Advantages	Limitations	Level of Evidence[14]
Biguanide (eg, metformin)	Reduces hepatic glucose production; improves insulin sensitivity	Effective in mild GCIH, especially with prednisone	Inexpensive; weight neutral	Contraindicated in renal dysfunction or risk of lactic acidosis; delayed onset	II-1
Sulfonylureas (eg, glipizide, gliclazide)	Stimulates insulin secretion	May be considered in selected cases of dexamethasone-induced hyperglycemia, particularly in non-hospitalized patients without significant risk factors for hypoglycemia	Low cost; potential effectiveness in glucocorticoid-associated hyperglycemia	Risk of hypoglycemia, especially in older adults or patients with variable oral intake; should be used with caution and individualized	II-2
Glinides (eg, repaglinide, nateglinide)	Stimulates rapid insulin release	Useful for postprandial glucose control	Lower hypoglycemia risk vs sulfonylureas	Requires frequent dosing; costly	II-2
Thiazolidinediones (eg, pioglitazone)	Improves insulin sensitivity in muscle and fat	Potentially effective, but limited by safety concerns.	Low hypoglycemia risk	Weight gain; fluid retention; slow onset	II-1
α-Glucosidase Inhibitors (eg, acarbose)	Delays intestinal carbohydrate absorption	Scarce evidence	Minimal hypoglycemia	GI side effects	III
DPP-4 Inhibitors (eg, sitagliptin, saxagliptin. linagliptin, alogliptin)	Inhibits breakdown of incretins	Inconsistent evidence in GCIH	Weight neutral; low hypoglycemia risk	Modest effect on glycemia	II-1
GLP-1 Receptor Agonists (eg, liraglutide, dulaglutide, semaglutide) or Dual GLP-1/GIP receptor Agonist (eg, tirzepatide)	Enhances insulin secretion; suppresses appetite	Potential benefit in overweight patients	Weight loss; reduced appetite	May worsen cachexia; GI side effects; costly	II-2
SGLT2 Inhibitors (eg, dapagliflozin, empagliflozin)	Increases urinary glucose excretion	Investigational in GCIH	May reduce insulin needs	Risk of euglycemic DKA, dehydration; avoid in acute illness	III

Abbreviations: DKA = diabetic ketoacidosis; DPP4 = Dipeptidyl Peptidase-4; GCIH = glucocorticoid-induced hyperglycemia; GI = gastrointestinal; GIP = Glucose-Dependent Insulinotropic Polypeptide; GLP-1 = Glucagon-Like Peptide-1; SGLT2 = Sodium–Glucose Cotransporter 2.

should be temporarily suspended to reduce the risk of hypoglycemia. In such scenarios, correctional insulin may be used alone to treat significant hyperglycemia, with basal insulin maintained or adjusted according to glucocorticoid exposure (Level of Evidence III).

Basal Insulin Strategies

Basal insulin choice should be selected based on the pharmacokinetics of the glucocorticoid being used.[16,43,58]

- NPH insulin is preferred for once-daily prednisone or prednisolone. It should be administered at the same time as the glucocorticoid, usually in the morning, to best match the timing of steroid-induced insulin resistance and the afternoon glycemic peak[16,18,58-63] (Level of Evidence I). We recommend initiating weight-based NPH insulin at a dose of 0.1 IU/kg for every 10 mg of prednisone (or equivalent), up to a maximum of 0.4 IU/kg, as suggested by Clore and Thurby-Hay (2009)[26,38,64] (Level of Evidence III). For example, in a 70 kg patient receiving 40 mg of prednisone daily, the initial NPH insulin dose would be calculated as 0.1 IU/kg x 4 (4 × 10 mg increments), resulting in 0.4 IU/kg, or 28 units of NPH insulin administered at same time as prednisone.
- Long-acting analogs (glargine, degludec) are better suited for prolonged glucocorticoid exposure (eg, dexamethasone or multiple daily doses) or when nocturnal control is needed[16,20,37,58] (Level of Evidence I).
- Combination strategies may be used in selected patients: for example, adding morning NPH insulin to a long-acting analog to provide postprandial coverage during intermediate-acting glucocorticoid therapy[16,18,37,60,65] (Level of Evidence I). When adding NPH to an existing basal regimen, a similar weight-based approach may be used (0.1 IU/kg per 10 mg of prednisone (or equivalent), up to 0.4 IU/kg.[16,26,38] (Level of Evidence III). For instance, in a 70 kg patient receiving 40 mg prednisone, the initial NPH dose would be 28 units administered concurrently with the glucocorticoid.

Basal-Bolus Regimen (BBI)

BBI regimen remains the most studied and recommended insulin regimen for hospitalized patients with GCIH, particularly those with poorly controlled diabetes or high-dose glucocorticoid therapy.[37,38] A typical starting dose is 0.5 to 0.6 IU/kg/d, divided equally between basal and bolus components.[66-68] This strategy is supported by inpatient glycemic control trials and expert consensus (Level of Evidence I). These insulin doses are suggested starting points and should be titrated based on glycemic response, prior insulin use, and the patient's clinical status.

In patients already on a BBI regimen who initiate glucocorticoid therapy, adjustments may be required to address GCIH. For patients already on BBI who start prednisone, consider adding supplemental NPH timed with prednisone administration. In contrast, for individuals receiving dexamethasone, the treatment strategy should be to increase the existing scheduled basal dose (long-acting insulin analog such as glargine or degludec) in a dose-dependent manner relative to the dexamethasone dose (Level of Evidence I).[60] Notably, postprandial hyperglycemia is often pronounced with glucocorticoid use, and prandial insulin doses frequently require intensification (Level of Evidence II-2).[6,37]

Both NPH insulin and long-acting analogs are effective basal options. Selection should consider glucocorticoid

pharmacodynamics, cost, and individual risk of hypoglycemia (Level of Evidence I).[16,65]

All individuals with type 1 diabetes and those with type 2 diabetes already on multiple daily insulin injections should be managed with BBI regimen to maintain glycemic stability and prevent metabolic decompensation (Level of Evidence I). Importantly, basal insulin should not be withheld in patients with reduced oral intake. In such cases, prandial insulin can be temporarily reduced or suspended, but basal insulin must be maintained to prevent metabolic decompensation and ketogenesis[22,69] (Level of Evidence II-2).

Dose Adjustment and Monitoring

Insulin doses should be tapered proportionally as glucocorticoids are reduced. A 50% reduction in glucocorticoid dose typically requires a ~20 to 25% reduction in insulin dose. Blood glucose should be monitored before meals and at bedtime, with dose adjustments every 2-3 days as needed[5] (Level of Evidence III).

Evidence Summary for Insulin Therapies

Multiple randomized trials and systematic reviews support insulin as the cornerstone of GCIH management (Level of Evidence I).[16,37,68,70] Among hospitalized patients, BBI regimen has been demonstrated superior glycemic control compared to sliding-scale insulin or non-insulin therapies (Level of Evidence I).[22,71,72]

The addition of NPH insulin to basal-bolus regimens has been shown to improve glycemic control when appropriately timed with glucocorticoid administration[16] (Level of Evidence I). NPH insulin and long-acting insulin analogs demonstrate similar efficacy; however, NPH insulin may be associated with a higher risk of hypoglycemia when administered multiple times daily (Level of Evidence II-2).[65,67,73]

Tailoring correctional insulin doses to the pharmacokinetics of the specific glucocorticoid can further enhance glycemic outcomes. Despite heterogeneity across studies, NPH insulin remains a cost-effective and clinically valuable option when its administration is aligned with steroid-induced glycemic peaks (Level of Evidence I).[16]

Importantly, no randomized trials have yet defined the optimal glucose threshold for initiating insulin in GCIH, and current practices are primarily based on extrapolated data from general inpatient diabetes management (Level of Evidence III).

Table 3 provides a practical insulin therapy approach summary according to glucocorticoid regimen.

Special Considerations in Patients with Cancer

Individuals with cancer are at increased risk for hyperglycemia due to multiple factors, including systemic inflammation, catabolic stress, altered nutritional intake, concurrent infections, and the use of glucose-altering therapies. Among these, glucocorticoids remain the most common cause, given their widespread use for symptom control, chemotherapy regimens, and management of immune-related adverse events.[9,10]

In addition to glucocorticoids, several oncologic agents can independently induce hyperglycemia via distinct mechanisms. For example, phosphoinositide 3-kinase (PI3K) inhibitors (eg, alpelisib), protein kinase B (Akt) inhibitors (eg, capivasertib), and mammalian target of rapamycin (mTOR) inhibitors (eg, everolimus) promote insulin resistance by interfering with PI3K-AKT signaling pathway, leading to transient hyperglycemia. Conversely, immune checkpoint inhibitors (ICI), particularly anti-programmed cell death-1 (anti-PD-1, such as pembrolizumab, nivolumab) and anti-programmed death ligand-1 (anti-PDL1, such as atezolizumab, durvalumab), have been associated with new-onset autoimmune diabetes, although this complication is considerably less common.[1,9,35] A summary of these therapies and monitoring recommendations is provided in Table 4.

GCIH may be further exacerbated by chemotherapy-related catabolism, appetite changes, total parenteral nutrition, infections, and co-administration of other hyperglycemia-inducing agents such as calcineurin inhibitors (eg, tacrolimus), or immunotherapies (Level of Evidence II-2).[1,9,31,32,35,74] In patients undergoing chimeric antigen receptor T-cell (CART) therapy, hyperglycemia may result from both high-dose glucocorticoids and cytokine release syndrome, though data on glycemic outcomes in this context remain limited (Level of Evidence III).[36,75,76]

Recent data suggest that up to 70% of individuals receiving chemotherapy experience hyperglycemia, especially when glucocorticoids are part of the regimen. Despite this high prevalence, recognition and management remain suboptimal in clinical practice. A multicenter study involving oncology providers identified barriers such as lack of standardized protocols, limited access to endocrinology consultation or CGM, and uncertainty regarding treatment thresholds in patients without preexisting diabetes.

Table 3
Practical Insulin Therapy Approach for Glucocorticoid-Induced Hyperglycemia (GCIH)

Glucocorticoid regimen	Preferred insulin strategy	Supplemental insulin dose for GCIH	Comments	Level of Evidence[14]
Prednisone (once daily, morning)	NPH insulin (intermediate-acting) should be administered at the same time as prednisone	0.1 units/kg for every 10 mg of prednisone (eg, 40 mg/d → suggested maximum dose: 0.4 units/kg NPH)	Aligns insulin peak with afternoon hyperglycemia; taper NPH dose proportionally as prednisone is reduced	I
Dexamethasone (long-acting) or multiple daily doses	Long-acting basal insulin analogs (eg, glargine, degludec) or split NPH	Glargine: 0.2-0.4 units/kg once daily or NPH: 0.1-0.2 units/kg/d split into 2 doses (eg, 0.05 U/kg between breakfast and late afternoon)	Prefer long-acting insulin for prolonged effect; NPH may help mitigate afternoon glycemic peaks when combined with basal analogs. Prolonged or higher doses of dexamethasone may require intensification of prandial insulin to manage sustained postprandial hyperglycemia	I
All regimens with moderate to severe hyperglycemia	Basal-Bolus Insulin (BBI) regimen (basal + rapid-acting prandial insulin)	0.5-0.6 units/kg/d total insulin dose Split: 50% basal, 50% prandial (divided among meals)	Standard for hospitalized patients; adjust doses every 2-3 d based on glucose patterns	I
Persistent postprandial hyperglycemia	Add or intensify rapid-acting insulin before meals	Start prandial insulin at 0.1 units/kg/meal, adjust by 0.04-0.08 units/kg depending on pre-meal glucose (200-300 or >300 mg/dL)	May be used with correction insulin; basal component may be required if hyperglycemia persists in the afternoon	II-2
Glucocorticoid tapering phase	Reduce insulin doses proportionally	If glucocorticoid dose is reduced by ~50%, reduce insulin by ~20-25%	Frequent monitoring essential to prevent hypoglycemia during dose reductions	III

Table 4
Antineoplastic Agents Associated with Hyperglycemia

Drug class/agent	Example agents	Glycemic effect	Monitoring recommendation
PI3K inhibitors	Alpelisib, Inavolisib	High incidence of hyperglycemia, especially during the first 2 wk.	Baseline fasting/random glucose; monitor weekly for 2 wk, then monthly; consider HbA1c every 3 mo
mTOR inhibitors	Everolimus	Moderate hyperglycemia; variable onset (wk to y)	Fasting/random glucose before treatment and at each visit; consider HbA1c every 3 mo
Akt inhibitors	Capivasertib	Can cause insulin resistance and hyperglycemia	Baseline and periodic glucose monitoring
Immune checkpoint inhibitors, particularly anti-PD-1 and anti-PD-L1	Nivolumab, Pembrolizumab, Atezolizumab Durvalumab	Autoimmune diabetes, often presenting as diabetic ketoacidosis	Baseline glucose and each visit; urgent referral if symptomatic

Abbreviations: Akt = Protein kinase B; HbA1c = Glycated hemoglobin; mTOR = Mammalian target of rapamycin; PD-1 = Programmed cell death protein 1; PD-L1 = Programmed death-ligand 1; PI3K = Phosphatidylinositol 3-kinase.

These factors contribute to delays in diagnosis and treatment of GCIH, and reinforce the need for multidisciplinary collaboration and education to improve outcomes.[32]

GCIH is associated with increased infection risk, delays in chemotherapy, longer hospital stays, and worsened prognosis (Level of Evidence II-2).[1,4,11,12,18,68] Moreover, symptoms such as fatigue, nausea, or polyuria may be misattributed to chemotherapy side effects, delaying recognition and treatment of hyperglycemia (Level of Evidence III).[32]

Insulin is the preferred treatment for moderate to severe GCIH in hospitalized or acutely ill patients with cancer (Level of Evidence III).[35]

In critically ill patients (eg, those requiring vasopressor support, mechanical ventilation, or experiencing hyperglycemic crises), intravenous insulin infusion protocols are preferred (Level of Evidence III).[9]

In non-critically ill inpatients, subcutaneous insulin therapy remains the treatment of choice, preferably using basal-bolus regimens tailored to the glucocorticoid type and schedule (Level of Evidence II-1). For once-daily prednisone or methylprednisolone, NPH insulin may be administered concomitantly, typically in the morning, to match the afternoon glycemic peak. For dexamethasone or multiple daily glucocorticoid doses, long-acting basal analogs (eg, glargine, degludec) are more appropriate (Level of Evidence II-1).[9,35] High-dose or pulse glucocorticoid regimens, commonly used in brain metastases, multiple myeloma, or aggressive lymphomas, can lead to profound and sustained hyperglycemia and may require intensified glucose monitoring and insulin titration strategies (Level of Evidence III).

In patients with impaired oral intake, such as those experiencing mucositis, anorexia, or nausea during chemotherapy, prandial insulin strategies should be re-evaluated to minimize the risk of hypoglycemia. In such scenarios, a basal-plus approach may be temporarily used in place of a full basal-bolus regimen,[22,55,63,77] adjusting basal insulin according to glucocorticoid regimen (e.g., NPH insulin with prednisone, long-acting analogs with dexamethasone), and reserving rapid-acting insulin only for the correction of significant hyperglycemia (Level of Evidence III). This approach may simplify management and reduce hypoglycemia risk in patients unable to maintain regular oral intake.

In outpatient settings with mild hyperglycemia, selected non-insulin agents (eg, metformin, DPP-4 inhibitors) may be cautiously considered (Level of Evidence III).[9,35] GLP-1 receptor agonists and dual GLP-1/GIP receptor agonist should be avoided in cachectic or malnourished patients, as they may suppress appetite and worsen weight loss (Level of Evidence III).[78]

Intensive glucose monitoring is essential, especially during chemotherapy cycles (Level of Evidence II-2), as GCIH is associated with increased risk of infection, poor wound healing, and inferior oncologic outcomes. Given the dynamic nature of cancer treatment, frequent insulin dose adjustments and flexible monitoring strategies are recommended throughout the treatment course.

In individuals with advanced cancer receiving palliative or end-of-life care, glycemic management priorities shift from preventing long-term complications to minimizing symptom burden and improving quality of life. Intensive glycemic control is generally not appropriate in this context. Instead, simplified regimens and more relaxed glucose targets (eg, 100-200 mg/dL; and in selected hospice settings, less stringent targets, eg, <300 mg/dL) may be appropriate, depending on the patient's clinical condition, treatment goals, and preferences. HbA1c targets lose relevance in this setting, and glucose monitoring frequency should be minimized or discontinued altogether when consistent with comfort-focused care. Individualized plans should balance the risks of symptomatic hyperglycemia (eg, dehydration, polyuria, fatigue) and hypoglycemia against treatment burden. Early integration of palliative care teams can help establish appropriate goals and facilitate deprescription of unnecessary antihyperglycemic agents (Level of Evidence III).[1]

Challenges and Unresolved Issues

Despite its clinical relevance, the management of GCIH remains suboptimal in practice. Several barriers hinder effective screening, diagnosis, and treatment, particularly in oncology settings. Table 5 summarizes the main challenges, clinical implications, and potential strategies for improvement.[3,4,6,11,16,18,26,34,36,38,40,44,55,79-81]

Key Challenges Include

- Lack of standardized definitions for GCIH, particularly in individuals without preexisting diabetes;
- Inconsistent recommendations across guidelines, often extrapolated from general diabetes or inpatient hyperglycemia management;
- Scarce clinical trial data specific to GCIH treatment in oncology, especially in individuals receiving chemotherapy, immunotherapy, or high-dose/pulse glucocorticoids;
- Uncertainty regarding glycemic targets, especially for short-term or palliative use;
- Limited availability of practical algorithms tailored to glucocorticoid type, dose, and schedule.

Addressing these gaps will require prospective research, including randomized trials comparing insulin strategies and evaluating outcomes such as infection rates, cancer treatment

Table 5
Key Challenges in Glucocorticoid-Induced Hyperglycemia (GCIH) in Oncology: Clinical Implications and Potential Solutions

Challenge	Clinical implication	Possible solutions
Lack of standardized definitions specific to GCIH[6,11,13,15,29,38,74]	Difficulty diagnosing and comparing outcomes across studies	Develop GCIH-specific diagnostic thresholds, considering postprandial and afternoon glucose patterns. There is currently no consensus on the definition of glucocorticoid-induced hyperglycemia or glucocorticoid-induced diabetes, including in oncology settings. Harmonized criteria are needed to improve comparability across studies and support timely diagnosis and intervention
Heterogeneity in guidelines and lack of practical algorithms[6,13,23,29,38,40,52,76]	Confusion among clinicians and variability in care	Develop glucocorticoid-specific insulin adjustment algorithms (eg, for prednisone vs dexamethasone), incorporating patterns seen in oncology (eg, intermittent vs continuous glucocorticoid use across chemotherapy regimens). Tailored protocols may help standardize care for patients receiving regimens such as R-CHOP, or dexamethasone pulses for multiple myeloma or brain metastases
Under recognition of GCIH in patients without diabetes[6,23,33]	Missed opportunities for early intervention	Implement routine glucose monitoring protocols for all patients receiving glucocorticoids, especially those at higher risk for GCIH (eg, older adults, prediabetes, or high-dose steroid use)
Limited use of CGM and fructosamine in clinical settings[3,4,23,31,75]	Inadequate detection of glycemic variability and acute excursions	Increase access to CGM and use fructosamine in dynamic or short-term glucocorticoid regimens
Lack of RCTs in oncology-specific GCIH settings[13,15,52]	Treatment decisions based on low-certainty evidence	Conduct RCTs comparing insulin and non-insulin strategies in various clinical settings (eg, oncology, palliative care)

Abbreviations: CGM = continuous glucose monitoring; GCIH = glucocorticoid-induced hyperglycemia; R-CHOP = rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone; RCT = randomized controlled trials.

delays, and cancer-related mortality. Emerging technologies such as CGM and clinical decision support systems may help individualize care and improve real-time management.

Future Directions and Perspectives

As understanding of GCIH continues to evolve, future strategies aim to improve both detection and management by integrating novel pharmacologic agents, advanced monitoring technologies, and individualized treatment approaches.[18,32]

Novel Pharmacological Strategies

Emerging pharmacologic interventions seek to mitigate the glycemic side effects of glucocorticoids without compromising their therapeutic efficacy.

- Selective glucocorticoid receptor modulators (SGRMs) have been developed to retain anti-inflammatory effects while minimizing peripheral insulin resistance and gluconeogenesis.[82]
- 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) inhibitors may reduce intracellular activation of glucocorticoids, thereby lowering hepatic glucose production and systemic glycemic burden.[83]
- Liver X receptor (LXR) antagonists are another promising class that modulate transcription of gluconeogenic genes, offering a potential pathway to control glucocorticoid-induced hyperglycemia without altering systemic steroid dosing.[84]

Advancements in Glucose Monitoring Technologies

Technological innovation plays a central role in improving the management of GCIH.

- Continuous glucose monitoring (CGM) provides real-time glycemic data and facilitates detection of circadian fluctuations, particularly the characteristic afternoon and evening peaks associated with morning glucocorticoid administration.[29,34,85]
- Automated insulin delivery (AID) systems, also known as closed-loop or "artificial pancreas" technologies, integrate CGM data with insulin pumps to dynamically adjust insulin dosing. Though primarily studied in type 1 diabetes, preliminary data suggest potential utility in managing GCIH, particularly in hospitalized patients receiving high-dose glucocorticoids[29,34,85]

Personalized Treatment Approaches

Personalized strategies are essential due to wide variability in glucocorticoid pharmacokinetics, patient characteristics, and glycemic response.

- Individualized insulin regimens should consider the timing, potency, and duration of the glucocorticoid, as well as baseline glucose control and comorbidities. Tailoring basal and bolus insulin doses to steroid pharmacodynamics may optimize outcomes and minimize hypoglycemia risk.
- Combination therapies, incorporating insulin with non-insulin agents such as metformin, sulfonylureas, or DPP-4 inhibitors, may be appropriate in select cases, particularly in outpatient or oncology settings. However, further studies are warranted to determine safety and efficacy across different cancer subtypes and treatment regimens.
- Artificial intelligence (AI)-based algorithms may enable more precise risk stratification and treatment selection by integrating clinical, pharmacologic, and even genetic data. Such decision-support systems have potential to improve real-time glycemic management and reduce treatment delays.

Conclusion

GCIH is a common yet often underrecognized complication in individuals with cancer. Its pathophysiology is multifactorial, marked by increased insulin resistance, enhanced hepatic gluconeogenesis, and impaired β-cell function. When unrecognized or inadequately managed, GCIH has been linked to worse clinical outcomes, including higher infection rates, treatment delays, and increased mortality.

Optimal management requires early identification, individualized glucose monitoring, and treatment strategies tailored to glucocorticoid type, dose, and patient-specific glycemic status. While insulin, particularly basal-bolus regimens, remains the cornerstone of therapy for moderate to severe hyperglycemia, non-insulin agents may be appropriate for selected patients with

mild GCIH. The strategic use of NPH insulin aligned with glucocorticoid pharmacokinetics is especially relevant in oncology.

Persistent gaps remain, including the lack of standardized diagnostic thresholds, inconsistent use of CGM, and limited high-quality evidence from randomized trials. Future research should prioritize refining GCIH definitions, validating insulin protocols, and exploring innovative therapies, such as automated insulin delivery systems and selective glucocorticoid receptor modulators. A personalized, evidence-based approach integrating pharmacologic, technological, and clinical variables will be critical to improving outcomes in this high-risk population.

References

1. Hiestand A, Flory J, Chitkara R. Hospitalization as an opportunity to optimize glycemic control in oncology patients. *Curr Diab Rep*. 2019;19(12):149. https://doi.org/10.1007/s11892-019-1283-6.
2. Jeong Y, Han HS, Lee HD, et al. A pilot study evaluating steroid-induced diabetes after antiemetic dexamethasone therapy in chemotherapy-treated cancer patients. *Cancer Res Treat*. 2016;48(4):1429–1437. https://doi.org/10.4143/crt.2015.464.
3. Toyoshima MTK, Cukier P, Souza ABC de, Pereira J, Hoff AO, Nery M. Effects of glucocorticoids on interstitial glucose concentrations in individuals with hematologic cancer and without known diagnosis of diabetes: a pilot study. *Einstein (Sao Paulo)*. 2022;20(1):eAO8031. https://doi.org/10.31744/einstein_journal/2022AO8031.
4. Limbachia V, Nunney I, Page DJ, et al. The effect of different types of oral or intravenous corticosteroids on capillary blood glucose levels in hospitalized inpatients with and without diabetes. *Clin Ther*. 2024;46(2):e59–e63. https://doi.org/10.1016/j.clinthera.2023.11.013.
5. Tamez-Pérez HE. Steroid hyperglycemia: prevalence, early detection and therapeutic recommendations: a narrative review. *World J Diabetes*. 2015;6(8):1073. https://doi.org/10.4239/wjd.v6.i8.1073.
6. Bonaventura A, Montecucco F. Steroid-induced hyperglycemia: an underdiagnosed problem or clinical inertia? A narrative review. *Diabetes Res Clin Pract Elsevier Ireland Ltd*. 2018;139:203–220. https://doi.org/10.1016/j.diabres.2018.03.006.
7. Fong AC, Cheung NWah. The high incidence of steroid-induced hyperglycaemia in hospital. *Diabetes Res Clin Pract*. 2013;99(3):277–280. https://doi.org/10.1016/j.diabres.2012.12.023.
8. Wright J, Nielsen T, Burns S, et al. Management of glucocorticoid-induced hyperglycemia in cancer patients: a feasibility study. *Clin Nurs Res*. 2025;34(1):3–11. https://doi.org/10.1177/10547738241291272.
9. Healy SJ, Dungan KM. Hyperglycemia in patients with hematologic malignancies. *Curr Diab Rep*. 2015;15(3):8. https://doi.org/10.1007/s11892-015-0581-x.
10. Lossignol D. A little help from steroids in oncology. *J Transl Int Med*. 2016;4(1):52–54. https://doi.org/10.1515/jtim-2016-0011.
11. Aberer F, Hochfellner DA, Sourij H, Mader JK. A practical guide for the management of steroid induced hyperglycaemia in the hospital. *J Clin Med*. 2021;10(10):2154. https://doi.org/10.3390/jcm10102154.
12. Joharatnam-Hogan N, Chambers P, Dhatariya K, Board R. A guideline for the outpatient management of glycaemic control in people with cancer. *Diabetic Med*. 2022;39(1):e14636. https://doi.org/10.1111/dme.14636.
13. Silva Júnior WS, Gabbay MAL, Lamounier RN, Calliari LE, Bertoluci MC. The 2021–2022 position of Brazilian diabetes society on insulin therapy in type 1 diabetes: an evidence-based guideline to clinical practice. *Diabetol Metab Syndr*. 2022;14(1):189. https://doi.org/10.1186/s13098-022-00949-z.
14. Liu D, Ahmet A, Ward L, et al. A practical guide to the monitoring and management of the complications of systemic corticosteroid therapy. *Allergy Asthma Clin Immunol*. 2013;9(1):30. https://doi.org/10.1186/1710-1492-9-30.
15. Faggiano A, Mazzilli R, Natalicchio A, et al. Corticosteroids in oncology: use, overuse, indications, contraindications. An Italian Association of Medical Oncology (AIOM)/Italian Association of Medical Diabetologists (AMD)/Italian Society of Endocrinology (SIE)/Italian Society of Pharmacology (SIF) multidisciplinary consensus position paper. *Crit Rev Oncol Hematol*. 2022;180:103826. https://doi.org/10.1016/j.critrevonc.2022.103826.
16. Brooks D, Schulman-Rosenbaum R, Griff M, Lester J, Low Wang CC. Glucocorticoid-induced hyperglycemia including dexamethasone-associated hyperglycemia in COVID-19 infection: a systematic review. *Endocr Pract*. 2022;28(11):1166–1177. https://doi.org/10.1016/j.eprac.2022.07.014.
17. Canadian Task Force. The periodic health examination. Canadian task force on the periodic health examination. *Can Med Assoc J*. 1979;121(9):1193–1254.
18. Li JX, Cummins CL. Fresh insights into glucocorticoid-induced diabetes mellitus and new therapeutic directions. *Nat Rev Endocrinol*. 2022;18(9):540–557. https://doi.org/10.1038/s41574-022-00683-6.
19. Beaupere C, Liboz A, Fève B, Blondeau B, Guillemain G. Molecular mechanisms of glucocorticoid-induced insulin resistance. *Int J Mol Sci*. 2021;22(2):1–30. https://doi.org/10.3390/ijms22020623.
20. Kwon S, Hermayer KL. Glucocorticoid-induced hyperglycemia. *Am J Med Sci*. 2013;345(4):274–277. https://doi.org/10.1097/MAJ.0b013e31828a6a01.
21. Kuo T, McQueen A, Chen TC, Wang JC. Regulation of glucose homeostasis by glucocorticoids. In: Wang JC, Harris C, eds. *Glucocorticoid Signaling From Molecules to Mice to Man*. Springer; 2015:99–126. https://doi.org/10.1007/978-1-4939-2895-8_5.
22. Bajaj M, McCoy RG, Balapattabi K, et al. 16. Diabetes care in the hospital: standards of care in Diabetes—2026. *Diabetes Care*. 2026;49(Supplement_1):S339–S355. https://doi.org/10.2337/dc26-S016.
23. Hu W, Jiang C, Kim M, et al. Individual-specific functional epigenomics reveals genetic determinants of adverse metabolic effects of glucocorticoids. *Cell Metab*. 2021;33(8):1592–1609.e7. https://doi.org/10.1016/j.cmet.2021.06.004.
24. Katsuyama T, Sada KE, Namba S, et al. Risk factors for the development of glucocorticoid-induced diabetes mellitus. *Diabetes Res Clin Pract*. 2015;108(2):273–279. https://doi.org/10.1016/j.diabres.2015.02.010.
25. Deutsch AJ, Schroeder PH, Mandla R, et al. Type 2 diabetes polygenic score predicts the risk of glucocorticoid-induced hyperglycemia in patients without diabetes. *Diabetes Care*. 2023;46(8):1541–1545. https://doi.org/10.2337/dc23-0353.
26. Suh S, Park MK. Glucocorticoid-induced diabetes mellitus: an important but overlooked problem. *Endocrinol Metab*. 2017;32(2):180–189. https://doi.org/10.3803/EnM.2017.32.2.180.
27. Ambery P, Adamsson Eryd S, Jenkins-Jones S, et al. The absolute risk of incident type 2 diabetes following exposure to systemic corticosteroids in selected steroid-related and phenotypic groups. *Diabetes Obes Metab*. 2022;24(11):2222–2231. https://doi.org/10.1111/dom.14808.
28. Bajaj M, McCoy RG, Balapattabi K, et al. 2. Diagnosis and classification of diabetes: standards of care in Diabetes—2026. *Diabetes Care*. 2026;49(Supplement_1):S27–S49. https://doi.org/10.2337/dc26-S002.
29. Lyall MJ, Thethy I, Steven L, et al. Diurnal profile of interstitial glucose following dexamethasone prophylaxis for chemotherapy treatment of gynaecological cancer. *Diabetic Med*. 2018;35(11):1508–1514. https://doi.org/10.1111/dme.13770.
30. He X, Duan G, Lu J, et al. Continuous glucose monitoring profile in COVID-19 patients with and without diabetes receiving methylprednisolone. *Endocrine*. 2024;86(2):556–563. https://doi.org/10.1007/s12020-024-03841-0.
31. Hammer M, Storey S, Hershey DS, et al. Hyperglycemia and cancer: a state-of-the-science review. *Oncol Nurs Forum*. 2019;46(4):459–472. https://doi.org/10.1188/19.ONF.459-472.
32. Salgado TM, Birari PB, Alshahawey M, et al. Optimal hyperglycemia thresholds in patients undergoing chemotherapy: a cross sectional study of oncologists' practices. *Support Care Cancer*. 2024;32(8):563. https://doi.org/10.1007/s00520-024-08756-0.
33. Barker HL, Morrison D, Llano A, Sainsbury CAR, Jones GC. Practical guide to glucocorticoid induced hyperglycaemia and diabetes. *Diabetes Ther*. 2023;14(5):937–945. https://doi.org/10.1007/s13300-023-01393-6.
34. Agarwal S, Demidowich AP, Soliman D, Umpierrez GE, Galindo RJ. Wearable diabetes technology for hospitalized people with diabetes and end-stage kidney disease, peripartum state, and steroid use. *J Diabetes Sci Technol*. Published online September 3, 2025. https://doi.org/10.1177/19322968251364276.
35. Kotwal A, Cheung YMM, Cromwell G, et al. Patient-centered diabetes care of cancer patients. *Curr Diab Rep*. 2021;21(12):62. https://doi.org/10.1007/s11892-021-01435-y.
36. Tienstra M, de Boer JW, van Doesum JA, et al. High frequency of severe hyperglycemia observed during intensive hematologic care: a prospective study using continuous glucose monitoring. *Endocr Pract*. 2024;30(12):1141–1148. https://doi.org/10.1016/j.eprac.2024.09.013.
37. Cho JH, Suh S. Glucocorticoid-induced hyperglycemia: a neglected problem. *Endocrinol Metab*. 2024;39(2):222–238. https://doi.org/10.3803/EnM.2024.1951.
38. Wallace MD, Metzger NL. Optimizing the treatment of steroid-induced hyperglycemia. *Ann Pharmacother*. 2018;52(1):86–90. https://doi.org/10.1177/1060028017728297.
39. Habib G, Dar-Esaif Y, Bishara H, et al. The impact of corticosteroid treatment on hemoglobin A1C levels among patients with type-2 diabetes with chronic obstructive pulmonary disease exacerbation. *Respir Med*. 2014;108(11):1641–1646. https://doi.org/10.1016/j.rmed.2014.08.006.
40. Radhakutty A, Burt MG. Management of endocrine disease: critical review of the evidence underlying management of glucocorticoid-induced hyperglycaemia. *Eur J Endocrinol*. 2018;179(4):R207–R218. https://doi.org/10.1530/EJE-18-0315.
41. Toyoshima MTK, Cukier P, Damascena AS, et al. Fructosamine and glycated hemoglobin as biomarkers of glycemic control in people with type 2 diabetes mellitus and cancer (GlicoOnco study). *Clinics*. 2023;78:100240. https://doi.org/10.1016/j.clinsp.2023.100240.
42. Pernicova I, Kelly S, Ajodha S, et al. Metformin to reduce metabolic complications and inflammation in patients on systemic glucocorticoid therapy: a randomised, double-blind, placebo-controlled, proof-of-concept, phase 2 trial. *Lancet Diabetes Endocrinol*. 2020;8(4):278–291. https://doi.org/10.1016/S2213-8587(20)30021-8.
43. Roberts A, James J, Dhatariya K, et al. Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group. *Diabetic Med*. 2018;35(8):1011–1017. https://doi.org/10.1111/dme.13675.
44. Elena C, Chiara M, Angelica B, et al. Hyperglycemia and diabetes induced by glucocorticoids in nondiabetic and diabetic patients: revision of literature and

personal considerations. *Curr Pharm Biotechnol*. 2019;19(15):1210–1220. https://doi.org/10.2174/1389201020666190102145305.
45. Hoogwerf B, Danese RD. Drug selection and the management of corticosteroid-related diabetes mellitus. *Rheum Dis Clin North America*. 1999;25(3):489–505. https://doi.org/10.1016/S0889-857X(05)70083-1.
46. Willi SM, Kennedy A, Brant BP, Wallace P, Rogers NL, Garvey WT. Effective use of thiazolidinediones for the treatment of glucocorticoid-induced diabetes. *Diabetes Res Clin Pract*. 2002;58(2):87–96. https://doi.org/10.1016/S0168-8227(02)00127-4.
47. Luther P, Baldwin D. Pioglitazone in the management of diabetes mellitus after transplantation. *Am J Transplant*. 2004;4(12):2135–2138. https://doi.org/10.1111/j.1600-6143.2004.00613.x.
48. He J, Xu C, Kuang J, et al. Thiazolidinediones attenuate lipolysis and ameliorate dexamethasone-induced insulin resistance. *Metabolism*. 2015;64(7):826–836. https://doi.org/10.1016/j.metabol.2015.02.005.
49. Yata Y, Hosojima M, Kabasawa H, et al. The assessment of the efficacy of dipeptidyl Peptidase-4 inhibitors in patients with glucocorticoid-induced diabetes by continuous glucose monitoring. *Intern Med*. 2017;56(19):2555–2562. https://doi.org/10.2169/internalmedicine.8296-16.
50. Ohashi N, Tsuji N, Naito Y, et al. Alogliptin improves steroid-induced hyperglycemia in treatment-naïve Japanese patients with chronic kidney disease by decrease of plasma glucagon levels. *Med Sci Monitor*. 2014;20:587–593. https://doi.org/10.12659/MSM.889872.
51. Katsuyama H, Sako A, Adachi H, Hamasaki H, Yanai H. Effects of 6-Month sitagliptin treatment on metabolic parameters in diabetic patients taking oral glucocorticoids: a retrospective cohort study. *J Clin Med Res*. 2015;7(6):479–484. https://doi.org/10.14740/jocmr2153w.
52. van Genugten RE, van Raalte DH, Muskiet MH, et al. Does dipeptidyl peptidase-4 inhibition prevent the diabetogenic effects of glucocorticoids in men with the metabolic syndrome? A randomized controlled trial. *Eur J Endocrinol*. 2014;170(3):429–439. https://doi.org/10.1530/EJE-13-0610.
53. van Raalte DH, van Genugten RE, Linssen MML, Ouwens DM, Diamant M. Glucagon-like Peptide-1 receptor agonist treatment prevents glucocorticoid-induced glucose intolerance and islet-cell dysfunction in humans. *Diabetes Care*. 2011;34(2):412–417. https://doi.org/10.2337/dc10-1677.
54. Gerards MC, Venema GE, Patberg KW, et al. Dapagliflozin for prednisone-induced hyperglycaemia in acute exacerbation of chronic obstructive pulmonary disease. *Diabetes Obes Metab*. 2018;20(5):1306–1310. https://doi.org/10.1111/dom.13209.
55. Korytkowski MT, Muniyappa R, Antinori-Lent K, et al. Management of hyperglycemia in hospitalized adult patients in non-critical care settings: an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 2022;107(8):2101–2128. https://doi.org/10.1210/clinem/dgac278.
56. Honarmand K, Sirimaturos M, Hirshberg EL, et al. Society of Critical Care Medicine Guidelines on glycemic control for critically ill children and adults 2024: executive summary. *Crit Care Med*. 2024;52(4):649–655. https://doi.org/10.1097/CCM.0000000000006173.
57. Umpierrez GE, Davis GM, ElSayed NA, et al. Hyperglycaemic crises in adults with diabetes: a consensus report. *Diabetologia*. 2024;67(8):1455–1479. https://doi.org/10.1007/s00125-024-06183-8.
58. Lakhani OJ, Kumar S, Tripathi S, Desai M, Seth C. Comparison of two protocols in the management of glucocorticoid-induced hyperglycemia among hospitalized patients. *Indian J Endocrinol Metab*. 2017;21(6):836–844. https://doi.org/10.4103/ijem.IJEM_226_17.
59. Khowaja A, Alkhaddo JB, Rana Z, Fish L. Glycemic control in hospitalized patients with diabetes receiving corticosteroids using a neutral protamine hagedorn insulin protocol: a randomized clinical trial. *Diabetes Ther*. 2018;9(4):1647–1655. https://doi.org/10.6084/m9.figshare.6633050.
60. Grommesh B, Lausch MJ, Vannelli AJ, et al. Hospital insulin protocol aims for glucose control in glucocorticoid-induced hyperglycemia. *Endocr Pract*. 2016;22(2):180–189. https://doi.org/10.4158/EP15818.OR.
61. Seggelke SA, Gibbs J, Draznin B. Pilot study of using neutral protamine hagedorn insulin to counteract the effect of methylprednisolone in hospitalized patients with diabetes. *J Hosp Med*. 2011;6(3):175–176. https://doi.org/10.1002/jhm.874.
62. Gerards MC, De Maar J, Steenbruggen TG, Hoekstra JBL, Vriesendorp TM, Gerdes VEA. Add-on treatment with intermediate-acting insulin versus sliding scale insulin for patients with type 2 diabetes or insulin resistance during cyclic glucocorticoid-containing antineoplastic chemotherapy-a randomized cross-over study. *Diabetes Obes Metab*. 2016;18(10):1041–1044.
63. Marino EC, Momesso D, Toyoshima MTK, et al. Screening and management of hospital hyperglycemia in non-critical patients: a position statement from the Brazilian Diabetes Society (SBD). *Diabetol Metab Syndr*. 2025;17(1):54. https://doi.org/10.1186/s13098-025-01585-z.
64. Clore JN, Thurby-Hay L. Glucocorticoid-induced hyperglycemia. *Endocr Pract*. 2009;15(5):469–474. https://doi.org/10.4158/EP08331.RAR.
65. Seisa MO, Saadi S, Nayfeh T, et al. A systematic review supporting the Endocrine Society Clinical Practice guideline for the management of hyperglycemia in adults hospitalized for noncritical illness or undergoing elective surgical procedures. *J Clin Endocrinol Metab*. 2022;107(8):2139–2147. https://doi.org/10.1210/clinem/dgac277.
66. Agudo-Tabuenca A, Gimeno-Orna JA, Sáenz-Abad D. Assessment of the efficacy and safety of a protocol to manage glucocorticoid-induced hyperglycemia in diabetic patients during hospital stay. *Endocrinología, Diabetes y Nutrición (English Ed)*. 2019;66(6):353–360. https://doi.org/10.1016/j.endien.2019.01.007.
67. Ruiz de Adana M, Colomo N, Maldonado-Araque C, et al. Randomized clinical trial of the efficacy and safety of insulin glargine vs. NPH insulin as basal insulin for the treatment of glucocorticoid induced hyperglycemia using continuous glucose monitoring in hospitalized patients with type 2 diabetes and res. *Diabetes Res Clin Pract*. 2015;110(2):158–165.
68. Gosmanov AR, Goorha S, Stelts S, Peng L, Umpierrez GE. Management of hyperglycemia in diabetic patients with hematologic malignancies during dexamethasone therapy. *Endocr Pract*. 2013;19(2):231–235. https://doi.org/10.4158/EP12256.OR.
69. Umpierrez GE, Hellman R, Korytkowski MT, et al. Management of hyperglycemia in hospitalized patients in non-critical care setting: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97(1):16–38. https://doi.org/10.1210/jc.2011-2098.
70. Struja T, Nitritz N, Alexander I, et al. Treatment of glucocorticoid- induced hyperglycemia in hospitalized patients - a systematic review and meta-analysis. *Clin Diabetes Endocrinol*. 2024;10(1):8. https://doi.org/10.1186/s40842-023-00158-1.
71. Umpierrez GE, Smiley D, Jacobs S, et al. Randomized study of basal-bolus insulin therapy in the inpatient management of patients with type 2 diabetes undergoing general surgery (RABBIT 2 surgery). *Diabetes Care*. 2011;34(2):256–261. https://doi.org/10.2337/dc10-1407.
72. Umpierrez GE, Smiley D, Zisman A, et al. Randomized study of basal-bolus insulin therapy in the inpatient management of patients with type 2 diabetes (RABBIT 2 trial). *Diabetes Care*. 2007;30(9):2181–2186. https://doi.org/10.2337/dc07-0295.
73. Radhakutty A, Stranks JL, Mangelsdorf BL, et al. Treatment of prednisolone-induced hyperglycaemia in hospitalized patients: insights from a randomized, controlled study. *Diabetes Obes Metab*. 2017;19(4):571–578. https://doi.org/10.1111/dom.12859.
74. Brady VJ, Grimes D, Armstrong T, LoBiondo-Wood G. Management of steroid-induced hyperglycemia in hospitalized patients with cancer: a review. *Oncol Nurs Forum*. 2014;41(6):E355–E365. https://doi.org/10.1188/14.ONF.E355-E365.
75. Greenbaum U, Kebriaei P, Srour SA, et al. Chimeric antigen receptor T-cell therapy toxicities. *Br J Clin Pharmacol*. 2021;87(6):2414–2424. https://doi.org/10.1111/bcp.14403.
76. Shimabukuro-Vornhagen A, Böll B, Schellongowski P, et al. Critical care management of chimeric antigen receptor T-cell therapy recipients. *CA Cancer J Clin*. 2022;72(1):78–93. https://doi.org/10.3322/caac.21702.
77. Umpierrez GE, Smiley D, Hermayer K, et al. Randomized study comparing a basal-bolus with a basal plus correction insulin regimen for the hospital management of medical and surgical patients with type 2 diabetes: basal plus trial. *Diabetes Care*. 2013;36(8):2169–2174. https://doi.org/10.2337/dc12-1988.
78. Miousse IR. GLP-1 receptor agonists in the context of cancer: the road ahead. *Am J Physiol-Cell Physiol*. 2025;328(6):C1822–C1828. https://doi.org/10.1152/ajpcell.00245.2025.
79. Hwang JL, Weiss RE. Steroid-induced diabetes: a clinical and molecular approach to understanding and treatment. *Diabetes Metab Res Rev*. 2014;30(2):96–102. https://doi.org/10.1002/dmrr.2486.
80. Burt MG, Roberts GW, Aguilar-Loza NR, Frith P, Stranks SN. Continuous monitoring of circadian glycemic patterns in patients receiving prednisolone for COPD. *J Clin Endocrinol Metab*. 2011;96(6):1789–1796. https://doi.org/10.1210/jc.2010-2729.
81. Tatalovic M, Lehmann R, Cheetham M, Nowak A, Battegay E, Rampini SK. Management of hyperglycaemia in persons with non-insulin-dependent type 2 diabetes mellitus who are started on systemic glucocorticoid therapy: a systematic review. *BMJ Open*. 2019;9(5):e028914. https://doi.org/10.1136/bmjopen-2019-028914.
82. Ambery P, Zajac G, Almquist J, et al. The effect of AZD9567 *vs* . Prednisolone on glycaemic control in patients with type 2 diabetes mellitus: results from a phase 2a clinical trial. *Br J Clin Pharmacol*. 2024;90(8):1921–1931. https://doi.org/10.1111/bcp.16082.
83. Gregory S, Hill D, Grey B, et al. 11β-hydroxysteroid dehydrogenase type 1 inhibitor use in human disease-a systematic review and narrative synthesis. *Metabolism*. 2020;108:154246. https://doi.org/10.1016/j.metabol.2020.154246.
84. Patel R, Magomedova L, Tsai R, Angers S, Orellana A, Cummins CL. Separating the anti-inflammatory and diabetogenic effects of glucocorticoids through LXRβ antagonism. *Endocrinology*. 2017;158(4):1034–1047. https://doi.org/10.1210/en.2017-00094.
85. Spanakis EK, Urrutia A, Galindo RJ, et al. Continuous glucose monitoring–guided insulin administration in hospitalized patients with diabetes: a randomized clinical trial. *Diabetes Care*. 2022;45(10):2369–2375. https://doi.org/10.2337/dc22-0716.