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Research Article

Insulin Management in the Inpatient Setting: A Comprehensive Review

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Abstract

Effective inpatient insulin management is a critical component of hospital medicine, aiming to maintain glycemic control, reduce complications, and optimize patient outcomes. Hyperglycemia, defined as a blood glucose greater than 140 mg/dL, is reported in 22–46% of non-critically ill hospitalized patients and is independently associated with increased mortality, infection, prolonged length of stay, and readmission. This comprehensive review synthesizes current evidence-based strategies and updated guideline recommendations — including the 2025 American Diabetes Association Standards of Care and the 2022 Endocrine Society Clinical Practice Guideline — for managing insulin across the inpatient spectrum. The review outlines insulin pharmacokinetics, dosing methodologies including basal-bolus regimens and correction-scale protocols, and specific considerations for special populations such as critically ill, perioperative, glucocorticoid-treated, and total parenteral nutrition (TPN) patients. It addresses the updated consensus on diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) management, the impact of concurrent non-insulin medications including emerging data on SGLT2 inhibitors and GLP-1 receptor agonists in the inpatient setting, and practical protocols for transitioning between intravenous and subcutaneous insulin. The paper also addresses emerging technologies, including continuous glucose monitoring (CGM) in the hospital, and the importance of structured discharge planning for glycemic continuity. The overarching goal is to provide hospital-based clinicians with a concise yet comprehensive reference to guide safe and effective inpatient insulin therapy.

Keywords: Basal bolus, Blood glucose/therapy, Diabetes mellitus, Hyperglycemia, Inpatients, Insulin, Insulin therapy, Continuous glucose monitoring, Perioperative management, Diabetic ketoacidosis, Steroid induced hyperglycemia, ICU glucose management, Insulin transition protocols, Stress hyperglycemia, ADA diabetes guidelines

Abbreviations

TPN: Total Parenteral Nutrition
ADA: American Diabetes Association
HbA1c : Glycated Hemoglobin
NPH: Neutral Protamine Hagedorn insulin
NPO: Nil Per Os ("Nothing by Mouth")
TDD: Total Daily Dose
CKD: Chronic Kidney Disease
SSI: Sliding Scale Insulin
ICU: Intensive Care Unit
IV: Intravenous
SGLT2: Sodium-Glucose Cotransporter 2
DPP-4: Dipeptidyl Peptidase-4
DKA: Diabetic Ketoacidosis
HHS: Hyperglycemic Hyperosmolar State
GLP-1: Glucagon-Like Peptide-1
GLP-1 RA: GLP-1 Receptor Agonist
ACE: Angiotensin-Converting Enzyme
ARB: Angiotensin Receptor Blocker
CGM: Continuous Glucose Monitoring
POC: Point-of-Care
PTDM : Post-Transplant Diabetes Mellitus
BG: Blood Glucose
CABG: Coronary Artery Bypass Grafting

1. Introduction

Glycemic dysregulation is among the most prevalent and consequential metabolic disturbances encountered in the hospital setting. Patients with diabetes mellitus carry a 3-4-fold greater risk of hospitalization compared to those without diabetes, and in 2020, there were over 7.86 million hospital discharges among U.S. adults with diabetes listed as a diagnosis. Hyperglycemia defined as blood glucose exceeding 140 mg/dL is reported in 22–46% of non-critically ill hospitalized patients, regardless of prior diabetes diagnosis [1].

Both hyperglycemia and hypoglycemia are independently associated with adverse clinical outcomes. Persistent hyperglycemia exacerbates microvascular complications (retinopathy, nephropathy, neuropathy) and macrovascular disease (myocardial infarction, stroke, peripheral arterial disease), increases susceptibility to nosocomial infection, and impairs wound healing. Conversely, iatrogenic hypoglycemia can precipitate life-threatening arrhythmias, seizures, altered mental status, and long-term neurological sequelae [2]. Glycemic variability fluctuations between hyperglycemia and hypoglycemia has emerged as an independent predictor of adverse outcomes and is now increasingly targeted in institutional glycemic protocols [3].

Current clinical guidelines from the American Diabetes Association (ADA) and the Endocrine Society emphasize early recognition of abnormal glucose values, individualized glycemic targets (generally 140–180 mg/dL for critically ill patients and 100–180 mg/dL for non-critically ill patients when achieved without significant hypoglycemia), and proactive rather than reactive insulin strategies [4,5]. Routine assessment of hemoglobin A1c (HbA1c) at admission if not available from the prior 3 months provides valuable insight into pre-hospital glycemic control and guides both inpatient management and outpatient follow-up planning [6]. This review synthesizes the most current evidence and guideline recommendations to support safe, individualized inpatient insulin therapy.

2. Types of Insulin and Their Uses

In the inpatient setting, insulin formulations are broadly classified into long-acting, intermediate-acting, short-acting, and rapid-acting types. An understanding of pharmacokinetics is essential for matching insulin action to each patient's physiologic and pathophysiologic state.

Long-acting insulin (e.g., glargine U-100/U-300, detemir, degludec) provides basal coverage for approximately 24 hours (degludec up to 42 hours) with minimal peak activity, making it suitable for once-daily administration to maintain steady-state insulin levels. Pharmacokinetic properties differ meaningfully across analogs: detemir requires twice-daily dosing in many patients and demonstrates a more pronounced peak than glargine, while degludec offers the flattest profile and longest duration. These differences become clinically relevant when selecting agents for patients with renal impairment or variable nutritional intake [7].

Intermediate-acting insulin (NPH/Neutral Protamine Hagedorn) has a shorter duration of 10–12 hours and a distinct peak of activity 6–10 hours post-injection. Its kinetics make it particularly advantageous for steroid-induced hyperglycemia when once-daily morning glucocorticoids are administered, because NPH's peak activity aligns with the postprandial hyperglycemia associated with morning steroid dosing.

Short-acting insulin (regular human insulin) has onset within 30 minutes, peaks at 2–3 hours, and lasts approximately 6 hours. Its slower onset makes it less suitable for mealtime coverage in most inpatients but it remains the preferred agent for continuous intravenous infusion given well-characterized stability in IV solutions and established dosing algorithms.

Rapid-acting insulin analogs (aspart, lispro, glulisine) peak within 1 hour and last 3–5 hours, closely mimicking physiologic mealtime insulin release. These agents are the preferred choice for prandial and correction dosing in the inpatient setting due to their predictable onset and reduced risk of late hypoglycemia compared to regular insulin. Premixed formulations are generally avoided in hospitalized patients because of inflexibility in adjusting the basal-to-bolus ratio in response to changing clinical conditions [8].

3. Insulin Regimens in the Hospital

3.1. Admission Glycemic Assessment

All hospitalized patients with known diabetes, stress hyperglycemia, or unexplained hyperglycemia (random plasma glucose ≥140 mg/dL) should undergo a comprehensive glycemic assessment at the time of admission. Measurement of HbA1c if not performed in the prior 3 months is recommended by both the ADA 2025 Standards of Care and the Endocrine Society 2022 guideline to distinguish chronic from acute dysglycemia and to inform therapy selection and intensity [4,5]. In non-critically ill patients, point-of-care glucose monitoring is recommended before meals and at bedtime; for patients who are nil per os (NPO), monitoring every 4-6 hours are standard [6]. Institutions are encouraged to implement validated written or computerized provider order entry sets for dysglycemia management that allow for personalized target ranges and patient- specific insulin dosing.

3.2. Determining Total Daily Dose (TDD)

Accurate estimation of the TDD is the foundation of safe inpatient insulin prescribing. Four methods are commonly employed:

- Home regimen method: Sum the patient's total basal and bolus doses over 24 hours. Adjust to 75% of home TDD if the patient is NPO or at elevated hypoglycemia risk; use 100% if the patient is persistently hyperglycemic or acutely ill with increased insulin resistance [8].
- Weight-based method: 0.25–0.3 units/kg/day for insulin-sensitive patients (elderly, chronic kidney disease); 0.4 units/kg/day for average insulin sensitivity (most patients with type 1 diabetes); 0.5–1.0 units/kg/day for insulin-resistant patients (type 2 diabetes, obesity, active infection, corticosteroid use).
- Insulin infusion rate method: Multiply the steady 4-hour IV infusion rate by 6 to estimate the 24-hour requirement.
- Correction-dose method: Total correction insulin administered over the prior 24 hours may be used as an empirical TDD estimate when other data are unavailable.

3.3. Basal-Bolus Insulin Therapy

The basal-bolus regimen is the preferred inpatient insulin strategy for non-critically ill patients with adequate nutritional intake, as recommended by the ADA 2025 Standards of Care (Evidence Level A) and the Endocrine Society [4,5]. Approximately 50% of TDD is administered as basal insulin (e.g., glargine once daily, detemir twice daily), with the remaining 50% divided into prandial bolus doses of rapid-acting insulin (aspart, lispro, or glulisine) given immediately before meals. This approach more closely mimics physiologic insulin secretion, maintaining stable interstitial glucose levels between meals while providing proportionate coverage for carbohydrate intake.

Pre-meal bolus doses are calculated based on the anticipated carbohydrate load and supplemented by a correction factor (also called a sensitivity factor) for preprandial hyperglycemia. Correction scales are individualized based on the patient's TDD, stratified as low (< 40 units/day), moderate (40–80 units/day), or high (> 80 units/day), and applied consistently at each monitoring interval. The seminal RABBIT 2 trial by Umpierrez et al. (2007) demonstrated that basal-bolus therapy significantly improved glycemic control compared to sliding scale insulin alone in hospitalized patients with type 2 diabetes [9].

It is important to note that emerging real-world evidence introduces nuance into the superiority of basal-bolus therapy. A 2024 community hospital study found that a correctional insulin-only regimen achieved greater euglycemic days compared to a full basal-bolus insulin regimen in a subset of noncritically ill type 2 diabetes patients, underscoring the need for individualization and ongoing reassessment rather than a one-size-fits-all approach [10]. A large retrospective analysis of 4,558 noncritically ill patients across three insulin strategies (basal-bolus, sliding scale, and basal-only) found that while basal-only therapy was associated with fewer hyperglycemic days and lower mean glucose compared to sliding scale, no significant differences in mortality, length of stay, or readmission were demonstrated across strategies, suggesting that careful patient selection and dose titration may be as critical as regimen choice [3]. These findings highlight the importance of ongoing daily dose adjustments and individualized target setting.

3.4. Sliding Scale Insulin (SSI)

Sliding scale insulin using reactive rapid-acting insulin doses based solely on point-of-care glucose readings is no longer recommended as monotherapy for inpatient glycemic management [11]. The ADA 2025 Standards and the AACE/ADA consensus statement both emphasize that SSI is reactive rather than preventive, leading to greater glycemic variability and increased risk of both hypoglycemia and rebound hyperglycemia [4,12]. Its primary utility is as an adjunct to scheduled basal-bolus therapy, functioning as a correction mechanism for unanticipated hyperglycemia not covered by the prandial component [12]. Despite its limitations, SSI remains prevalent in practice due to ease of implementation and familiarity among nursing and medical staff; educational efforts targeting providers remain an important institutional priority.

4. Special Populations

4.1. Critically Ill Patients – ICU

Critically ill patients experience frequent and clinically significant glucose fluctuations attributable to stress-induced hyperglycemia (driven by catecholamine and cortisol surges), variable and often interrupted enteral nutrition, altered insulin pharmacokinetics, and concurrent glucogenic medications. The ADA 2025 Standards recommend initiating insulin therapy when blood glucose persistently exceeds 180 mg/dL on two occasions within 24 hours, with a glycemic target of 140–180 mg/dL for most ICU patients (Evidence Level A) [4].This target is supported by the landmark NICE-SUGAR trial, which demonstrated that intensive glucose control (81–108 mg/dL) increased 90-day mortality compared to conventional management in critically ill adults, primarily driven by increased hypoglycemia events [13].

Continuous IV insulin infusion using regular insulin is the recommended method in this setting, allowing for rapid titration based on hourly glucose monitoring. Studies suggest a meaningful underutilization of IV insulin in eligible ICU patients: one investigation found that only 13% of septic ICU patients meeting criteria for IV insulin were actually started on it [14]. More stringent individualized glycemic targets (100–140 mg/dL) may be appropriate in selected patients such as post-CABG or those without pre-existing diabetes if achievable without significant hypoglycemia [4].

4.2. Transitioning from IV to Subcutaneous Insulin

The transition from IV to subcutaneous insulin is a high-risk procedural step that requires careful planning to prevent rebound hyperglycemia or hypoglycemia. A key principle is ensuring sufficient overlap between the IV infusion and the first subcutaneous basal insulin dose to account for the delayed onset of subcutaneous absorption. Current evidence, including a retrospective study of critically ill adults in burn, medical, and surgical/trauma ICUs, suggests that transitioning to 50–70% of the prior 24-hour IV insulin requirement as basal subcutaneous insulin achieves the highest rate of blood glucose concentrations within target range (70–150 mg/dL) in the 48 hours post-transition [15]. Pharmacist-managed transition protocols implementing this 50–70% range have demonstrated high adherence rates and appropriate timing of the IV-to-subcutaneous overlap in clinical practice [15]. Transition should be considered once the patient is hemodynamically stable, no longer requiring vasoactive support, and tolerating or resuming enteral nutrition.

4.3. Perioperative Patients

Poor perioperative glycemic control is independently associated with higher surgical site infection rates, impaired wound healing, fluid and electrolyte disturbances, and cardiovascular complications [16]. Most surgical teams

target an HbA1c < 8.5% prior to elective procedures, with some institutions preferring < 7.5% for high-risk surgeries. Preoperatively, all oral antihyperglycemic agents should be held on the day of surgery. SGLT2 inhibitors require discontinuation at least 72 hours prior to any surgical procedure given the risk of euglycemic ketoacidosis, which can occur in the setting of surgical fasting and physiologic stress even in patients with type 2 diabetes [4].

DPP-4 inhibitors (e.g., sitagliptin) may be continued in selected low-risk surgical patients with mild to moderate hyperglycemia. Evidence from the SITA-HOSPITAL trial demonstrated that sitagliptin combined with a basal-plus insulin regimen achieved glycemic control comparable to a full basal-bolus regimen with a potentially lower risk of hypoglycemia, supporting its use in carefully selected non-critically ill patients [17]. For short surgeries (< 4 hours), subcutaneous correctional rapid-acting insulin every 2 hours with frequent glucose checks is appropriate. For longer procedures or hemodynamically unstable cases, continuous IV regular insulin infusion is preferred with glucose monitoring every 1–2 hours. Postoperatively, patients tolerating oral intake may resume their preoperative regimens; those remaining hospitalized should receive standard inpatient basal-bolus therapy with daily dose adjustments.

4.4. Glucocorticoid-Treated Patients

Glucocorticoids induce hyperglycemia through multiple mechanisms: reducing peripheral glucose uptake via downregulation of GLUT-4 transporters, increasing hepatic gluconeogenesis, and enhancing insulin resistance in hepatic and peripheral tissues [18]. The resulting hyperglycemia is characteristically postprandial and peaks 4–8 hours following steroid administration. For patients receiving once-daily morning glucocorticoids (e.g., prednisone), NPH insulin administered concurrently in the morning is an effective strategy because its peak action at 6–10 hours aligns well with the steroid-induced postprandial hyperglycemic surge [18]. For patients receiving multiple daily or long-acting steroid doses, a full basal-bolus regimen with appropriate correction scaling is preferred. Glucose monitoring is particularly important in this population as insulin requirements may change substantially with steroid tapering, creating a dynamic and evolving dosing challenge.

4.5. Patients on Total Parenteral Nutrition (TPN)

Hyperglycemia during TPN administration is associated with increased infectious complications, impaired immune function, delayed wound healing, and higher in-hospital mortality [19]. Glycemic control can be achieved through subcutaneous basal-bolus regimens or IV insulin infusion; insulin may also be admixed directly into the TPN solution, though this approach reduces flexibility and limits real-time dose adjustment. Evidence-based insulin-to-dextrose ratios range from 1:10 to 1:4 (units of insulin per gram of carbohydrate), requiring individualized titration based on daily glucose monitoring [20]. Recommended carbohydrate delivery in TPN is approximately 2 g/kg/day, with total caloric intake for ICU patients targeted at 20–25 kcal/kg/day. TPN infusion rates exceeding 4 mg/kg/min markedly increase hyperglycemia risk and should be avoided [21]. When TPN is unexpectedly discontinued, a 10% dextrose infusion should be initiated to prevent hypoglycemia until the TPN is resumed or nutritional goals are met by another route.

4.6. Diabetic Ketoacidosis (DKA)

DKA is a life-threatening complication most commonly seen in type 1 diabetes, characterized by the triad of uncontrolled hyperglycemia, ketonemia, and high anion gap metabolic acidosis resulting from absolute or relative insulin deficiency [22]. The updated ADA 2024 consensus report on hyperglycemic crises introduces several significant revisions: 1) quantitative beta-hydroxybutyrate measurement (ideally point-of-care) is now incorporated into diagnostic criteria and used to assess severity and resolution; 2) management of mild to uncomplicated moderate DKA with subcutaneous insulin in a noncritical care setting is now supported in appropriate clinical circumstances; 3) treatment pathways have been simplified to focus on three core components fluids, insulin, and potassium eliminating the previous requirement for arterial blood sampling to assess acid-base status and unifying several parameters between DKA and HHS protocols [23].

For standard moderate-to-severe DKA, initial management priorities remain volume resuscitation with isotonic saline, electrolyte correction (especially potassium, which must be ≥ 3.5 mmol/L before insulin is initiated to avoid life-threatening hypokalemia), and continuous IV insulin at 0.1 units/kg/hr. Primary therapeutic goals include reducing ketones by ≥ 0.5 mmol/L/hr, increasing bicarbonate by 3 mmol/L/hr, and lowering glucose by approximately 50–75 mg/dL/hr while maintaining potassium between 4.0–5.5 mmol/L [24]. Once blood glucose falls below 200 mg/dL, a dextrose-containing infusion (D5W or D10W) is added to allow continued insulin infusion to suppress ongoing ketogenesis. Transitioning to subcutaneous basal insulin early with a mandatory 1–2 hour overlap before discontinuing the IV infusion is critical to prevent recurrence of ketoacidosis.

4.7. Hyperglycemic Hyperosmolar State (HHS)

HHS is distinguished from DKA by the absence of significant ketoacidosis, extreme hyperosmolality, and severe dehydration, and is most commonly encountered in elderly patients with type 2 diabetes. The updated 2024 ADA consensus provides the first formalized HHS resolution criteria and unifies several management elements between DKA and HHS protocols [23]. A defining management principle is that insulin should not be initiated as the first intervention in HHS. Aggressive IV fluid resuscitation with isotonic saline must precede insulin, as early insulin administration in the setting of severe dehydration can precipitate osmotic shifts leading to circulatory collapse, cerebral edema, and osmotic demyelination [23].

Fluid replacement alone with 0.9% sodium chloride typically produces a falling blood glucose; insulin infusion (at lower doses than used in DKA approximately 0.05 units/kg/hr) should only be initiated if blood glucose fails to decline at the target rate of approximately 40–80 mg/dL/hr with adequate fluid resuscitation alone, or when glucose remains above 300 mg/dL. Serum osmolality should fall no faster than 3–8 mOsm/kg/hr to minimize the risk of neurological complications. For patients not previously on insulin, initiation of weight-based long-acting insulin (approximately 0.3 units/kg glargine subcutaneously) concurrent with or shortly after initiating fluids may be appropriate in select patients, particularly those with a mixed HHS/DKA picture [23].

5. Non-Insulin Medication Management in the Inpatient Insulin Setting

5.1 Oral and Non-Insulin Injectable Antihyperglycemics

Most oral and non-insulin injectable antihyperglycemic agents are discontinued upon hospital admission in favor of insulin because of altered pharmacokinetics, safety concerns in the acute illness setting, and the need for rapid, flexible titration [14]. Key considerations include:

Agent Class	Inpatient Concern	Recommendation
Metformin	Lactic acidosis risk with hypoperfusion, contrast exposure, renal impairment	Hold on admission; resume at discharge if renal function adequate
Sulfonylureas	Prolonged hypoglycemia risk, especially with variable oral intake	Discontinue
GLP-1 Receptor Agonists	Nausea, delayed gastric emptying; perioperative aspiration risk	Generally, hold; emerging reassurance for continued use in medical inpatients
SGLT2 Inhibitors	Euglycemic DKA risk; contraindicated with prolonged fasting or post-op	Hold ≥ 72 hours before surgery; may continue in T2DM with HF if no contraindications per ADA 2025
DPP-4 Inhibitors	Generally safe; evidence for mild-moderate hyperglycemia	May be continued in select non-critically ill patients (SITA-HOSPITAL evidence)

A notable 2025 ADA update extends the use of SGLT2 inhibitors to selected inpatients: in people with type 2 diabetes and established heart failure, SGLT2 inhibitors may be started or continued during hospitalization if there are no contraindications (i.e., no prolonged fasting or postoperative recovery). This marks a meaningful departure from previous guidance that universally withheld SGLT2 inhibitors in the inpatient setting [4].

Regarding GLP-1 receptor agonists (GLP-1 RAs), while general inpatient use is not endorsed given their nausea, delayed gastric emptying, and the lack of robust inpatient safety trials, emerging data are increasingly reassuring. A Stanford Health Care study analyzing nearly 25,000 ICU admissions over a decade found that prior GLP-1 RA use was not associated with increased in-hospital mortality or prolonged ICU/hospital stay, and that GLP-1 RAs may even be protective during critical illness through anti-inflammatory and insulin-sensitizing effects [25]. GLP-1 RAs should still be held perioperatively given the risk of delayed gastric emptying and retained gastric contents under anesthesia, but the evidence for withholding them during routine medical admissions is less definitive [25].

5.2. Non-Antihyperglycemic Agents Affecting Glycemic Control

Several commonly prescribed inpatient medications have clinically significant effects on blood glucose and require proactive consideration in insulin dosing:

- Beta-blockers: Blunt tachycardia and diaphoresis key warning symptoms of hypoglycemia increasing the risk of unrecognized hypoglycemic episodes; also impair glycogenolysis and may prolong hypoglycemia duration.
- **ACE inhibitors / ARBs:** Increase insulin sensitivity and may lower insulin requirements; continued use is beneficial for hemodynamic and renal indications but warrants monitoring for hypoglycemia.
- Thiazide and loop diuretics: Induce hyperglycemia via hypokalemia-mediated suppression of insulin secretion.
- Atypical antipsychotics (e.g., olanzapine, clozapine, quetiapine): Promote weight gain, insulin resistance, and new-onset hyperglycemia through antagonism of multiple metabolic receptors [26].
- Glucocorticoids: Exacerbate hyperglycemia through multiple mechanisms (discussed in Section 4.4).
- Calcineurin inhibitors (tacrolimus, cyclosporine): Common in post-transplant patients; directly impair pancreatic beta-cell function and induce post-transplant diabetes mellitus (PTDM).
- Vasopressors/catecholamines: Norepinephrine and epinephrine promote glycogenolysis and gluconeogenesis, contributing substantially to stress hyperglycemia in the ICU.

Each agent should be individually evaluated for continuation, dose modification, or substitution, and insulin regimens should be proactively adjusted in anticipation of their glycemic effects.

6. Continuous Glucose Monitoring in the Inpatient Setting

Continuous glucose monitoring (CGM) represents an emerging and increasingly evidence-supported adjunct to

traditional point-of-care (POC) glucose testing in the hospital. POC capillary testing provides only intermittent "snapshots" of glucose levels, missing significant nocturnal hypoglycemia, postprandial hyperglycemia, and rate-of-change trends that would otherwise inform timely insulin adjustment. CGM devices provide real-time glucose data every 1–5 minutes, enabling proactive detection of glycemic excursions, trend-guided insulin dosing, and reduced nursing burden.

Randomized trials in non-critically ill inpatients have demonstrated that CGM-guided management reduces recurrent hypoglycemic events and time spent below 70 mg/dL without compromising overall glycemic control or increasing insulin requirements or length of stay.27 A real-world protocol implementation at Stanford Health Care found that 87.8% of CGM readings met the FDA-adapted validation criterion (within 20% of higher fingerstick reads or within 20 mg/dL of lower reads), supporting the accuracy of CGM in clinical practice.27 The 2025 ADA Standards of Care acknowledge the promise of CGM technology in both outpatient and inpatient settings, and consensus statements from ADA expert panels now provide good-practice recommendations for hospital CGM implementation.4

Several important limitations exist: no CGM devices are currently FDA-approved for standalone inpatient use (most are approved for home use); critical care settings pose additional accuracy concerns due to edema, vasopressors, and poor peripheral perfusion. A hybrid protocol combining CGM with periodic fingerstick confirmatory readings has demonstrated clinical feasibility in ICU settings [27]. Pharmacists are well-positioned to lead CGM protocol development, staff education, and coordination of inpatient-to-outpatient CGM continuation, particularly for patients being newly initiated on CGM during their hospitalization.

7. Discharge Planning and Glycemic Continuity

Structured discharge planning is an essential and often underemphasized component of inpatient diabetes management. Up to 20% of hospitalized patients with diabetes are readmitted within 30 days of discharge, and inadequate diabetes education at the time of discharge is a recognized contributor to preventable readmissions [28]. The ADA and the Joint Commission recommend that patients leave the hospital with a clear, documented post-discharge glycemic plan accessible to the outpatient care team.

Effective discharge planning includes the "survival skills" framework: understanding how to monitor blood glucose, recognize and treat hypoglycemia, administer insulin correctly (including proper injection technique, site rotation to avoid lipohypertrophy, and sharps disposal), adjust for sick days, and schedule timely follow-up. When glycemic medications have been changed during hospitalization particularly for patients newly started on insulin an earlier follow-up appointment within 1–2 weeks is preferred to reassess dosing needs in the ambulatory context. Studies have shown that glycemic variability in the 48 hours preceding discharge is inversely correlated with patient comprehension scores and is a predictor of 90-day readmission, emphasizing the interplay between inpatient glycemic stability and post-discharge outcomes [28].

Provision of sufficient discharge insulin supplies, syringes or pen devices, and a glucometer with test strips must be confirmed before discharge, as lapses in access to medications and supplies are a significant driver of early readmission. Social determinants of health including insurance coverage, food security, and health literacy should be assessed and addressed as part of a multidisciplinary discharge process involving physicians, nurses, pharmacists, diabetic educators, and social workers [28].

8. Conclusion

Effective inpatient insulin management requires individualized, evidence-based regimens tailored to patient comorbidities, nutritional status, illness severity, and rapidly evolving clinical circumstances. Basal-bolus strategies, supplemented with correctional dosing, remain the preferred approach for most non-critically ill patients, with daily adjustments guided by glucose trends rather than static dose prescriptions. Special populations including the critically ill, perioperative patients, those receiving glucocorticoids or TPN, and those experiencing DKA or HHS require modification of standard protocols based on the specific pathophysiologic drivers in each context. The 2024 ADA consensus update on hyperglycemic crises introduces simplified, evidence-based diagnostic criteria and management pathways for both DKA and HHS, reinforcing the central role of fluids, insulin, and potassium as the core treatment triad.

Emerging data are beginning to reshape longstanding restrictions on inpatient non-insulin agents: SGLT2 inhibitors may now be continued in selected hospitalized patients with heart failure, and evidence is accumulating that prior GLP-1 RA use does not confer harm in the ICU. CGM technology represents the most significant technological advance in inpatient glycemic monitoring in decades and will likely become standard of care as regulatory approvals and institutional infrastructure expand. Finally, structured discharge planning including patient education, medication access, and early outpatient follow-up is integral to sustaining the glycemic improvements achieved during hospitalization.

The overarching goal of inpatient glycemic management is to achieve safe, stable glucose levels that support recovery and reduce complications during hospitalization, through a proactive, multidisciplinary approach that incorporates physicians, nurses, pharmacists, dietitians, and diabetic educators.

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Consent for Publication

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Availability of Data and Materials

Not Applicable

Conflicts of Interest

The authors declare no conflicts of interest

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