World Journal of Current Medical and Pharmaceutical Research
Content available at www.saap.org.in ISSN: 2582-0222

HYPERGLYCAEMIC EMERGENCIES AND GLYCAEMIC SAFETY IN THE CRITICALLY ILL ADULT WITH DIABETES: A NARRATIVE REVIEW WITH IMPLEMENTATION CONSIDERATIONS FOR INDIAN INTENSIVE CARE UNITS

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ARTICLE HISTORY
Received on: 14-06-2026
Revised on: 09-07-2026
Accepted on: 26-08-2026

ABSTRACT

Background. Diabetes is a common comorbidity in intensive care, and India has one of the world's largest diabetes burdens. Consensus guidance on hyperglycaemic emergencies was substantially revised in 2024, altering diagnostic thresholds, severity grading and treatment targets used since 2009. Many intensive care protocols have not yet been updated.

Objective. To summarise current guidance on recognition and management of diabetic ketoacidosis (DKA), euglycaemic DKA, sodium-glucose cotransporter-2 (SGLT2) inhibitor-associated ketoacidosis, hyperosmolar hyperglycaemic state (HHS) and mixed presentations in critically ill adults; review ICU glycaemic targets and insulin safety; and identify practical challenges in Indian units.

Methods. Narrative review of current international consensus documents, society guidelines and landmark randomised trials, identified through targeted PubMed searches and hand-searching references of major guidelines. No systematic search protocol or formal risk-of-bias/certainty assessment was undertaken; recommendations are therefore presented narratively, using the strength descriptors of their source guidelines.

Synthesis. The 2024 consensus report lowered the DKA glucose threshold to 200 mg/dL or a prior history of diabetes, recommended quantitative beta-hydroxybutyrate as the preferred ketone measure, removed the anion gap as a first-line criterion, and lowered HHS thresholds for effective osmolality and bicarbonate to 300 mOsm/kg and 15 mmol/L, respectively. Subcutaneous insulin is endorsed for mild and uncomplicated moderate DKA outside intensive care. Moderate glycaemic targets remain standard, whereas tight control (80–110 mg/dL) increases harm. Routine bicarbonate, aggressive potassium replacement and excessively slow osmolar correction are not supported.

Conclusions. Avoidable harm commonly results from superseded thresholds, treating glucose rather than metabolic state, and unsafe insulin administration without attention to potassium and feeding continuity. In Indian ICUs, implementation depends particularly on bedside ketone testing and adequate nursing capacity for safe insulin infusions.

Keywords: Diabetic ketoacidosis; hyperosmolar hyperglycaemic state; euglycaemic ketoacidosis; SGLT2 inhibitors; critical care; glycaemic control; beta-hydroxybutyrate; India.

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DOI: https://doi.org/10.37022/wjcmpr.v8i2.414

1. INTRODUCTION

Diabetes reaches the intensive care unit in two ways. Sometimes it is the reason for admission, as diabetic ketoacidosis or hyperosmolar hyperglycaemic state. More often it is a background condition in a patient admitted for something else, where it changes how that other illness behaves and how safely it can be treated. Both routes are common, and in India both are becoming more common: national survey data place the number of adults living with diabetes above one hundred million [1].

The habit that causes most trouble at the bedside is treating the glucose number in isolation. A blood glucose of 160 mg/dL in a breathless post-operative patient taking an SGLT2 inhibitor may represent severe ketoacidosis. A glucose of 900 mg/dL in an elderly patient may reflect a water deficit of several litres, where the urgent problem is the deficit rather than the number. Insulin given without checking potassium, or continued after feeding stops, causes harm that has nothing to do with the glucose reading at all.

The evidence base for this area changed materially in 2024. A joint consensus report of the American Diabetes Association, the European Association for the Study of Diabetes, the Joint British Diabetes Societies, the American Association of Clinical Endocrinology and the Diabetes Technology Society replaced guidance that had been in place since 2009 [1]. It altered diagnostic thresholds for both DKA and HHS, changed how severity is graded, and moved the preferred ketone measurement from urine to blood. Many published intensive care protocols, including ones in current local use, still reflect the older framework. That is the main practical justification for this review.

2. REVIEW QUESTION AND OBJECTIVES

This review addresses one question: how should the critically ill adult with diabetes and a hyperglycaemic emergency, or with hyperglycaemia complicating another critical illness, be recognised and managed according to current consensus guidance, and what limits the application of that guidance in Indian intensive care units?

The objectives are to set out current diagnostic criteria for DKA, euglycaemic DKA, SGLT2 inhibitor-associated ketoacidosis, HHS and mixed presentations; to summarise fluid, insulin and electrolyte management as currently recommended, distinguishing what is guideline-supported from what is customary; to review glycaemic targets and the principal sources of iatrogenic hypoglycaemia; to summarise the handling of non-insulin glucose-lowering agents during critical illness; and to describe the implementation constraints that determine whether any of this is achievable in a district or mid-sized private unit in India.

Infection in the patient with diabetes, surgical source control, nutrition and organ support are outside the scope of this review and are addressed separately.

3. METHODS

This is a narrative review. It was not conducted as a systematic review and does not claim the reproducibility of one.

Sources were identified by targeted PubMed searching between January and August 2026, using combinations of the terms diabetic ketoacidosis, hyperosmolar hyperglycaemic state, euglycaemic ketoacidosis, SGLT2 inhibitor, glycaemic control, critical illness and intensive care, restricted to English-language publications in adults. The reference lists of the major consensus documents were hand-searched. Priority was given to current international consensus statements and society guidelines, to randomised controlled trials informing the questions above, and to Indian national guidance where it exists. Older sources were retained where they remain the primary reference for an established formula or observation.

No search protocol was registered, no formal eligibility criteria were applied, study selection and data extraction were performed by a single author, and no structured risk-of-bias or certainty-of-evidence assessment was undertaken. For that reason no independent grading scheme is applied in this review. Where a recommendation is described, the strength attached to it is the strength stated by its source guideline, and the distinction is preserved throughout between recommendations, conditional suggestions, physiological rationale and local practice. Statements resting on physiological reasoning or on the author's practice are identified as such in the text.

4. DIABETES AS A CRITICAL-ILLNESS PHENOTYPE

It is useful to think of diabetes in the critically ill as acting through four interacting domains rather than as a single metabolic abnormality (Figure 01). This framework is explanatory. It draws on physiological and observational evidence, and it has not been prospectively validated as a triage instrument; it is offered as a way of organising bedside assessment, not as a scoring system.

The immune-metabolic domain covers impaired neutrophil and macrophage function, insulin resistance driven by counter-regulatory hormones and inflammatory cytokines, and unrestrained lipolysis when insulin action falls below the threshold needed to suppress it. The vascular-endothelial domain covers chronic microvascular disease and a thinned glycocalyx, which plausibly contribute to capillary leak during acute illness. The renal domain matters twice over: diabetic kidney disease increases susceptibility to acute kidney injury, and reduced renal clearance of insulin prolongs its effect, which is a recurring and under-recognised cause of late hypoglycaemia in the recovery phase. The autonomic and cardiac domain covers autonomic neuropathy, diastolic dysfunction and gastroparesis.

The practical consequence of each domain is a specific bedside caution rather than a protocol. Reduced insulin clearance means the infusion that was appropriate on day one may be excessive on day four. Diastolic dysfunction means a standard fluid bolus may be poorly tolerated. Gastroparesis means feeding is more likely to be interrupted, and interrupted feeding during an insulin infusion is dangerous.

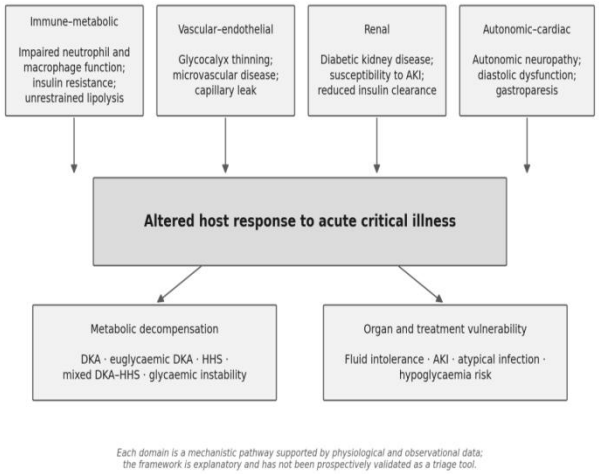

Figure 01: Diabetes as a critical-illness phenotype: four interacting domains.

The framework is explanatory. Each domain rests on physiological and observational evidence; the framework as a whole has not been prospectively validated as a triage or risk-stratification tool.

5. INITIAL ASSESSMENT AND MONITORING

No single laboratory value should determine management. A normal glucose does not exclude ketoacidosis; a normal creatinine in a sarcopenic elderly patient may conceal substantial loss of glomerular filtration; a normal temperature does not exclude sepsis. The initial panel should be taken as a set, and interpreted as a set (Table 2).

Two points deserve emphasis. First, quantitative blood beta-hydroxybutyrate should be measured at presentation in any acutely unwell patient with diabetes who is tachypnoeic, acidotic, vomiting or taking an SGLT2 inhibitor, irrespective of the glucose value. Second, venous blood gas analysis is sufficient for assessing acid-base status; the 2024 consensus explicitly moves away from requiring arterial sampling [1], which spares patients repeated arterial punctures and is easier to sustain in units without indwelling arterial lines.

Point-of-care capillary glucose measurement is acceptable for routine monitoring, and the consensus specifies capillary testing every one to two hours during DKA treatment [1]. Accuracy does degrade in shock, with vasopressor infusion, in marked peripheral oedema and in severe anaemia, and in those situations glucose should be measured on a blood gas analyser or in the central laboratory where available. This is a preference in defined circumstances, not a prohibition on capillary testing.

Table 01: Distinguishing DKA, euglycaemic DKA, HHS and mixed DKA–HHS.

Feature	DKA	Euglycaemic DKA	HHS	Mixed DKA–HHS
Glucose	≥ 200 mg/dL, or any value with a prior history of diabetes	< 200 mg/dL; may be entirely normal	≥ 600 mg/dL	Usually ≥ 600 mg/dL
Ketones (BOHB)	≥ 3.0 mmol/L (or urine ketones ≥ 2+ if BOHB unavailable)	≥ 3.0 mmol/L	< 3.0 mmol/L (urine ketones ≤ 2+)	≥ 3.0 mmol/L
Acid–base	pH < 7.3 and/or bicarbonate < 18 mmol/L	pH < 7.3 and/or bicarbonate < 18 mmol/L	pH ≥ 7.3 and bicarbonate ≥ 15 mmol/L	pH < 7.3, bicarbonate < 18 mmol/L
Osmolality	Variable	Usually normal	Effective > 300 mOsm/kg, or total > 320 mOsm/kg	Meets the HHS osmolality criterion
Typical setting	Type 1 or insulin-deficient type 2 diabetes; insulin omission; intercurrent illness	SGLT2 inhibitor use, reduced intake, pregnancy, surgery, alcohol	Older patient, type 2 diabetes, insidious onset over days	Severe illness, often sepsis or stroke
Key pitfall	Anion gap no longer a first-line criterion	Missed because the glucose looks reassuring	Threshold lowered in 2024; older criteria will miss cases	Manage as DKA while correcting hyperosmolality

BOHB, beta-hydroxybutyrate; DKA, diabetic ketoacidosis; HHS, hyperosmolar hyperglycaemic state; SGLT2, sodium-glucose cotransporter-2. Criteria are those of the 2024 international consensus report [1]. Impaired mental status is no longer a diagnostic criterion for HHS. Effective osmolality = 2 × [Na^+] + glucose (mg/dL)/18. Institutional protocols and the current version of the source guideline must be checked before clinical use.

Table 02: Initial assessment and monitoring priorities in the critically ill adult with diabetes.

Domain	At presentation	Ongoing
Metabolic	Plasma glucose; quantitative blood BOHB; venous blood gas; electrolytes, urea, creatinine; calculated effective osmolality	Glucose 1–2 hourly during insulin infusion; electrolytes, phosphate, creatinine, BOHB and venous pH approximately 4-hourly until resolution [1]
Potassium	Before the first dose of insulin, without exception	At 2 hours after starting insulin, then approximately 4-hourly [1]
Volume and cardiac reserve	Clinical assessment; consider echocardiography or lung ultrasound where prior cardiac or renal disease is known or suspected	Reassess before each fluid bolus rather than at fixed intervals
Renal	Baseline creatinine where available; recognise that a normal value may conceal reduced filtration in sarcopenia	Daily; anticipate falling insulin requirement as function recovers
Precipitant	Deliberate search: infection,	Reassess if the metabolic picture

	myocardial infarction, stroke, drug omission, new SGLT2 inhibitor	fails to improve as expected
Medication reconciliation	Identify SGLT2 inhibitors, metformin, sulfonylureas, GLP-1 receptor agonists and recent corticosteroids	Review daily; plan restart criteria before discharge from the unit

BOHB, beta-hydroxybutyrate; GLP-1, glucagon-like peptide-1; SGLT2, sodium-glucose cotransporter-2. Monitoring intervals follow the 2024 consensus report [1] and should be adapted to institutional protocol and available staffing.

6. STRESS HYPERGLYCAEMIA AND GLYCAEMIC VARIABILITY

Acute hyperglycaemia in critical illness reflects counter-regulatory hormone release and cytokine-mediated insulin resistance [18]. An observation of some prognostic interest is that, for any given glucose concentration, patients without known diabetes appear to fare worse than those with established diabetes [18,19]. The mechanistic explanation usually offered-adaptive downregulation of glucose transport in chronically hyperglycaemic cells — is plausible physiology rather than demonstrated fact, and should be described as such.

Attempts to quantify the relative elevation of glucose above a patient's own baseline, using the stress hyperglycaemia ratio or the glycaemic gap, have shown associations with adverse outcomes in several cohorts [17]. These are prognostic associations. No trial has shown that acting on such a ratio improves outcome, and specific thresholds should not be used to direct treatment.

Glycaemic variability warrants a similar caution. Greater variability is associated with mortality in observational intensive care data [16]. The coefficient of variation target of 36% that appears in many protocols, however, derives from ambulatory continuous glucose monitoring consensus in outpatients [30]; it has not been validated as an intensive care target and no trial has randomised patients to a variability target. Reducing swings in glucose is a reasonable aim. Auditing units against a specific numerical variability threshold is not currently evidence-based.

7. DIABETIC KETOACIDOSIS, EUGLYCAEMIC DKA AND SGLT2 INHIBITOR-ASSOCIATED KETOACIDOSIS

7.1 Diagnosis

The 2024 consensus defines DKA by three criteria: diabetes or hyperglycaemia, with a glucose of 200 mg/dL or greater or a prior history of diabetes; ketosis, with a beta-hydroxybutyrate of 3.0 mmol/L or greater, or urine ketones of 2+ or higher where quantitative measurement is unavailable; and metabolic acidosis, with a pH below 7.3, a bicarbonate below 18 mmol/L, or both [1].

Two changes matter. Allowing a prior history of diabetes to satisfy the first criterion regardless of the glucose value is what brings euglycaemic presentations formally within the definition. And the anion gap is no longer a first-line diagnostic criterion; it retains value where ketone measurement is unavailable, but mixed acid-base disturbances are common in DKA and the gap is an unreliable primary marker [1,22].

Blood beta-hydroxybutyrate is preferred to urine testing for a specific reason. During acidosis the ratio of beta-hydroxybutyrate to acetoacetate rises substantially; the urine nitroprusside reaction detects acetoacetate only. Ketosis is therefore underestimated at presentation and overestimated during recovery, when beta-hydroxybutyrate is oxidised back to acetoacetate and the dipstick remains positive for some time after the patient has biochemically resolved [1,20,21]. Where quantitative testing is genuinely unavailable, urine ketones of 2+ or more remain an accepted criterion and normalisation of the anion gap remains an acceptable surrogate for resolution [1]. This qualification matters in Indian practice and is stated here deliberately.

7.2 Severity and level of care

Severity is now graded on beta-hydroxybutyrate, pH, bicarbonate and mental status. Glucose is not a severity criterion, and neither is the anion gap [1]. Mild DKA is characterised by beta-hydroxybutyrate of 6 mmol/L or less, pH above 7.25, bicarbonate of 15 mmol/L or more and normal mental status; moderate by pH 7.0 to 7.25 and bicarbonate 10 to below 15; severe by beta-hydroxybutyrate above 6 mmol/L, pH below 7.0, bicarbonate below 10, and stupor or coma [1].

The consensus now recommends that mild and uncomplicated moderate DKA be managed with subcutaneous rapid-acting insulin analogues given every one to two hours, under close nursing supervision, outside the intensive care unit [1,23,24]. Severe DKA, HHS, and DKA accompanied by critical illness require intensive care. For units operating at capacity this is the single most consequential change in the 2024 document, and it is supported by randomised evidence rather than by expert preference alone.

7.3 Fluids, insulin and dextrose

For patients without cardiac or renal compromise, the consensus specifies isotonic saline or a balanced crystalloid at 500 to 1,000 mL per hour for the first two to four hours, with subsequent replacement guided by haemodynamics, fluid balance and sodium concentration; smaller boluses of around 250 mL should be considered in older patients and in those with heart or kidney failure [1]. Balanced crystalloids are suggested where available, on the basis of faster resolution of acidosis, less hyperchloraemic acidosis and shorter hospital stay [1,9,10]. This is a conditional suggestion. Isotonic saline remains acceptable, and the larger general critical care trials of balanced solutions have produced mixed results, with SMART showing a small reduction in major adverse kidney events and BaSICS and PLUS showing no mortality difference [11,12,13].

Insulin should be given as a fixed-rate intravenous infusion beginning at 0.1 units/kg/hour in severe DKA, or by a nurse-driven variable-rate protocol, and titrated to hold glucose around 200 mg/dL until ketoacidosis has resolved [1]. Dextrose 5% to 10% should be added once glucose falls below 250 mg/dL, so that insulin can continue [1]. This is the point at which the reasoning behind insulin therapy in DKA becomes visible: insulin is being given to suppress ketogenesis, and

stopping it because the glucose has normalised leaves the ketoacidosis untreated.

For patients already established on basal insulin, the consensus now permits continuing the usual basal dose alongside the intravenous infusion, which may reduce rebound hyperglycaemia and recurrence [1]. When transitioning off the infusion, subcutaneous basal insulin should be given at least one to two hours before the infusion is stopped [1].

7.4 Euglycaemic and SGLT2 inhibitor-associated ketoacidosis

Ketoacidosis with a normal or near-normal glucose is the presentation most often missed, and SGLT2 inhibitors are now its commonest precipitant [1,25]. Continued urinary glucose loss lowers the plasma glucose while ketogenesis proceeds unchecked; the patient looks metabolically unremarkable on a glucose meter while being profoundly acidotic. Typical triggers are reduced oral intake, surgery, acute illness and fasting.

The practical rule is simple: in any acidotic patient taking an SGLT2 inhibitor, measure blood ketones. Management involves withholding the drug-noting that glucosuria persists for some days after the last dose-and giving insulin with concurrent dextrose, since glucose is not elevated and insulin cannot otherwise be sustained. Specific infusion rates for this situation are not specified in consensus guidance and vary between institutions; local protocol should govern them.

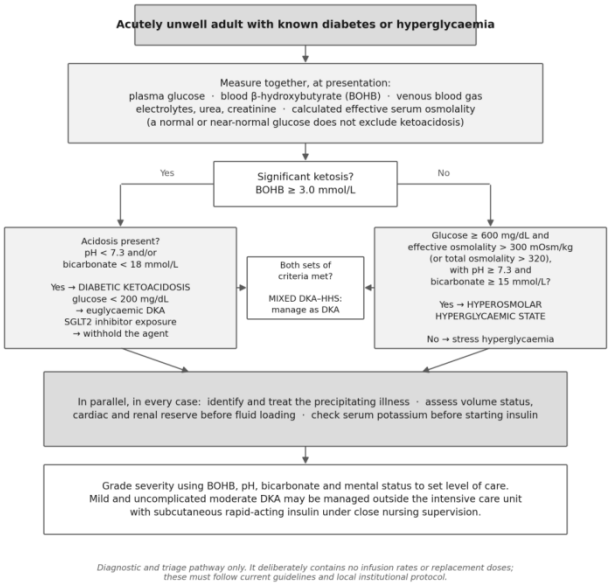

Figure 02: Diagnostic and triage pathway for suspected hyperglycaemic emergency.

Adapted from the diagnostic criteria of the 2024 international consensus report [1]. The pathway is deliberately confined to diagnosis and triage and contains no infusion rates or replacement doses; these must follow current guidelines and local institutional protocol.

8. HYPEROSMOLAR HYPERGLYCAEMIC STATE AND MIXED PRESENTATIONS

The diagnostic criteria for HHS were revised substantially in 2024 and now require all four of: glucose 600 mg/dL or greater; calculated effective serum osmolality above 300 mOsm/kg or total serum osmolality above 320 mOsm/kg; beta-hydroxybutyrate below 3.0 mmol/L or urine ketones of 2+ or less; and pH of 7.3 or above with bicarbonate of 15 mmol/L or above [1]. The effective osmolality threshold was lowered from the long-standing figure of 320 mOsm/kg, the bicarbonate criterion from 18 to 15 mmol/L, and impaired mental status was removed as a requirement because many severely affected patients are alert [1]. Protocols still using the older thresholds will fail to identify a proportion of genuine cases.

Fluid replacement is the principal therapy and the deficit is large, but the rate of correction is now specified more permissively than many local protocols allow. Glucose should fall by no more than 90 to 120 mg/dL per hour, effective osmolality by no more than 3.0 to 8.0 mOsm/kg per hour, and serum sodium by no more than 10 mmol/L in twenty-four hours [1]. Correction slower than this is not automatically safer: prolonged hypoperfusion carries its own risk of acute kidney injury.

A point of terminology requires correction because it appears widely in teaching material. The neurological risk of over-rapid correction in HHS is cerebral oedema. Osmotic demyelination syndrome, sometimes called central pontine myelinolysis, is a complication of over-rapid correction of chronic hyponatraemia and is not the mechanism at issue in a hypertonic patient.

Insulin in HHS is given at a lower fixed rate of 0.05 units/kg/hour [1]. Where features of both syndromes coexist-hyperosmolality together with significant ketonaemia or acidosis-the consensus is explicit that the patient should be treated as DKA, with a fixed-rate infusion at 0.1 units/kg/hour [1]. The 2024 report also provides formal HHS resolution criteria for the first time, which local protocols should adopt alongside the DKA criteria they usually already contain.

Standard pharmacological thromboprophylaxis is appropriate in HHS in the absence of contraindication, given hyperviscosity and immobility [1,6]. Therapeutic-intensity anticoagulation should be reserved for a documented or strongly suspected thrombotic event; routine full anticoagulation on the basis of the diagnosis alone is not recommended and carries real bleeding risk in this elderly population.

9. ELECTROLYTES, ACID-BASE AND OSMOLALITY

The most important safety rule in this whole area is that potassium precedes insulin. Insulin drives potassium intracellularly, and a patient who is hypokalaemic before treatment can become dangerously so within an hour. If potassium is below 3.5 mmol/L at presentation, insulin should be withheld and replacement begun at approximately 10 mmol/hour, with hourly rechecking, until potassium exceeds 3.5 mmol/L [1]. Replacement is then started once potassium falls below 5.0 mmol/L, aiming to hold it between 4 and 5 mmol/L [1]. Faster replacement rates require central access, continuous cardiac monitoring and specific institutional authorisation, and should not appear in a general protocol.

Routine bicarbonate is not recommended. It may be considered only in severe acidaemia with a pH below 7.0, and even there the evidence of benefit is weak [1]. Routine phosphate replacement is likewise not recommended; replacement may be considered when phosphate falls below 1.0 mmol/L, particularly in the presence of muscle weakness or cardiac or

respiratory impairment [1]. Care should be taken with units here, since 1.0 mmol/L corresponds to approximately 3.1 mg/dL, and protocols quoting a threshold of 1.0 mg/dL are treating a considerably more extreme derangement.

Sodium requires correction for hyperglycaemia before it can be interpreted. The Katz factor of 0.016 per mg/dL of glucose above 100 is conventionally applied at lower glucose values and the Hillier factor of 0.024 at higher ones [14,15]; the latter was derived experimentally in a small study and should not be regarded as validated at extreme glucose concentrations. Effective osmolality, calculated as twice the sodium plus glucose divided by 18, is the relevant quantity for assessing tonicity, since urea distributes freely across cell membranes.

Table 03: Principles of glycaemic management in the intensive care unit.

Principle	Statement	Basis
Glycaemic target	140–180 mg/dL (7.8–10.0 mmol/L) for most critically ill adults	Guideline recommendation [1,3,4]
Threshold to start insulin	Persistent glucose ≥ 180 mg/dL	Guideline recommendation [3,4]
Tight control	Targeting 80–110 mg/dL increases severe hypoglycaemia and 90-day mortality and should be avoided	Randomised trial [2]
Target during DKA treatment	Hold glucose around 200 mg/dL until ketoacidosis resolves; add dextrose once glucose < 250 mg/dL	Guideline recommendation [1]
Mortality benefit	No trial has shown that a 140–180 mg/dL target reduces mortality relative to other moderate targets; the evidence establishes harm from tight control, not benefit from this range	Interpretation of [2]
Glycaemic variability	Reducing swings is reasonable; the coefficient of variation ≤ 36% target derives from ambulatory monitoring and is not validated in critical illness	Extrapolation; not an ICU-validated target [16,30]
Glucose measurement	Capillary testing is acceptable for routine monitoring; prefer blood gas or laboratory measurement in shock, vasopressor dependence, marked oedema or severe anaemia	Guideline monitoring schedule [1]; analytical considerations

ICU, intensive care unit; DKA, diabetic ketoacidosis. Targets are for adults and exclude pregnancy. Institutional protocols and the current version of each source guideline must be checked before clinical use.

Table 04: Potassium, phosphate, acid-base and osmolality: safety considerations.

Parameter	Current guidance	Safety note
Potassium < 3.5 mmol/L	Withhold insulin; replace at approximately 10 mmol/hour; recheck hourly; start insulin once > 3.5 mmol/L [1]	Higher replacement rates require central access, continuous ECG monitoring and institutional authorisation
Potassium 3.5–5.0 mmol/L	Start insulin; begin potassium replacement, targeting 4–5 mmol/L [1]	Recheck at 2 hours after starting insulin, then approximately 4-hourly
Potassium > 5.0 mmol/L	Start insulin; withhold potassium until the level falls below 5.0 mmol/L [1]	Anticipate a rapid fall once insulin is running
Bicarbonate	Not recommended routinely; consider only if pH < 7.0 [1]	Benefit is unproven even below this threshold; risks include hypokalaemia and paradoxical CSF acidosis
Phosphate	Not recommended routinely; consider if < 1.0 mmol/L (≈ 3.1 mg/dL), particularly with muscle weakness or cardiorespiratory impairment [1]	Check units carefully; mg/dL and mmol/L thresholds differ approximately threefold
Osmolality correction (HHS)	Effective osmolality to fall by no more than 3.0–8.0 mOsm/kg/hour; sodium by no more than 10 mmol/L per 24 hours; glucose by no more than 90–120 mg/dL/hour [1]	Risk of over-rapid correction is cerebral oedema, not osmotic demyelination

CSF, cerebrospinal fluid; ECG, electrocardiogram; HHS, hyperosmolar hyperglycaemic state. Conditional and low-certainty recommendations are identified as such. Institutional protocols must be checked before clinical use.

10. GLYCAEMIC TARGETS AND INSULIN SAFETY

A target of 140 to 180 mg/dL is recommended for most critically ill adults [3,4]. The evidence underpinning it is essentially the evidence against tighter control: the NICE-SUGAR trial randomised over six thousand patients and found that targeting 81 to 108 mg/dL increased 90-day mortality and severe hypoglycaemia compared with a target of 180 mg/dL or

below [2]. It is worth being precise about what that shows. It establishes harm from tight control. It does not establish that 140 to 180 mg/dL reduces mortality relative to some other moderate range, and the claim that it does-which appears in a good deal of teaching material-reverses the direction of the inference.

Most severe hypoglycaemia in intensive care is iatrogenic and predictable. Three situations account for the majority. The first is interruption of enteral feeding or parenteral nutrition while an insulin infusion continues, typically for a procedure or transfer; the insulin rate should be reduced or stopped and a dextrose infusion substituted, and this should be an automatic feature of the order set rather than a decision made under time pressure at three in the morning. The second is recovering renal function, which increases insulin clearance and reduces requirement over days rather than hours. The third is corticosteroid tapering, where insulin requirements fall as quickly as the steroid dose does.

Steroid-associated hyperglycaemia deserves a short note. Once-daily morning corticosteroids produce a characteristic afternoon and evening rise, and intermediate-acting insulin given with the morning dose is a widely used and physiologically rational response, although the supporting trial evidence is limited. What is more important than the regimen chosen is anticipating the fall in requirement when the steroid is reduced.

11. NON-INSULIN GLUCOSE-LOWERING AGENTS DURING CRITICAL ILLNESS

Most non-insulin agents are withheld during critical illness, for reasons that differ by class (Table 05). The general principle is that these drugs were selected for a stable outpatient and their pharmacology does not suit an unstable inpatient with fluctuating renal function and unpredictable oral intake. Restart criteria matter as much as the decision to hold, and are more often forgotten: a patient discharged from intensive care without a documented plan for resuming their usual therapy is a patient at risk of prolonged uncontrolled hyperglycaemia on the ward.

Table 05: Non-insulin glucose-lowering agents during acute critical illness.

Class	Concern in critical illness	Action	Restart
Metformin	Accumulation in acute kidney injury; association with lactic acidosis in hypoperfusion, though causation is debated	Withhold on admission	After resolution of shock, with stable and adequate renal function, generally after intensive care discharge
SGLT2 inhibitors	Euglycaemic ketoacidosis during fasting, surgery or acute illness; glucosuria persists for some days after the last dose [1,25]	Withhold immediately; withhold before elective surgery per current product labelling	When eating normally, euvolaemic and off intravenous insulin
Sulfonylureas and meglitinides	Prolonged hypoglycaemia, particularly with renal impairment or poor intake	Withhold on admission	On the ward with established oral intake; avoid in significant renal impairment
GLP-1 receptor agonists	Delayed gastric emptying with implications for airway management; the magnitude of aspiration risk is debated and guidance has moved toward individualised assessment	Withhold; consider gastric ultrasound before airway manipulation where relevant	When gastrointestinal motility and oral intake are established
DPP-4 inhibitors	Low hypoglycaemia risk but limited efficacy against stress hyperglycaemia	Withhold in the unit; may have a role as ward step-down therapy	Ward step-down; note agent-specific renal dosing
Thiazolidinediones	Fluid retention; worsens heart failure and pulmonary oedema	Withhold in shock, heart failure or fluid overload	Generally avoided in patients with cardiac instability

DPP-4, dipeptidyl peptidase-4; GLP-1, glucagon-like peptide-1; SGLT2, sodium-glucose cotransporter-2. Perioperative withholding intervals are agent-specific and should be verified against current regulatory product information applicable in the reader's jurisdiction, including CDSCO-approved labelling in India. Institutional protocols must be checked before clinical use.

12. IMPLEMENTATION CONSIDERATIONS IN INDIAN INTENSIVE CARE UNITS

This section describes constraints and local practice. It is separated deliberately from the evidence-based recommendations above, and nothing in it should be read as a general recommendation.

The first constraint is ketone measurement. The 2024 consensus makes quantitative beta-hydroxybutyrate central to diagnosis, severity grading and confirmation of resolution [1],

and point-of-care meters have been available for over a decade, but access is uneven across Indian units and consumables are a recurring cost. Where testing is unavailable, the consensus itself provides the fallback: urine ketones of 2+ or more satisfy the ketosis criterion, and normalisation of the anion gap remains a reasonable surrogate for resolution [1]. Units that cannot yet fund routine bedside testing should not be left without a defensible pathway, and the fallback should be written into local protocol rather than improvised.

The second is nursing ratio. Hourly to two-hourly glucose measurement, hourly potassium checks during replacement, and safe titration of an insulin infusion all assume a level of bedside staffing that is not uniformly available. Where it is not, the consensus recommendation permitting subcutaneous rapid-acting insulin for mild and uncomplicated moderate DKA outside intensive care [1,23,24] becomes more valuable rather than less: it directs scarce intensive care capacity toward the patients who need it.

The third is drug and consumable availability. Balanced crystalloids are suggested over saline where available [1], and availability varies; isotonic saline remains an acceptable alternative and this should be stated explicitly in local protocol so that its use is not treated as a deviation. Diabetes-specific enteral formulations and newer lipid emulsions carry cost implications that are material in self-funding patients.

The fourth is patient population. Presentation is often late, with a higher proportion of patients arriving with established organ dysfunction, and a substantial number are newly diagnosed at the time of the crisis. The sarcopenic phenotype described in Indian cohorts means that serum creatinine may substantially understate renal impairment, which matters both for drug dosing and for anticipating reduced insulin clearance [27,28].

Two governance measures are, in this author's practice, worth the effort they cost: including blood ketones in the standard admission panel for any unwell patient with diabetes, and building automatic insulin reduction and dextrose substitution into the order set that governs feed interruption. Both address failures that are common, predictable and avoidable. Neither has been formally evaluated, and they are offered as local practice rather than as recommendations.

Table 06: Implementation constraints and defensible alternatives in resource-limited units.

Constraint	Preferred practice	Defensible alternative where unavailable
Point-of-care beta-hydroxybutyrate	Quantitative BOHB at presentation and to confirm resolution [1]	Urine ketones ≥ 2+ satisfy the ketosis criterion; anion gap normalisation as a resolution surrogate [1]
Arterial line for blood gas	Not required; venous blood gas is sufficient for acid-base assessment [1]	Venous sampling throughout
Continuous 1:1 nursing for insulin infusion	Fixed-rate or nurse-driven infusion with 1–2 hourly glucose monitoring [1]	Subcutaneous rapid-acting insulin every 1–2 hours for mild and uncomplicated moderate DKA outside the unit [1,23,24]
Balanced crystalloid	Suggested where available [1,9,10]	Isotonic saline is an accepted alternative; document as a supply decision, not a protocol deviation
Blood gas analyser glucose in shock	Preferred where perfusion is poor or vasopressors are running	Capillary testing with awareness of its limitations; corroborate with laboratory glucose

BOHB, beta-hydroxybutyrate; DKA, diabetic ketoacidosis. This table describes local implementation choices and does not constitute a general clinical recommendation. Institutional protocols and current guidelines must be checked before clinical use [25-26].

13. LIMITATIONS

This review has several limitations that bear on how much weight its conclusions can carry. It is narrative rather than systematic: no protocol was registered, no formal eligibility criteria were applied, and study selection and interpretation were performed by a single author, so selection bias cannot be excluded. No structured certainty-of-evidence assessment was undertaken, and the review therefore reports the strength of recommendation stated by each source rather than offering an independent appraisal [27-34].

Much of the evidence in this field is of limited certainty in its own right. The 2024 consensus is a consensus document, and several of its treatment specifications rest on expert agreement rather than randomised comparison. Fluid rates, insulin titration schedules and electrolyte replacement rates are conventions with long use rather than trial-derived optima. The four-domain framework in Section 4 is explanatory and unvalidated. The implementation section reflects one author's experience in one setting and has not been formally surveyed; Indian intensive care units vary enormously in resource and case mix, and the constraints described will not generalise uniformly.

14. FUTURE RESEARCH

Several questions are answerable and unanswered. Whether glycaemic variability is a modifiable target in critical illness, rather than a marker of illness severity, has not been tested in a randomised design. Whether the subcutaneous insulin pathway for mild and moderate DKA performs as well in resource-limited units as in the trials that established it is a question well suited to Indian multicentre study, and one with immediate operational value. The optimal rate of osmolar correction in HHS remains defined by consensus rather than by comparative trial. Finally, the accuracy of point-of-care glucose and ketone devices in shock states, and the resulting decision

errors, deserve systematic evaluation rather than the assumption of inaccuracy that currently governs practice.

15. CONCLUSIONS

Consensus guidance on hyperglycaemic emergencies changed materially in 2024, and a substantial number of intensive care protocols have not been updated. The thresholds most often out of date-the HHS effective osmolality criterion, the bicarbonate criterion, the role of the anion gap, the grading of DKA severity-are not academic details; using the superseded values will cause real cases to be missed or misclassified. Beyond the thresholds, the recurring clinical error is treating the glucose value rather than the metabolic state: stopping insulin when the glucose normalises while ketoacidosis persists, starting insulin without checking potassium, and continuing insulin when feeding stops. None of these requires new technology to avoid. They require the initial panel to be taken as a set, the protocol to be current, and the order set to anticipate the predictable failure.

16. FUNDING

None

17. CONFLICTS OF INTEREST

Not Declared

18. ETHICS APPROVAL

Not applicable. This review contains no individual patient data and no identifiable clinical information; institutional review board approval was therefore not required.

19. INFORM CONSENT

Not applicable.

20. DATA AVAILABILITY

No new data were generated or analysed in the preparation of this review.

21. AUTHOR CONTRIBUTIONS

SG conceived the review, performed the literature search, drafted and revised the manuscript, and approved the final version.

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