CME MOC

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Hyperglycemic crises in adults: A look at the 2024 consensus report

ABSTRACT

Hospital admissions for diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS), the most severe hyperglycemic emergencies in patients with diabetes, have increased considerably over the past decade. The previous version of the American Diabetes Association's consensus report on the diagnosis and treatment of DKA and HHS was published 15 years ago. The updated consensus report (June 2024) introduces revised criteria for the diagnosis and resolution of DKA and HHS, as well as new recommendations for assessment, management, and prevention.

KEY POINTS

The new report's most relevant update incorporates quantitative beta-hydroxybutyrate measurement (ideally through bedside testing) into DKA and HHS diagnostic criteria, and also recommends its use when assessing severity and determining DKA resolution.

Managing mild and uncomplicated moderate DKA with subcutaneous insulin in a noncritical care setting, when clinically appropriate, is now recommended.

Treatment pathways for DKA and HHS have been simplified and now focus only on 3 main areas—fluids, insulin, and potassium; eliminate the use of arterial blood samples to assess acid-base status; and unify some parameters between DKA and HHS management.

The new report provides the first HHS resolution criteria, updates DKA resolution criteria, and emphasizes the use of clinical judgment when making management decisions.

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Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) are the most serious hyperglycemic emergencies in diabetes.[1] Recent data show that hospital admissions for both conditions have increased substantially over the past decade,[2] underscoring the importance of early diagnosis and effective management.

The first consensus statement on hyperglycemic crises in adults was published by the American Diabetes Association in 2001[3] and was updated in 2009.[4] Here, we review the 2024 consensus report and compare current recommendations with previous guidelines.[5]

WHO WROTE THE CONSENSUS REPORT?

An international panel of experts representing the American Diabetes Association, American Association of Clinical Endocrinology, European Association for the Study of Diabetes, Joint British Diabetes Societies for Inpatient Care, and the Diabetes Technology Society reviewed the literature from 2009 to mid-2023 to provide an updated evidence-based consensus report. Published in June 2024, the report covers the epidemiology, pathogenesis, diagnosis, treatment, and prevention of DKA and HHS in adults. It is directed to the full spectrum of clinicians who care for patients with diabetes and to individuals with diabetes.[5]

WHAT ARE THE MAIN RECOMMENDATIONS?

The updated consensus includes the following recommendations for diagnosing and managing DKA and HHS.

Diagnosis

The diagnosis of DKA requires the presence of 3 criteria: (1) diabetes or hyperglycemia (D criterion), with a glucose level of 200 mg/dL or greater or a prior history of diabetes; (2) ketosis (K criterion), with a beta-hydroxybutyrate level of 3.0 mmol/L or greater or urine ketones at 2+ or higher; and (3) metabolic acidosis (A criterion), with pH less than 7.3, a bicarbonate concentration less than 18 mmol/L, or both.

The diagnosis of HHS requires the presence of 4 criteria: (1) hyperglycemia, with a plasma glucose level of 600 mg/dL or greater; (2) hyperosmolarity, with a calculated effective serum osmolality greater than 300 mOsm/kg or total serum osmolality greater than 320 mOsm/kg; (3) absence of significant ketonemia, with beta-hydroxybutyrate less than 3.0 mmol/L or a urine ketone strip of 2+ or lower; (4) absence of acidosis, with pH of 7.3 or greater and bicarbonate concentration of 15 mmol/L or greater.

Direct measurement of beta-hydroxybutyrate is strongly recommended for diagnosing DKA and monitoring treatment, using either serum samples in a central laboratory or capillary blood with point-of-care testing devices. Although both are acceptable methods with comparable precision, point-of-care testing offers easier testing and quicker results, potentially reducing admission duration and DKA recovery time. If beta-hydroxybutyrate determination is not available, a urine ketone strip of 2+ or higher will meet this criterion.

Anion gap is not recommended as a first-line diagnostic criterion, but it may have some value if ketone measurement is unavailable.

Management

Categorizing DKA severity helps guide decisions on the required level of care:

- Individuals with mild DKA (beta-hydroxybutyrate ≤ 6 mmol/L, pH > 7.25, bicarbonate ≥ 15 mmol/L, normal mental status) can be managed in a regular or observation nursing unit
- For those with moderate DKA (beta-hydroxybutyrate ≤ 6 mmol/L, pH 7.0–7.25, bicarbonate 10 to < 15 mmol/L, normal or drowsy mental status), management in a step-down or intermediate care unit is suggested
- Those with severe DKA (beta-hydroxybutyrate > 6 mmol/L, pH < 7.0, bicarbonate < 10 mmol/L, stupor or coma), HHS, or a concomitant critical illness should be managed in an intensive care unit.

DKA and HHS management involves administering intravenous fluids, insulin, and electrolytes, along with treating the precipitating cause. During treatment of DKA, capillary blood glucose should be checked every 1 to 2 hours, and electrolytes, phosphate, creatinine, beta-hydroxybutyrate, and venous pH should be checked every 4 hours until DKA resolution. For HHS, blood glucose, creatinine, electrolytes, and serum osmolality should be measured every 4 hours.

TABLE 1
2024 consensus report criteria for resolution of diabetic ketoacidosis and hyperglycemic hyperosmolar state

Resolution criteria[5]	
Diabetic ketoacidosis	**Hyperglycemic hyperosmolar state**
Plasma or capillary beta-hydroxybutyrate < 0.6 mmol/L AND Venous pH ≥ 7.3 OR Bicarbonate ≥ 18 mmol/L	Serum osmolality < 300 mOsm/kg AND Blood glucose < 250 mg/dL AND Urine output > 0.5 mL/kg/hour AND Cognitive status improved

Patients without cardiac or renal compromise should receive isotonic saline or balanced crystalloid solutions at 500 to 1,000 mL per hour for the first 2 to 4 hours. Once intravascular volume is restored, subsequent fluid replacement depends on hemodynamics, fluid balance, and sodium concentration.

Dextrose 5% to 10% should be added once glucose is less than 250 mg/dL to prevent hypoglycemia and permit insulin administration to continue until ketonemia resolves (**Table 1**).[5] For HHS, glucose reduction should not exceed 90 to 120 mg/dL per hour to avoid cerebral edema; sodium decline should not exceed 10 mmol/L in 24 hours, and osmolality should fall no more than 3.0 to 8.0 mOsm/kg per hour to minimize neurologic risks. Smaller fluid boluses (eg, 250 mL) should be considered in older adults and individuals with heart or kidney failure.

Insulin therapy for severe DKA should begin as soon as possible, either through a fixed-rate intravenous insulin infusion started at 0.1 units/kg per hour or by a nurse-driven insulin infusion protocol with a variable rate. Insulin should be adjusted to maintain glucose levels around 200 mg/dL and continued until ketoacidosis resolves (**Table 1**).

Most individuals with uncomplicated mild or moderate DKA can be treated with subcutaneous

TABLE 2
Changes in diabetic ketoacidosis diagnostic criteria between 2009 consensus statement and 2024 consensus report

Diagnostic criteria	2009 Consensus statement[4]	2024 Consensus report[5]
Plasma glucose (D criterion)	Glucose > 250 mg/dL	Glucose ≥ 200 mg/dL OR History of diabetes, irrespective of the presenting glucose value
Ketosis (K criterion)	Serum ketones: positive Urine ketones: positive	Beta-hydroxybutyrate ≥ 3 mmol/L OR Urine ketone strip ≥ 2+
Metabolic acidosis (A criterion)	pH ≤ 7.3 Bicarbonate ≤ 18 mmol/L Anion gap > 10	pH < 7.3 with or without bicarbonate < 18 mmol/L Anion gap was removed as a diagnostic criterion

TABLE 3
Changes in hyperglycemic hyperosmotic state diagnostic criteria between 2009 consensus statement and 2024 consensus report

Diagnostic criteria	2009 Consensus statement[4]	2024 Consensus report[5]
Hyperglycemia	Plasma glucose > 600 mg/dL	Plasma glucose ≥ 600 mg/dL
Hyperosmolality	Calculated effective serum osmolality > 320 mOsm/kg	Calculated osmolality: Effective[a] > 300 mOsm/kg OR Total[b] > 320 mOsm/kg
Absence of significant ketosis	Serum ketones: Small Urine ketones: Small	Beta-hydroxybutyrate < 3 mmol/L OR Urine ketones < 2+
Absence of significant acidosis	pH > 7.3 Bicarbonate > 18 mmol/L	pH ≥ 7.3 AND Bicarbonate ≥ 15 mmol/L
Mental status	Stupor or coma	Removed as a diagnostic criterion

[a]Effective osmolality calculated as 2[sodium (mmol/L)] + glucose (mmol/L)

[b]Total osmolality calculated as 2[sodium (mmol/L)] + glucose (mmol/L) + urea (mmol/L)

rapid-acting insulin analogues every 1 to 2 hours, with close nursing supervision.

For HHS, a fixed-rate intravenous insulin infusion should be started at 0.05 units/kg per hour. If there are mixed features (hyperosmolality with significant ketonemia or acidosis), the condition should be treated as DKA and a fixed-rate intravenous insulin infusion should be started at 0.1 units/kg per hour.

For patients already taking basal insulin at the time of hospitalization for DKA or HHS, basal insulin can be continued at the usual dose and adjusted as needed during hospitalization, in addition to the continuous intravenous insulin infusion. This may reduce rebound hyperglycemia and prevent recurring DKA.

To transition from intravenous to subcutaneous insulin, an estimation of the total daily insulin requirement is needed, considering hypoglycemia risk and anticipated nutritional intake. Estimations can be based on weight (estimating a total daily dose of 0.3–0.6 units/kg per day), preadmission insulin dose,

TABLE 4
Main changes in treatment recommendations between 2009 consensus statement and 2024 consensus report

		2009 Consensus statement[4]	2024 Consensus report[5]
Fluids	Type	Isotonic saline (0.9% NaCl) during the first hour Subsequently, use 0.45% NaCl if serum sodium is high or normal; continue 0.9% NaCl if serum sodium is low Change to dextrose 5% with 0.45% NaCl when glucose reaches 200 mg/dL in DKA and 300 mg/dL in HHS	Isotonic saline or balanced crystalloid solutions, with subsequent choice of fluids depending on fluid balance, hemodynamics, and sodium concentration 0.45% NaCl is indicated only if osmolality is not declining in HHS despite adequate fluid and insulin therapy Add dextrose 5% or 10% when glucose reaches < 250 mg/dL for both DKA and HHS
	Volume	15–20 mL/kg/hour or 1–1.5 L in the first hour Subsequently, 250–500 mL/hour	500–1,000 mL/hour during the first 2–4 hours Subsequently, adjust rate as clinically appropriate
	Time to correction of estimated fluid deficit	24 hours	24–48 hours (replace 50% of fluid deficit in the first 8–12 hours)
Insulin	Initial	Both DKA and HHS: 0.1 units/kg in IV bolus, followed by FRIII at 0.1 units/kg/hour OR FRIII at 0.14 units/kg/hour	Moderate and severe DKA: FRIII at 0.1 units/kg/hour (consider 0.1 units/kg IV bolus if IV access is delayed) OR Nurse-driven insulin infusion protocol Mild and moderate DKA: Subcutaneous rapid-acting insulin analogue 0.1 units/kg every 1 hour or 0.2 units/kg every 2 hours HHS: FRIII at 0.05 units/kg/hour Mixed DKA/HHS: treat as DKA
	Initial glucose goal for dextrose initiation	DKA: < 200 mg/dL HHS: < 300 mg/dL	DKA and HHS: < 250 mg/dL
	Maintenance after dextrose initiation	Decrease infusion to 0.02–0.05 units/kg/hour until resolution	Decrease infusion to 0.05 units/kg/hour until resolution
	Glucose goal until resolution	DKA: 150–200 mg/dL HHS: 200–300 mg/dL	DKA: 150–200 mg/dL HHS: 200–250 mg/dL
Potassium	Low	< 3.3 mmol/L: give 20–30 mmol/hour and postpone insulin therapy until serum potassium > 3.3 mmol/L	< 3.5 mmol/L: give 10–20 mmol/hour and postpone insulin therapy until serum potassium > 3.5 mmol/L
	Normal	3.3–5.2 mmol/L: give 20–30 mmol in each liter of IV fluid to maintain serum potassium of 4–5 mmol/L	3.5–5.0 mmol/L: give 10–20 mmol in each liter of IV fluid to maintain serum potassium of 4–5 mmol/L
	High	> 5.2 mmol/L: do not give potassium but check serum potassium every 2 hours	> 5.0 mmol/L: do not give potassium but check serum potassium every 2 hours

DKA = diabetic ketoacidosis; FRIII = fixed-rate intravenous insulin infusion; HHS = hyperglycemic hyperosmolar state; IV = intravenous

or in-hospital insulin requirements. A basal-bolus regimen is recommended, starting basal insulin at least 1 to 2 hours before stopping the insulin infusion.

Potassium should be measured at baseline, 2 hours after starting insulin, and every 4 hours thereafter until resolution of DKA. Potassium replacement should start after serum levels fall below 5.0 mmol/L to maintain levels between 4 and 5 mmol/L. If potassium levels are lower than 3.5 mmol/L at presentation, replacement should begin at a rate of 10 mmol per hour, and insulin therapy should be postponed until a potassium level higher than 3.5 mmol/L is reached.

Routine bicarbonate and phosphate administration is not recommended. Bicarbonate should be considered only in severe acidosis (pH < 7.0), and phosphate replacement should be considered if levels are under 1.0 mmol/L, particularly if muscle weakness or cardiac or respiratory impairment is present.

Patient education

Before discharge, all patients admitted with DKA or HHS should receive education focused on both the current event and overall diabetes management, including injection techniques, glucose monitoring, and urine or blood ketone testing.

WHAT IS DIFFERENT FROM PREVIOUS GUIDELINES?

Updates in the diagnosis of DKA and HHS are summarized in **Table 2** and **Table 3**, respectively, and DKA and HHS treatment updates are summarized in **Table 4**.[4,5]

Diagnosis of DKA

D criterion. Reducing the glucose cutoff to 200 mg/dL or greater is reasonable, as this level is typically diagnostic of diabetes in the general population, and there is no justification for it to be different in the context of DKA (**Table 2**). Adding a history of diabetes as an alternative to glucose values (and irrespective of them) allows for the inclusion of patients with euglycemic DKA. Mentioned in the 2009 consensus report, euglycemic DKA has become more common with the advent of sodium-glucose cotransporter 2 inhibitors. Use of these agents has been shown to increase the risk of euglycemic DKA in individuals with type 2 diabetes and in those with type 1 diabetes using them off-label.[6,7]

K criterion. This is a major update, as it involves the key diagnostic feature of DKA. The recommendation to measure beta-hydroxybutyrate is largely based on the pathophysiology of ketosis in DKA, in which the ratio of beta-hydroxybutyrate to acetoacetate rises from a physiologic 1:1 to up to 10:1. Then, during resolution, beta-hydroxybutyrate is oxidized to acetoacetate, causing its levels to decrease long before those of acetoacetate. Since the nitroprusside reaction (used to measure ketones semiquantitatively in urine and blood) measures only acetoacetate, it can underestimate the degree of ketosis at presentation but overestimate it during resolution.[8,9] Both tests have similar sensitivity, but beta-hydroxybutyrate is more specific for DKA diagnosis. Additionally, drugs can interfere with urine ketone testing[10]; in particular, false positives can be seen with commonly used medications like captopril and valproic acid. Therefore, the preferred method for assessing ketosis, both at diagnosis and during treatment, is quantitative assessment of beta-hydroxybutyrate, when available.

A criterion. The 2024 consensus removed the anion gap criterion to better account for the various factors influencing acid-base status in individuals with DKA. While an increased anion gap indicates a net gain in ketoacid anions, the accumulation of ketoacids in the extracellular fluid results in bicarbonate loss, which may not be immediately apparent due to extracellular volume contraction. Hyperglycemia-induced diuresis and natriuresis cause marked volume contraction, affecting the determination of the severity of metabolic acidemia, as standard calculations are based on concentrations rather than total content.[10] There is also associated hyperventilation due to acidosis, all of which contribute to the frequent occurrence of mixed acid-base disorders in individuals presenting with DKA.

Severity of DKA

Quantitative beta-hydroxybutyrate is now recommended for assessing DKA severity, with the introduction of quantitative cutoffs for mild and moderate (3–6 mmol/L) and severe DKA (> 6 mmol/L). Anion gap is no longer a severity criterion for DKA. The new consensus suggests assigning the level of hospital care based on DKA severity at presentation, including the possibility of managing mild DKA in the general ward. Not all criteria must be met to classify a patient as mild, moderate, or severe; clinical judgment and resource availability should ultimately determine severity and guide decisions on admission and level of care.

Diagnosis of HHS

The 2024 consensus report lowers the effective serum osmolality cutoff for diagnosing HHS and introduces total serum osmolality as a new criterion (**Table 3**). Including urea (ie, using total serum osmolality) in the diagnostic criteria, despite it not being an effective

osmolyte, accounts for the severe dehydration commonly seen in these patients.[11]

Mental status impairment is no longer a diagnostic criterion. Although past studies linked osmolality with mental status, many individuals, though very ill, do not necessarily have mental impairment, so this is no longer a requirement for diagnosis of HHS.[4,11]

Quantitative cutoffs were added for the allowed ketonemia in the diagnosis of HHS, and the bicarbonate level was lowered from 18 to 15 mmol/L to allow for a degree of acidosis that can occur due to insulinopenia.

Treatment of DKA and HHS

One key change (**Table 4**) involves the choice of fluids for initial resuscitation, now suggesting balanced crystalloids (when available) because their use is associated with faster recovery, less hyperchloremic metabolic acidosis, and shorter hospital stay.[12,13] Additionally, suggested fluid replacement speed and time are more conservative in the new consensus report, likely due to an older, more comorbid patient profile.

The 2009 consensus report introduced the concept of managing mild DKA with subcutaneous insulin, but the strength of the evidence now supports a formal recommendation of using it as an alternative to intravenous infusion in mild and uncomplicated moderate DKA,[14,15] thus avoiding the need for an intensive care unit admission.

Resolution criteria for DKA and HHS are outlined in **Table 1**. Criteria for DKA were updated to incorporate quantitative beta-hydroxybutyrate, and HHS criteria were established for the first time. The report also offers guidance on treating DKA and HHS in special populations, including older adults, those on sodium-glucose cotransporter 2 inhibitors, patients undergoing dialysis, pregnant patients, and those with COVID-19.

HOW WILL THE NEW CONSENSUS CHANGE DAILY PRACTICE?

This 2024 consensus report represents a highly anticipated update in the management of hyperglycemic emergencies. It incorporates substantial changes, 2 of which we believe directly call for changes in daily clinical practice: the inclusion of direct measurement of beta-hydroxybutyrate for diagnosis, severity assessment, management, and resolution of DKA, and the exclusion of the anion gap from the aforementioned scenarios.

Ketonemia is the hallmark of DKA, with beta-hydroxybutyrate serving as the primary marker. Given the availability of direct beta-hydroxybutyrate measurement, its use should be strongly considered, as it is associated with reduced time to recovery and greater cost-effectiveness compared with urine ketone assessments.[16] Portable ketone meters have been widely available for more than a decade and are standard of care in many countries.[17,18] When portable meters are not available, central laboratory measurement of beta-hydroxybutyrate is an alternative. We believe that efforts should be made to ensure all hospitals caring for individuals with DKA have access to direct beta-hydroxybutyrate measurement.

Approximately 30% of patients with DKA present with mixed acid-base disorders, and resuscitation with isotonic saline (the most frequently used fluid worldwide) often results in associated hyperchloremic metabolic acidosis during treatment. Therefore, using anion gap to assess treatment adequacy and resolution is unjustified whenever beta-hydroxybutyrate measurement is available. The resolution of DKA depends on the adequate suppression of ketonemia, and measurement of beta-hydroxybutyrate now represents best practice in monitoring treatment response. However, in settings where beta-hydroxybutyrate measurement is not available, we believe that normalization of anion gap is still a good surrogate marker for DKA resolution.

DISCLOSURES

Dr. Morey-Vargas has disclosed teaching and speaking for Asofarma. The other authors report no relevant financial relationships which, in the context of their contributions, could be perceived as a potential conflict of interest.

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