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Current Concepts in Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State

Miguel Angel Ruiz-Barrera[1,2,3*#], Ginna Rojas[4], Andrés Santamaría[5], Andres Fernando Ramirez Maldonado[6,7], Felipe Bernate[8], Sandy Mariottiz[9], Cecilia Senikulaciri Chong[1], Maryana Rivera Salazar[2], Daniela Sofía Guevara Castellanos[10]

[1]U.S. Army, Fort Hood, Killeen, TX, USA
[2]Juan N. Corpas University, Bogota, Colombia
[3]Rosario University, Bogota, Colombia
[4]Argentine Society of Integral Aesthetics and Nutrition (SAENI), Buenos Aires, Argentina
[5]University of Buenos Aires (UBA), Buenos Aires, Argentina
[6]U.S. Army National Guard, Jacksonville, FL, USA
[7]Community Health Outreach, Jacksonville, FL, USA
[8]Flushing Medical Center, New York, NY, USA
[9]Colombian Army, Bogota, Colombia
[10]El Bosque University, Bogota, Colombia
Email: #miguelangelruizbarrera@gmail.com, ladinoginna@gmail.com, siso.soan@hotmail.es, andresefe2208@gmail.com, felipebernate07@gmail.com, samdymariottiz31@gmail.com, Cecilia.sd.chong@gmail.com, maryana-rivera@juanncorpas.edu.co, danielasofiagc3@gmail.com

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Abstract

Introduction: Diabetic Ketoacidosis (DKA) and Hyperglycemic Hyperosmolar State (HHS) represent the most common acute hyperglycemic emergencies in diabetes mellitus (DM). This review synthesizes current evidence on the epidemiology, pathophysiology, diagnosis, and management of these critical conditions. **Methods:** We conducted a literature review utilizing PubMed, Embase, and Google Scholar, using the search terms "Hyperglycemic crises," "Diabetic ketoacidosis," and "Hyperosmolar hyperglycemic state" combined with "Epidemiology," "Physiopathology," "Clinical presentation," and "Treatment." Twenty-three articles were selected for full-text analysis. The AI tools GPT-5 (OpenAI, 2025) and Microsoft Copilot 365 (Microsoft, 2025) were used for methodological optimization and manuscript refinement. All co-authors performed a final review and approved the manuscript. **Conclusions:** DKA and HHS are life-threatening complications of DM. DKA is defined by ketosis and metabolic acidosis, whereas HHS is characterized by severe hyperosmolarity and dehydration, with minimal or absent acidosis. Key precipitants for both

*First author.
#Corresponding author.

include infections and non-adherence to treatment. Diagnosis relies on specific clinical and laboratory criteria. Management is centered on intravenous rehydration, electrolyte replacement, insulin therapy, and continuous monitoring.

Keywords

Hyperglycemic Crises, Glycemic Crises, Hyperglycemic Emergencies, Diabetic Ketoacidosis, Hyperosmolar Hyperglycemic State

1. Current Concepts in Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State

1.1. Introduction

Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) represent the most severe, life-threatening acute metabolic emergencies of diabetes mellitus (DM), forming a pathophysiological continuum driven by absolute or relative insulin deficiency coupled with excess counter-regulatory hormone activity. DKA is characterized by marked ketonemia and high-anion gap metabolic acidosis (pH < 7.3; bicarbonate < 18 mEq/L), with inpatient mortality rates generally < 1% despite accounting for up to ~50% of DM-related deaths among individuals < 24 years. HHS, by contrast, presents with profound hyperosmolarity (serum osmolality > 320 mOsm/kg) and severe hyperglycemia (glucose > 600 mg/dL), primarily affecting older adults with type 2 diabetes and conferring a nearly tenfold higher mortality risk. Insulin omission or suboptimal adherence frequently precipitates DKA, whereas HHS more commonly arises in the setting of sepsis or other major infections. Management of both conditions requires rapid intravascular volume restoration with isotonic fluids, followed by individualized insulin therapy—often via continuous intravenous infusion—correction of electrolyte abnormalities (particularly hypokalemia), and definitive treatment of the underlying trigger. As epidemiological patterns shift, with increasing DKA incidence, growing HHS occurrence in younger patients, the emergence of contributors such as SGLT2 inhibitor-associated euglycemic DKA, and evidence supporting the use of subcutaneous rapid-acting insulin analogs for selected mild-to-moderate DKA cases outside intensive care, continual reassessment of treatment strategies is essential to ensure cost-effective, evidence-based care and to reduce global disparities in outcomes [1]-[3].

This article provides the most updated information on the epidemiology, pathophysiology, clinical presentation, diagnosis, and treatment of DKA and HHS.

1.2. Methodology

A narrative review was conducted using PubMed, Embase, and Google Scholar. The key search terms included “Hyperglycemic crises,” “Glycemic crisis,” “Hy-

perglycemic emergencies," "Diabetic ketoacidosis," and "Hyperosmolar hyperglycemic state." These were combined with terms such as "Epidemiology," "Physiopathology," "Clinical presentation," and "Treatment" using Boolean operators as follows: ("Diabetic Ketoacidosis" [Mesh] OR "Hyperosmolar Hyperglycemic State" [Mesh] OR "hyperglycemic emergency" OR "hyperglycemic emergencies") AND ("Therapeutics" [Mesh] OR "Patient Care Management" [Mesh] OR treatment OR management OR "clinical approach" OR therapy). The search strategy was refined with the assistance of GPT-5 (OpenAI, 2025) and Microsoft Copilot 365 (Microsoft, 2025), which facilitated the development of effective, database-specific Boolean search strings tailored to PubMed, Embase, and Google Scholar. This approach enabled the selection of twenty-three articles in English and Spanish for full-text review.

Artificial intelligence tools did not affected the technical accuracy or scientific content of this manuscript. Their role was confined to optimizing the search strategy, enhancing paragraph transitions, simplifying complex explanations, and maintaining consistency in medical terminology. The final version underwent a thorough manual review by all co-authors to ensure accuracy, coherence, and integrity.

2. Diabetic Ketoacidosis

2.1. Definition

DKA is the most common hyperglycemic emergency in type 1 diabetes mellitus (T1DM); however, it can also develop in patients with type 2 diabetes mellitus (T2DM). This condition is characterized by hyperglycemia, metabolic acidosis with an elevated anion gap, and ketonemia or ketonuria. Triggering factors may vary but often occur in the context of absolute or relative insulin deficiency [1].

2.2. Epidemiology

In the United States, the incidence of DKA increased from 32 cases per 10,000 hospital admissions in 2003 to 53.4 in 2014 and 61.6 in 2017. This condition is more common in males, with an incidence of 71.2 cases per 10,000 admissions, and is most frequently observed in individuals under 45 years of age. The mortality rate among males with DKA is 40.5 deaths per 10,000 cases, with overall mortality rising significantly in older age groups. DKA occurs much more frequently in patients with type T1DM, at 82.6 episodes per 1000 person-years, compared to 3.2 episodes per 1000 person-years in T2DM [2].

2.3. Physiopathology

Total or partial insulin deficiency, combined with elevated levels of counter-regulatory hormones (including catecholamines, cortisol, glucagon, and growth hormone), stimulates hepatic gluconeogenesis, peripheral glycogenolysis, and lipolysis. These processes markedly increase plasma concentrations of glucose, free fatty

acids, and glycerol, resulting in hyperglycemia and metabolic acidosis with an elevated anion gap. Additionally, the upregulation of pro-inflammatory cytokines and oxidative stress markers (such as TNF-α, IL-6, IL-8, and CRP), along with increased production of reactive oxygen species, plasminogen activator inhibitor-1, and free fatty acids, further exacerbates and perpetuates DKA, while also heightening the risk of thrombotic complications (**Figure 1**) [1] [3] [4].

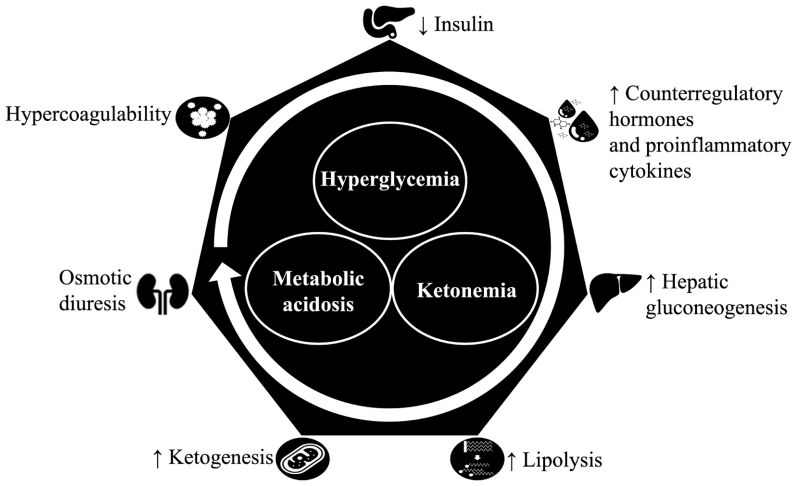


Figure 1. Pathophysiology of DKA. (↑) Increase, (↓) Decrease.

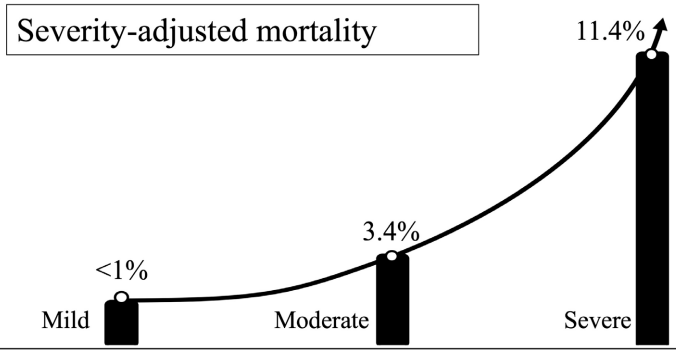
2.4. Clinical Presentation

DKA typically develops within 24 hours or less. Clinical presentation can include polydipsia, polyuria, polyphagia, weakness, fatigue, acute abdominal pain, nausea, and vomiting. Key signs of severe acidosis are Kussmaul respiration (compensatory deep, labored breathing) and a characteristic "fruity" breath odor due to acetone. Compared to HHS, altered mental status is less common and is usually associated with severe complications such as cerebral edema, which may result from the rapid correction of electrolyte and osmolar imbalances. Other potential complications include heart failure due to fluid overload, electrolyte disturbances, acute respiratory distress, spontaneous pneumothorax, and both arterial and venous thromboembolic events. Key precipitating factors for DKA include poor adherence to treatment (60%) and infections (17.2% to 23.8%) most commonly urinary tract infections, pneumonia, dental or skin abscesses, and viral illnesses. Additional triggers may include acute medical conditions (appendicitis, pancreatitis, abdominal inflammation, trauma, pregnancy, cerebrovascular disease, and acute myocardial infarction) and the use of certain drugs or substances (cocaine, alcohol, sympathomimetics, atypical antipsychotics, corticosteroids, and thiazide diuretics) [5]-[9].

2.5. Diagnosis

DKA can be classified as mild, moderate, or severe (**Figure 2**). The diagnostic approach should include assessment of plasma glucose levels, arterial blood gases,

and a comprehensive panel of electrolytes (potassium, sodium, calcium, phosphorus, and magnesium). Additional laboratory tests should include blood urea nitrogen (BUN), serum creatinine, serum bicarbonate, urine and blood ketones, effective serum osmolarity, anion gap, and plasma β-hydroxybutyrate levels. Most patients present with plasma glucose levels ≥ 250 mg/dL, blood pH < 7.3, bicarbonate between 10 and 15 mEq/L, and an anion gap > 12. Measurement of HbA1c can help distinguish between an acute event and an exacerbation of a chronic condition. Further diagnostic workup, such as complete blood count, chest X-ray, and microbiological tests, may provide valuable information about the underlying etiology [1] [10] [11].



Classification of DKA			
Criteria	**Mild**	**Moderate**	**Severe**
Plasma glucose	≥200 mg/dL	≥200 mg/dL	≥200 mg/dL
Arterial pH	>7.25 to <7.30	7.0 to 7.25	<7.0
β-hydroxybutyrate	3.0 to 6.0 mmol/L	3.0–6.0 mmol/L	>6.0 mmol/L
bicarbonate	15 to 18 mEq/L	10 to 14 mEq/L	<10 mEq/L
Mental status	Alert	Alert or Somnolent	Stuporous or Comatose

Figure 2. Classification of DKA and severity-adjusted mortality.

3. Hyperglycemic Hyperosmolar State

3.1. Definition

HHS is characterized by severe hyperglycemia (blood glucose > 600 mg/dL) and elevated serum osmolarity (> 320 mOsm/kg) in the absence of significant ketoacidosis, however, in many patients, features of the two disorders (DKA and HHS) may co-exist. This condition is more prevalent in individuals with T2DM and is commonly precipitated by infections, acute illnesses, or poor adherence to hypoglycemic therapy. Unlike DKA, patients with HHS typically present with severe dehydration and varying degrees of altered mental status [9] [11].

3.2. Epidemiology

In the United States, HHS accounts for 35% of all admissions related to hyperglycemic emergencies. This condition primarily affects individuals aged 45 to 64

(47.5%), the vast majority of whom have T2DM (88.1%). Its geographic distribution shows higher incidence rates in developed countries with a high prevalence of T2DM, such as the United States, Western Europe, and Japan. The mortality rate for HHS ranges from 5% to 13.2%. Key risk factors include advanced age, chronic comorbidities (cardiovascular, renal, and neurological diseases), infections (most commonly urinary and respiratory infections which precipitate approximately 40% to 60% of DKA cases), the use of hyperglycemic medications (thiazide diuretics, glucocorticoids, beta-blockers, atypical antipsychotics), poor adherence to insulin or oral hypoglycemics, and acute events such as surgical procedures or trauma [12] [13].

3.3. Physiopathology

HHS is driven by peripheral insulin resistance and low insulin levels, combined with a surge in counter-regulatory hormones (glucagon, catecholamines, cortisol, growth hormone). This hormonal state strongly promotes hepatic gluconeogenesis and glycogenolysis while simultaneously reducing peripheral glucose uptake. The resulting severe hyperglycemia triggers an osmotic diuresis, leading to glucosuria, massive water loss, electrolyte imbalances, and plasma hyperosmolarity. This severe metabolic disruption places patients at high risk for neurological, vascular, and other systemic complications [13].

3.4. Clinical Presentation

HHS typically has an insidious onset, developing slowly over days or weeks. Initial symptoms include polyuria, polydipsia, polyphagia, and weight loss. As the condition progresses, signs of profound dehydration appear, such as dry mucous membranes, enophthalmos (sunken eyes), loss of skin turgor, tachycardia, and potentially hypotension or shock. A key feature distinguishing HHS is the prevalence of neurological symptoms; changes in mental status are common, ranging from mild confusion to coma [14]-[16].

3.5. Diagnosis

Assessment of HHS should include the following laboratory parameters: BUN, serum creatinine, serum bicarbonate, urine and blood ketones, effective serum osmolarity, anion gap, and plasma β-hydroxybutyrate levels. Diagnostic criteria for HHS include serum osmolarity > 320 mOsm/kg, pH > 7.3, β-hydroxybutyrate < 3 mmol/L, and serum bicarbonate > 15 mmol/L. Notably, up to 30% of patients may simultaneously exhibit clinical and laboratory features of both DKA and HHS. Key precipitating factors include infections (30% to 60% of cases), poor treatment adherence, inadequate diabetes management, comorbidities (acute myocardial infarction, cerebrovascular events, pancreatitis, appendicitis, and trauma), use of certain medications (thiazide diuretics, atypical antipsychotics, sympathomimetics, corticosteroids, and pentamidine), and substances (alcohol and cocaine). Importantly, HHS may be the initial presentation of T2DM in up to 17%

of cases [11] [14] [17]-[19].

3.6. Management of DKA and HHS

In addition to identifying and addressing the underlying cause, the management of DKA and HHS focuses on correcting dehydration, plasma hyperosmolarity, hyperglycemia, ketonemia, electrolyte imbalances, and metabolic acidosis. During treatment, it is essential to monitor vital signs, maintain fluid balance, individualize insulin dosing, and assess plasma glucose, electrolytes concentration, and arterial blood gases every two to four hours. The formulas shown in Table 1 can be used to calculate the anion gap, corrected sodium (Na), and plasma osmolarity.

Table 1. Anion gap > 12 mEq/L may indicate metabolic acidosis. Na: Sodium (mEq/L), Cl: Chloride (mEq/L), HCO3: Bicarbonate (mEq/L).

Parameter	Formula
Anion gap	Na-(Cl + HCO3)
Corrected sodium when glucose < 400 mg/dL	Na + 0.016 [glucose-100]
Corrected sodium when glucose > 400 mg/dL	Na + 0.024 [glucose-100]
Plasma osmolarity	(2 Na) + (glucose/18)

Aggressive intravenous rehydration aims to restore circulatory volume, improve tissue perfusion and glomerular filtration rate, correct plasma hyperosmolarity, and reduce hyperglycemia. To achieve these objectives, 500 to 1000 mL/h of 0.9% sodium chloride (NaCl) should be administered during the first two hours. This is followed by 250 to 500 mL/h of 0.9% or 0.45% NaCl, depending on plasma sodium levels and the severity of dehydration. In adults, fluid replacement should be guided by dynamic monitoring of hemodynamic status, urine output, and serial electrolyte checks. In children, maintenance fluids should continue following the "4-2-1" rule: 4 mL/kg/h for the first 10 kg of body weight, 2 mL/kg/h for the next 10 kg (11 to 20 kg), and 1 mL/kg/h for each additional kilogram above 20 kg. Once plasma glucose levels reach ≤ 250 mg/dL (13.9 mmol/L), dextrose (5% or 10%) should be added to the NaCl solution to reduce the risk of hypoglycemia [9] [11] [20]. To prevent cerebral edema and other neurological complications in HHS, the correction of plasma glucose and sodium levels should not exceed 90 - 120 mg/dL/h (5 - 6.7 mmol/L/h) and 10 mEq/L/24 hours (10 mmol/L/24 hours), respectively. The reduction rate in osmolarity should be maintained between 3.0 and 8.0 mOsm/kg/h [11].

3.7. Insulin Therapy

Insulin administration should only be initiated when serum potassium is >3.3 mEq/L. The primary goal of this therapy is to restore cellular metabolism, reduce hepatic gluconeogenesis, suppress lipolysis and ketogenesis, and correct plasma osmolarity. In patients with DKA, a bolus of rapid-acting insulin should be administered intravenously (IV) or intramuscularly (IM) at 0.1 U/kg, followed by a continuous infusion of 0.1 U/kg/h to target blood glucose levels between 150 - 200

mg/dL (8.3 - 11.1 mmol/L). For patients with HHS without significant ketosis or acidosis, the insulin infusion should be 0.05 U/kg/h to maintain a plasma glucose level between 250 - 300 mg/dL until mental status improves and hyperosmolarity is corrected [10] [11].

The transition from IV to subcutaneous (SC) insulin should be considered when the patient is alert and able to eat; this switch is made once ketoacidosis is resolved. It is advisable to continue the IV infusion for 1 to 2 hours after the first SC injection to prevent rebound hyperglycemia and ketoacidosis, as IV regular insulin has a short half-life (approximately 10 minutes). The SC dose for newly diagnosed patients is 0.5 - 0.8 U/kg/day, using a basal-bolus regimen. For patients previously on insulin, their home dose can be resumed with appropriate adjustments [1] [10] [11].

3.8. Potassium

Patients with DKA and HHS present with a total potassium deficit estimated between 3 - 5 mEq/kg. Despite this deficit, the serum potassium level is often normal or even elevated upon admission. This is due to metabolic acidosis and insulin deficiency, which cause an extracellular shift of intracellular potassium to compensate for hypertonicity and acidosis. Insulin administration, acidosis correction, and fluid resuscitation all drive potassium back into the cells, decreasing the serum concentration [1] [11].

Potassium replacement should be initiated when the serum concentration is below 5.0 mEq/L to maintain levels between 4 - 5 mEq/L. For most patients, administering 20 - 30 mEq of potassium per liter of fluids is adequate; however, patients with acute or chronic kidney failure require lower doses. When serum potassium falls below 3.3 mEq/L, replacement should begin at a rate of 10 - 20 mEq/h, and insulin therapy should be withheld until potassium levels reach at least 3.3 mEq/L due to the high risk of cardiac arrhythmias and respiratory muscle weakness [1] [11].

3.9. Bicarbonate and Phosphate

Bicarbonate infusion therapy has not been shown to reduce mortality or complications in DKA patients with a blood pH > 7.0. Its use may increase the risk of hypokalemia, reduce tissue oxygen uptake, impair cardiac function, and contribute to cerebral edema. In patients with severe acidosis (pH ≤ 7.0), 50 - 100 mEq of sodium bicarbonate in an isotonic solution is recommended to achieve a pH > 7.0.

The administration of potassium phosphate (0.25 - 0.5 mmol/kg/IV/4 - 6 hours) may be indicated for patients with respiratory muscle weakness or reduced cardiac contractility if serum phosphate levels are <1.0 mmol/L. However, insufficient evidence supports its routine use [11].

3.10. Antithrombotic Prophylaxis

Hyperglycemia is associated with a hypercoagulable state, which is particularly

relevant in patients with HHS. This risk is even higher during the three months post-hospitalization. The British guideline and other reviews suggest using prophylactic doses of low-molecular-weight heparin during the hospital stay. This recommendation must be weighed against the higher risk of bleeding associated with advanced age and common comorbidities in these patients [21].

3.11. Ulcer Prevention

Once the patient is stabilized, a detailed physical examination should be performed to assess for diabetic neuropathy or pre-existing vascular lesions. This examination should be conducted daily, especially in individuals with altered mental status, given their high risk of developing ulcers and requiring non-traumatic amputations [22].

3.12. Conclusion

DKA and HHS are severe complications of DM. Both conditions are characterized by severe hyperglycemia; however, DKA is distinguished by ketosis and metabolic acidosis, while HHS is marked by profound dehydration and hyperosmolality without significant acidosis. The main precipitating factors include infections, poor treatment adherence, and the use of certain medications and substances. The diagnosis and classification of both conditions are primarily based on evaluating specific biochemical parameters and the patient's mental status. Finally, treatment involves vigorous rehydration, correction of electrolyte imbalances, insulin administration, and continuous monitoring to prevent complications.

Statement

All authors had access to the data and equally contributed to develop this manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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