The Journal of Emergency Medicine, Vol. 45, No. 5, pp. 797–805, 2013

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2013.03.040

Clinical Reviews

HYPERGLYCEMIC CRISIS

Ronald Van Ness-Otunnu, MD, MS* and Jason B. Hack, MD, FACEP†

*Emergency Care Center, Sturdy Memorial Hospital, Attleboro, Massachusetts and †Division of Medical Toxicology, Department of Emergency Medicine, Warren Alpert Medical School of Brown University, Providence, Rhode Island

Reprint Address: Ronald Van Ness-Otunnu, MD, MS, Emergency Care Center, Sturdy Memorial Hospital, 211 Park Street, Attleboro, MA 02703

☐ **Abstract—Background: Hyperglycemic crisis is a metabolic emergency associated with uncontrolled diabetes mellitus that may result in significant morbidity or death. Acute interventions are required to manage hypovolemia, acidemia, hyperglycemia, electrolyte abnormalities, and precipitating causes. Despite advances in the prevention and management of diabetes, its prevalence and associated health care costs continue to increase worldwide. Hyperglycemic crisis typically requires critical care management and hospitalization and contributes to global health expenditures. Objective: Diagnostic and resolution criteria and management strategies for diabetic ketoacidosis and hyperosmolar hyperglycemic crisis are provided. A discussion of prevalence, mortality, pathophysiology, risk factors, clinical presentation, differential diagnosis, evaluation, and management considerations for hyperglycemic crisis are included. Discussion: Emergency physicians confront the most severe sequelae of uncontrolled diabetes and provide crucial, life-saving management. With ongoing efforts from diabetes societies to incorporate the latest clinical research to refine treatment guidelines, management and outcomes of hyperglycemic crisis in the emergency department continue to improve. Conclusion: We provide an overview of the evaluation and treatment of hyperglycemic crisis and offer a concise, targeted management algorithm to aid the practicing emergency physician.** © **2013 Elsevier Inc.**

☐ **Keywords—diabetes; diabetic ketoacidosis; hyperglycemic crisis; hyperglycemic emergency; hyperosmolar hyperglycemic state; metabolic acidosis**

INTRODUCTION

Hyperglycemic crisis includes diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS). Both are extreme metabolic derangements associated with uncontrolled types 1 and 2 diabetes mellitus that may result in shock, coma, or death. These life-threatening endocrine emergencies demand swift, repeated clinical and laboratory assessment; monitoring; correction of hypovolemia, acidemia, hyperglycemia, ketonemia, and electrolytes; and treatment of the precipitating causes. Consensus statements provided by the American Diabetes Association (ADA) for the care of adult patients with hyperglycemic crisis and by the International Society for Pediatric and Adolescent Diabetes (ISPAD) for the care of children and adolescents with DKA are excellent primary resources for diagnosis and management (1,2).

As of 2010, >285 million adults worldwide have diabetes, with estimated yearly global health expenditures totaling >$376 billion (3). In the United States (US), the number of Americans with diabetes has more than quadrupled, from 5.6 million in 1980 to 25.8 million in 2010, with direct and indirect health care costs of >$174 billion (3,4). The incidence of type 1 diabetes is increasing globally, particularly in children <5 years of age, and the earlier onset of type 2 diabetes is a growing concern (5). In a multicenter, population-based study of

RECEIVED: 29 May 2012; FINAL SUBMISSION RECEIVED: 14 December 2012;
ACCEPTED: 28 March 2013

patients <20 years of age who were diagnosed with diabetes, the prevalence of DKA at the initial diagnosis was >25% (6). US population–based studies report the annual incidence of DKA to range from four to eight episodes per 1000 diabetic patient admissions, with an average duration of hospital stay of 3.6 days (4,7). Hyperglycemic crises often require critical care management and are associated with significant health care costs, morbidity, and mortality. The mortality rate from DKA in children ranges from 0.15% to 0.30%, with cerebral edema responsible for 60% to 90% of these deaths (2). Among adults, DKA-associated mortality is often attributable to precipitating or concurrent events, such as sepsis, pneumonia, hypokalemia, acute myocardial infarction (MI), and acute respiratory distress syndrome (8).

Improved understanding of pathophysiology and advances in diabetes prevention and management has resulted in sharply declining death rates in the United States (9). In 1980, among the 0- to 44-year-old age group, 45.5 deaths per 100,000 diabetic patients were attributable to hyperglycemic crisis, compared with 26.2 in 2005 (4). In patients >75 years of age, even greater improvement was observed, with 20.5 deaths per 100,000 in 2005 compared with 140.2 per 100,000 in 1980 (4). Ongoing research holds promise for further decreases, including the early identification and management of patients at risk, improvements in the accuracy and efficiency of acidosis measurement, and trials of alternative insulin regimens for acute management (10).

DISCUSSION

Diagnostic Criteria for DKA and HHS

The diagnosis of hyperglycemic crisis is possible within minutes of a diabetic patient's presentation to the emergency department if classic signs and symptoms are appreciated and point-of-care testing is used. DKA is distinguished by a blood glucose of >250 mg/dL, moderate ketonuria or ketonemia, arterial pH of <7.3, and a bicarbonate of <15 mEq/L (1). A diagnosis of HHS may be presumed in a diabetic patient with an altered sensorium, severely elevated glucose (usually >600 mg/dL), minimal or no ketonuria or ketonemia, serum osmolality >320 mOsm/kg, arterial pH (typically) >7.3, and a bicarbonate of >15 mEq/L (1). Distinct pathophysiologic features account for the laboratory findings that define both DKA and HHS.

Pathophysiology

Diabetes mellitus (DM) is a broad term for diseases distinguished by insufficient endogenous insulin that result in hyperglycemia. The role of insulin is crucial to understanding the pathophysiology of diabetes and hyperglycemic crisis. Insulin stimulates hepatocellular glucose uptake, glycogen storage, and lipogenesis. Opposite to glucagon, insulin inhibits hepatic glycogenolysis and gluconeogenesis. Type 1 DM is defined by progressive and irreversible autoimmune-mediated destruction of pancreatic beta cells, typically leading to absolute insulin deficiency (11). Type 2 DM is distinguished by a progressive insulin resistance and defects in insulin secretion leading to a relative insulin deficiency that may eventually require exogenous insulin (11).

DKA and HHS are severe complications of DM. A combination of hormonal imbalances causes DKA. In the setting of insulin deficiency, increased glucagon, catecholamines, cortisol, and growth hormones lead to increased extracellular glucose, decreased glucose use, and hyperglycemia (1). These counter-regulatory and stress hormones stimulate lipolytic pathways, and the resultant free fatty acids are oxidized to ketone bodies, such as acetone, acetoacetate, and beta-3-hydroxybutyrate. Beta-3-hydroxybutyrate contributes most prominently to an anion gap metabolic acidosis.

In contrast, patients with HHS have some pancreatic beta cell function, and the degree of lipolysis required to produce a measurable ketonemia may not occur. Significantly higher hyperglycemia (>600 mg/dL) is often observed in comparison with DKA. HHS is characterized by severe hyperglycemic diuresis and dehydration, hypernatremia, minimal to absent ketonemia, and serum osmolality of >320 mOsm/kg. Because of severe hypernatremia and elevated serum osmolality, HHS patients more often present with severe mental status changes, including coma (1).

Hyperglycemia itself imposes an osmotic load that favors an intravascular fluid shift, osmotic diuresis, and dehydration. Nausea and vomiting induced by ketonemia also contributes to fluid losses and a profound hypovolemic state. The typical total body water deficit is 6 L in DKA and 9 L in HHS (1). In addition, there are total body losses of key minerals and electrolytes, including sodium, chloride, potassium, phosphate, calcium, and magnesium. Serum electrolytes measured in the setting of intravascular contraction may offer falsely normal results and not accurately represent total body depletion. The net result of these combined biochemical imbalances is an acutely ill, acidotic, ketonemic, hyperglycemic, dehydrated, and electrolyte-depleted patient.

Risk Factors for Hyperglycemic Crisis

Usher-Smith et al. reviewed 46 studies in 31 countries to identify factors associated with the presence of DKA at diagnosis of diabetes among children and adolescents.

They reported data from two US studies that revealed that young patients without health insurance or with Medicaid coverage alone had a combined odds ratio of 3.20 when compared to the privately insured for presenting with DKA at diagnosis of diabetes (12). Worldwide, major factors included age <2 years, ethnic minority status, inciting infection, low body mass index, and delayed or earlier missed diagnosis (12). In adults, hyperglycemic crisis may be precipitated by the stressors outlined in Table 1. Among these, infection and inadequate exogenous insulin are the most common (1). Other risks include prescribed drugs that interfere with carbohydrate metabolism, eating disorders that lead to starvation and anorexia, pregnancy, and stress imposed by surgery, trauma, or shock (1,13,14).

Table 1. Precipitants of Hyperglycemic Crisis*

Infectious
Sepsis
Pneumonia
Urinary tract infection
Meningitis
Cardiac
Myocardial infarction
Psychological/social
Inadequate exogenous insulin
Anorexia
Starvation ketosis
Gastrointestinal
Pancreatitis
Neurologic
Cerebrovascular accident
Toxicologic
Cocaine
Calcium channel blockers
Pharmacologic
Sympathomimetics
Corticosteroids
Pentamidine
Thiazide diuretics
Atypical antipsychotics
Endocrine
Gestational diabetes mellitus
Hyperthyroidism
Adrenal disorders (e.g., pheochromocytoma, Cushing syndrome)
Other
Pregnancy
Trauma
Surgery
Shock states (e.g., hypovolemic, cardiogenic)

* From references (1,13,14).

Clinical Presentation and Precipitating Factors

In contrast to the acute onset of DKA, which occurs over hours to days, patients with HHS evolve signs and symptoms over days to weeks and frequently present with severely altered mentation (1). Additional causes of altered mentation that may also be seen with DKA include uremic or lactic acidosis, stroke, meningitis, and alcohol or illicit drug intoxication. Because of difficulties in history-taking from a lethargic or near comatose patient, soliciting the help of family, obtaining a full medication list from a pharmacy, or questioning emergency medical first responders for additional information may provide critically important clues to life-threatening etiologies. A screening electrocardiogram (ECG) should be obtained early in the evaluation to identify a possible MI. If a patient's medication list includes antidepressants or if the history reveals depression or suicidality, a toxicologic investigation may be warranted. Toxic causes of acid-base imbalance, including aspirin, methanol, ethylene glycol, and cyanide, must be entertained.

History

A patient's history and review of systems should include questions that may point to an infection, the single most common precipitant of hyperglycemic crisis (1). A recent study suggests that infection more often accounts for severe DKA and that mild to moderate DKA is associated with missed insulin doses or a change in regimen (15). Noninfectious precipitants may include prescribed or illicit drugs, MI, cerebrovascular accident, and pancreatitis (1). Patients with eating disorders may withhold their insulin to avoid weight gain, inadvertently precipitating DKA (1). Pregnancy is an insulin-resistant state, and gestational diabetes or pregnancy in established diabetics may also provoke hyperglycemic crisis (16).

A multicenter, population-based study of diabetics diagnosed before 20 years of age revealed that patients with lower family income, those with Medicaid compared to those having no insurance, and patients from families with less than a high school education have increased odds ratios to present with DKA at diagnosis (6).

Review of Systems

Polyuria, polydipsia, weight loss, profuse vomiting, and diffuse abdominal pain are pertinent positive symptoms that are classically associated with hyperglycemic crisis.

Physical Examination

Dehydration, poor skin turgor, altered mentation, lethargy, tachycardia, and hypotension are often present on examination, and patients may have a fruity, ketotic breath odor. Kussmaul breathing—a deep, labored pattern indicative of a hyperventilatory response to metabolic acidosis—is often seen in patients with DKA.

Differential Diagnosis

Causes of severe hyperglycemia include DKA, HHS, new onset diabetes, gestational diabetes, insulin noncompliance, metabolic syndrome, medication effect (e.g., steroids, cyclosporine, and atypical antipsychotics), toxicity (e.g., calcium channel blocker overdose), and endocrine diseases affecting the adrenal gland. Other causes of significant ketonemia include ethanol, salicylate poisoning, and isopropanol toxicity.

Although infection is the most common precipitant of hyperglycemic crisis, it is important to maintain a broad differential diagnosis. DKA is both a systemic inflammatory illness and a cause of vascular endothelial injury that can result in disseminated intravascular coagulation and pulmonary interstitial edema, as well as hypercoagulable pathologies, such as stroke, pulmonary embolism, and dural sinus thrombosis (17). Acute MI is another reported precipitant of hyperglycemic crisis that must not be missed (1). A high level of clinical suspicion for concurrent life-threatening illness, precipitants, or sequelae should be maintained.

If symptoms such as abdominal pain do not resolve as expected with treatment or the pain becomes more localized, persistent or changing symptoms should lead to additional work-up. In DKA, diffuse abdominal pain typically follows periods of protracted vomiting, dehydration, and worsening acidemia. Pancreatitis is a well-known precipitant of DKA and may be a source of pain. Reassessment of any abdominal complaint is important because persistent or localized pain after initial fluid boluses and a correction of acidosis may reveal a "hidden" surgical etiology, such as appendicitis.

Diagnostic Testing

The diagnosis of hyperglycemic crisis is suggested by history and classic signs and symptoms and can be confirmed with routine laboratory tests. Obtaining a bedside glucose measurement is a critical first step. Although much less common, the phenomenon of "euglycemic diabetic ketoacidosis," first elucidated by Munro et al. in 1973 and thereafter defined as glucose levels $\leq$250 mg/dL in the setting of DKA, may account for up to 10% of DKA patients (1,18). Additional diagnostic tests should be directed by clinical suspicion for particular precipitants of the hyperglycemic crisis. Leukocytosis is often present as a reaction to stressors; however, it is prudent to investigate potential causes of elevated white blood cells and maintain a high level of suspicion for infection. Especially critical is the need for a screening ECG to evaluate for myocardial ischemia as a trigger leading to DKA.

Basic laboratory tests include urine ketones, sodium, potassium, chloride, bicarbonate, blood urea nitrogen, creatinine, glucose, lactate, venous or arterial blood gas, serum osmolality, and beta-hydroxybutyrate or serum ketones. Additional blood tests are based on clinical circumstance and may include cardiac enzymes, a disseminated intravascular coagulation panel, qualitative beta human chorionic gonadotropin, aspirin and acetaminophen levels, liver function testing, thyroid function tests, lipase, and alcohol levels. Urine drug screen, urinalysis, cerebrospinal fluid studies, stool studies, and sputum and blood cultures may also be considered. Imaging directed at specific anatomic areas may add clinically relevant information when appropriate; these include a chest radiograph, brain, abdomen and pelvis, or chest computed tomography (CT).

The growing availability of point-of-care analyzers capable of providing data within minutes for ketones, beta-hydroxybutyrate, pH, bicarbonate, and other electrolytes is changing the approach to assessment and management (19,20). The Joint British Diabetes Societies 2011 guidelines suggest the use of either ketone meters or traditional bicarbonate and glucose measurements to guide insulin therapy (8). Although the ADA currently recommends serum beta-hydroxybutyrate as a more specific method over a urine dip test for ketones to screen for DKA, it does not yet recommend bedside analyzers to guide therapy in a hospital setting because of concerns over the precision and accuracy of currently available devices (19,21,22).

Management of Hyperglycemic Crisis in Adults

Goals of treatment include uncovering and managing the underlying cause, replacing fluid volume, resolving ketonemia, correcting acidosis, re-establishing euglycemia, improving mental status, optimizing renal perfusion, repleting electrolytes and minerals, and avoiding complications (Figure 1). During the initial clinical assessment, adequate intravenous access should be established for resuscitation. As stated above, a finger stick blood glucose measurement is a critical first step in the recognition and management of these patients. Electrolytes and venous pH should be checked every 2 hours until the bicarbonate and anion gap have normalized and electrolyte abnormalities are resolved. Critical management tips and pitfalls are listed in Table 2.

Fluids and sodium. Volume resuscitation with 0.9% NaCl infused intravenously at a rate of 15 to 20 mL/kg/h should begin immediately and hydration status should be reassessed hourly. Fluid resuscitation beyond the initial boluses depends on hemodynamics, examination findings,

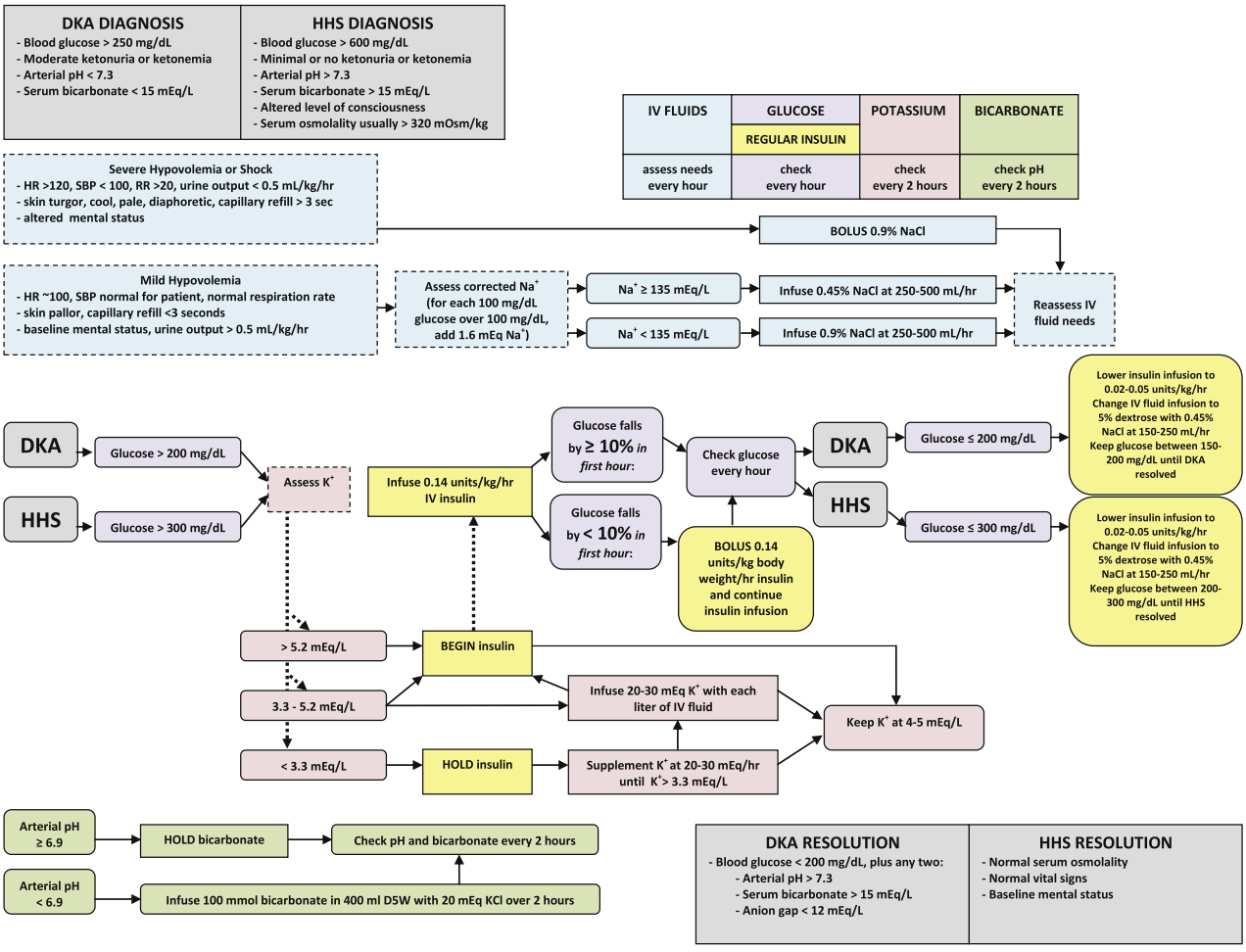

Figure 1. Management protocol for adults with diabetic ketoacidosis or hyperosmolar hyperglycemic state (1,8,13,14,20,26). DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state; HR = heart rate; IV = intravenous; RR = respiratory rate; SBP = systolic blood pressure.

electrolyte levels, and urine output, with severe hypovolemia as an indication for a greater normal saline infusion (1). After improvement in hydration status, corrected serum sodium guides the selection of intravenous (IV) fluids. For hyponatremia, 0.9% NaCl should continue at a rate of 250 to 500 mL/h. If the corrected serum sodium level reveals hypernatremia or a normal sodium level, ADA guidelines recommend the initiation of 0.45% NaCl at 250 to 500 mL/h. Adequate urine output of 0.5 to 1 mL/kg/h is a goal of hypovolemia correction to avoid oliguric renal failure.

The osmotic effect of hyperglycemia introduces intravascular water, resulting in decreased sodium concentration. In 1973, Katz derived what most view as the standard correction of 1.6 mEq/L decrease in sodium concentration per 100 mg/dL increase in glucose (23). The experimental data of Hillier et al. have since shown that 2.4 mEq/L may be a more appropriate overall correction factor and 4.0 mEq/L may be better for glucose concentrations >400 mg/dL (24). Despite this, current guidelines still recommend a correction factor of 1.6 mEq/L. Recent data from pediatric patients with DKA seem to validate this approach (25).

Special fluid considerations for pediatric and elderly patients. In pediatric patients, rapid changes in serum osmolality caused by early over-resuscitation may be a cause of cerebral edema requiring IV mannitol therapy (2). Elderly patients with underlying cardiac or renal disease may require tailored management to address hypovolemia or hypotension, because routine management can lead to acute pulmonary edema that may require positive pressure ventilation.

Insulin. Bedside glucose checks should be obtained hourly in the initial stage, and no less frequently than every 1 to 2 hours while on an insulin infusion. If the patient has a continuous subcutaneous insulin pump, it should be

Table 2. Critical Management Tips and Pitfalls

1. Identify cause of hyperglycemic crisis. Most are caused by infection or inadequate insulin administration. Obtain a thorough history, review of systems, and physical examination to identify precipitants.
2. Avoid hypokalemia. Check potassium before administration of insulin. Insulin may cause hypokalemia and hypoglycemia and lead to iatrogenic cardiac dysrhythmia, seizure, or cerebral edema.
3. Avoid hypoglycemia. Frequently check glucose levels for additional dextrose requirement during insulin administration. In acute, emergent treatment, serum glucose levels should be maintained between 150–200 mg/dL in diabetic ketoacidosis and between 250–300 mg/dL in hyperosmolar hyperglycemic state.
4. Avoid rapid volume correction. Total body losses of 6 to 9 L are typically corrected over a 24- to 48-h period. Rapid fluid shifts may contribute to cerebral edema, and large volumes of normal saline may lead to hyperchloremic metabolic acidosis.
5. Avoid phosphate over-repletion. Hyperphosphatemia is a cause of hypocalcemia. Replete only in patients with skeletal muscle weakness or rhabdomyolysis.
6. Avoid use of bicarbonate. Bicarbonate is only recommended for pH < 6.9. It may delay resolution of ketonemia and contribute to hypokalemia and cerebral edema.
7. Overlap intravenous and subcutaneous insulin by 1 to 2 h. After criteria for resolution of hyperglycemic crisis has been met, do not abruptly halt insulin infusion and begin subcutaneous insulin without an overlap period. Doing so may cause a relapse of hyperglycemic crisis.
8. Frequently re-examine patient and check laboratory values every 1 to 2 h. Infrequent physical examination reassessments and laboratory monitoring may miss early signs of decompensation.

inactivated prior to initiation of treatment. After the initial normal saline bolus, continuous infusion of regular insulin IV should begin at 0.14 units/kg/h (26). Bolus regular IV insulin dosing followed by a lower rate of infusion has been recommended as an alternative; however, equivalence testing revealed no clinically relevant differences in anion gap resolution, rate of change of glycemia, or alteration in IV fluid management with the bolus method (27). If after the first hour of insulin infusion the serum glucose does not decrease by at least 10%, a bolus of 0.14 units/kg of regular IV insulin is administered and glucose is reassessed after 1 hour. The expected rate of decrease in glucose concentration is 50 to 75 mg/dL/h (13).

In DKA, when serum glucose falls to ≤200 mg/dL, the insulin infusion is decreased to 0.02 to 0.05 units/kg/h. At this point, 5% dextrose with 0.45% NaCl should be initiated at a rate of 150 to 250 mL/h and titrated to keep serum glucose between 150 and 200 mg/dL until DKA is resolved (1). In HHS, when the glucose falls to ≤300 mg/dL, the rate of insulin is switched to 0.02 to 0.05 units/kg/h and 5% dextrose with 0.45% NaCl is infused at a rate of 150 to 250 mL/h and titrated to keep serum glucose between 200 and 300 mg/dL until HHS has resolved (1).

Potassium. Total body depletion of potassium caused by emesis and redistribution secondary to dehydration and insulin therapy mandates potassium assessment and replenishment as needed to avoid life-threatening cardiac dysrhythmia (1). Potassium should be monitored every 2 hours during hyperglycemic crisis. If laboratory assessments are delayed, an ECG should be considered to evaluate for hypo- or hyperkalemia while the tests are in process. In a retrospective study of 29 patients with DKA, 82% presented with hyperkalemia or normal potassium levels and 63% developed hypokalemia during the course of treatment (13). When strong clinical suspicion for hypokalemia exists, insulin therapy may need to be delayed until laboratory values for potassium have returned. For serum potassium of <3.3 mEq/L, insulin should be held and 20 to 30 mEq/h of potassium added to IV fluids until the potassium level is >3.3 mEq/L. For levels >5.2 mEq/L, potassium should be held and reassessed at 2-hour intervals. When serum potassium falls between 3.3 and 5.2 mEq/L, 20 to 30 mEq/L of potassium should be added to each liter of IV fluid, with a goal of maintaining serum potassium between 4 and 5 mEq/L.

Bicarbonate. The 2009 ADA guidelines recommend that for an arterial pH of <6.9, 100 mmol of bicarbonate in 400 mL of sterile water with 20 mEq of potassium should be infused over 2 hours. Bicarbonate is not recommended for an arterial pH ≥ 6.9. In 2011, Chua et al. reviewed 44 articles discussing bicarbonate administration in DKA and concluded that bicarbonate administration in the setting of an arterial pH > 6.85 may worsen hypokalemia, delay a decrease in blood lactate and ketonemia, increase the risk of cerebral edema in children, and provide no sustained benefits (28).

Phosphate. The typical phosphate deficit in DKA is approximately 1 mmol/kg. With the exception of patients presenting with severe skeletal muscle weakness or rhabdomyolysis associated with hypophosphatemia, repletion of phosphate is not recommended because there does not appear to be a benefit and hyperphosphatemia may cause severe hypocalcemia (13).

Resolution of DKA and HHS. Criteria for the resolution of DKA include a blood glucose <200 mg/dL and two of the following: serum bicarbonate ≥15 mEq/L, venous pH > 7.3, and calculated anion gap <12 mEq/L. Resolution of HHS is distinguished by normal serum

osmolality, resolution of vital sign abnormalities, and restored mentation.

With resolution of hyperglycemic crisis and after the patient has shown an ability to eat, a subcutaneous long-acting insulin dose is administered. The insulin infusion should overlap this dose by 1 to 2 hours before being discontinued to avoid relapse of hyperglycemia. The dextrose infusion may also then be discontinued. Although there is no specific guideline for frequency of finger stick glucose checks at this stage, checking finger stick glucose measurements every 2 hours in the immediate postinfusion period is prudent to detect hypoglycemia from remaining circulating insulin.

Management of Hyperglycemic Crisis in Infants, Children, and Adolescents

Unique challenges in the clinical evaluation and treatment of hyperglycemic crisis in pediatric patients have resulted in specific pediatric management guidelines (2,29). Obtaining a history may be more difficult in younger patients, and a diagnosis may therefore be missed or delayed. Consideration of a child's larger surface area relative to total body mass and a child's higher basal metabolic rate must be accounted for in fluid and electrolyte management (29). Because of higher morbidity and mortality rates associated with pediatric hyperglycemic crisis, institutional protocols often include pediatric flowcharts to precisely track and guide fluid and electrolyte resuscitation (30).

Cerebral edema, which accounts for 90% of the deaths associated with DKA in children, has traditionally been attributed to osmolality changes during rapid lowering of serum glucose (2,31). Treatment-induced injury is a focus of major concern and debate (32). Recent studies question the significance of osmolality-induced injury and suggest that cerebral hypoperfusion injury may be a predominant cause of cerebral edema that begins even prior to treatment (33,34). The optimal fluid type and rate of administration to treat pediatric DKA is currently under study. A large, multicenter, randomized, controlled study in association with The Pediatric Emergency Care Applied Research Network (PECARN) is gathering data on >1500 pediatric patients in DKA using four treatment protocols with different types and rates of fluid administration, with mental status assessments during treatment and neurocognitive testing 3 months after DKA (35). The results may suggest an optimal fluid management strategy to improve neurocognitive outcomes and reduce the greatest cause of mortality associated with pediatric hyperglycemic crisis.

Disposition

Almost all patients presenting to the emergency department with DKA or HHS will require admission to resolve hyperglycemic crisis and for further investigation and treatment of the precipitating event(s). Patients who are septic, hypoxic, hypotensive, in a state of disseminated intravascular coagulation, persistently tachycardic, severely acidotic (bicarbonate <5 mmol/L or pH < 7.1), ketonemic, or neurologically altered (Glasgow Coma Scale score <12) or have moderate to severe electrolyte abnormalities require admission to an intensive care unit. Concurrent conditions also requiring a higher level of care include pulmonary embolus, MI, and stroke.

Patients with a mild presentation distinguished by clear mentation, normalization of vital signs after volume and electrolyte resuscitation, resolving acidosis, demonstrated closure of an anion gap, with treatment initiated for an underlying cause may be considered for admission to a medical floor. An even smaller fraction of patients presenting with hyperglycemia and mild ketonemia, no emesis, minimal volume loss, and demonstrated adequate liquid and solid intake after fluid resuscitation who do not meet the definition for hyperglycemic crisis on presentation may be considered for a shorter observation admission or possibly discharge if a new insulin regimen can be confidently initiated and outpatient providers are contacted to provide close follow up.

CONCLUSION

Hyperglycemic crisis demands early recognition and prompt initiation of treatment with reassessments and adjustments of the care plan as needed to reduce morbidity and mortality. Although insufficient exogenous insulin and infection are common precipitants, an appreciation of the range of possible causes and sequelae can help avoid missed diagnoses. Emergency physicians should be facile in managing this physiologically complex disease because they more often identify and treat the critical early stages before specialists contribute to patient care. An organized approach to correcting hyperglycemia, fluid balance, electrolyte abnormalities, and normalizing acid-base status favors improved outcomes.

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ARTICLE SUMMARY

1. Why is this topic important?

Hyperglycemic crisis is a common metabolic emergency associated with significant morbidity, mortality, and cost that requires prompt critical care management in the emergency department.

2. What does this review attempt to show?

This article reviews the latest guidelines available for the management of hyperglycemic crisis and describes its associated pathophysiology, clinical presentation, examination findings, risk factors, and differential diagnoses to assist physicians in its diagnosis and treatment.

3. What are the key findings?

An appropriate care plan for hyperglycemic crisis includes identifying and addressing precipitating causes, appreciating the risks of adverse, unintended iatrogenic treatments, and resolving hyperglycemia and electrolyte abnormalities while normalizing acidemia and fluid balance.

4. How is patient care improved?

A concise management algorithm for hyperglycemic crisis is provided to assist the emergency physician in providing optimal care.