Diabetic Ketoacidosis

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Practice Gaps

1. Clinicians should be aware of the etiology and clinical presentation of diabetic ketoacidosis.
2. Clinicians should understand the appropriate management and risks associated with treatment of children with diabetic ketoacidosis.

Objectives

After completing this article, readers should be able to:

1. Understand the etiology of diabetic ketoacidosis (DKA).
2. Understand the basic clinical presentation, diagnostic tests, and management of DKA.
3. Recognize the risks associated with fluid and electrolyte therapy in patients with DKA.
4. Understand the causes of recurrent DKA.

INTRODUCTION

Diabetic ketoacidosis (DKA) occurs when there is a relative or absolute decrease in circulating insulin levels in relation to an increase in counterregulatory hormone levels. In response to this imbalance, normal physiologic mechanisms are exaggerated, resulting in hyperglycemia, hyperosmolality, ketosis, and acidosis. (1) The biochemical criteria for the diagnosis of DKA are hyperglycemia (blood glucose level >200 mg/dL [>11.1 mmol/L]), venous pH less than 7.3 or serum bicarbonate level less than 15 mEq/L (<15 mmol/L), and ketonemia (blood β-hydroxybutyrate concentration ≥3 mmol/L) or moderate or severe ketonuria. (1)(2)(3)

Overall, the most common cause of DKA is new-onset type 1 diabetes mellitus (T1DM). DKA can also be seen in children with T1DM and infection, other intercurrent illness, or inadequate insulin administration. Children with type 2 diabetes mellitus (T2DM) may also present in DKA. High-dose corticosteroids, atypical antipsychotic agents, diazoxide, and immunosuppressive medications have been reported to precipitate DKA in patients without a diagnosis of T1DM. (4)(5)

Treatment of DKA involves careful fluid resuscitation, insulin administration, electrolyte replacement, and close monitoring for signs of cerebral edema. This review focuses on the epidemiology, pathogenesis, diagnosis, management, and

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ABBREVIATIONS

DKA	diabetic ketoacidosis
GCS	Glasgow Coma Scale
PECARN	Pediatric Emergency Care Applied Research Network
T1DM	type 1 diabetes mellitus
T2DM	type 2 diabetes mellitus

morbidity of DKA. We highlight diagnostic criteria, risk factors, treatment, and the risks associated with fluid and electrolyte therapy in these patients.

EPIDEMIOLOGY

Diabetes is one of the most common chronic diseases in the United States. In 2009, at least 192,000 children in the United States had a diagnosis of diabetes, and the population incidence for DKA hospitalizations continues to increase, with 188,965 total admissions in 2014. (6) Approximately 11% of these admissions for DKA were in children younger than 17 years. (7) Despite an overall increase in hospital admissions, both hospital length of stay and mortality have decreased, with mortality decreasing to 0.33%. (7)

Nearly 30% of children with a new diagnosis of T1DM present with DKA and 10% of children with a new diagnosis of T2DM present with DKA. (8)(9) Risk factors for DKA on initial diagnosis are younger age (<2 years), delayed diagnosis, and lower socioeconomic status. (2) In children with known T1DM, the risk of DKA is 1% to 10% per patient year, and risk factors for DKA include insulin omission, previous episodes of DKA, inadequate dosing of insulin, and infection. (2)(9)(10) In recurrent DKA, psychological considerations play a major role, including stress of chronic disease, rebellion against authority, fear of weight gain, and eating disorders, which have all been implicated as contributing factors. Increased risk of recurrence of DKA has also been reported in peripubertal and adolescent girls and children with challenging social situations or limited access to medical services. (2)(9)

PATHOGENESIS

DKA occurs when serum insulin concentrations are inadequate due to an absolute deficiency (as in the setting of progressive pancreatic β-cell failure due to autoimmune destruction in undiagnosed T1DM) or relative deficiency (stress, infection, inadequate insulin intake) in relation to elevated counterregulatory hormone levels (catecholamines, cortisol, glucagon, and growth hormone).

Figure 1 depicts the pathophysiology of DKA. The combination of insulin deficiency and increased counterregulatory hormone levels leads to gluconeogenesis and glycogenolysis with increased glucose production and decreased peripheral glucose utilization. This causes hyperglycemia, hyperosmolality, increased lipolysis, and ketogenesis. When the renal threshold for glucose is exceeded (~170–200 mg/dL [~9.4–11.1 mmol/L]), glucosuria and hyperketonemia cause osmotic diuresis, dehydration, and electrolyte wasting (including sodium, potassium, magnesium, calcium, and phosphate loss). (11)(12) This further stimulates stress hormone production, and if insulin, fluid, and electrolytes are not replaced, then worsening dehydration, metabolic and lactic acidosis, and even death can occur.

The pathogenesis of cerebral edema is incompletely understood. Multiple mechanisms have been suggested, and controversy exists about whether intravenous fluid administration rate and content contribute. Earlier theories suggested that rapid rehydration with hypotonic fluid and resultant fluid shifts due to a rapid decrease in osmolality between the extravascular and intravascular intracranial compartment explained the development of cerebral edema. Newer theories suggest that cerebral hypoperfusion with reperfusion injury, neuroinflammation, and vasogenic edema play a role, as does increased permeability of the blood brain barrier. A recent randomized controlled trial performed at 13 large pediatric centers, the Pediatric Emergency Care Applied Research Network (PECARN) DKA FLUID Study, compared the effects of the rate and content of intravenous fluid administration on neurologic outcomes in children with DKA. (13) In this study, only 0.9% of patients had clinically apparent brain injury, and there was no difference in neurologic outcome between the groups, suggesting that the rate of infusion is not associated with increased neurologic injury. (13) The rate of clinically apparent brain injury has remained static despite improved treatment and monitoring pathways, which suggests that the mechanism is multifactorial and that providers still have much to learn to fully understand this complex pathophysiology.

Demographic factors associated with an increased risk of cerebral edema include younger age (<5 years old), new onset of diabetes, longer duration of symptoms (often associated with severe dehydration), and severe acidosis.

CLINICAL ASPECTS

Signs and Symptoms

The classic clinical signs of DKA include polyuria, polydipsia, polyphagia, and weight loss. A good history and physical examination are necessary to prevent misdiagnosis in children who do not have classic presenting symptoms, and, even with classic signs, inexperienced providers may misdiagnose DKA. Clinical signs may progress rapidly and include vomiting, abdominal pain, dehydration, weakness, and lethargy. Abdominal pain and ileus can result from potassium depletion, acidosis, and poor splanchnic perfusion. Abdominal pain may be severe enough to mimic an acute abdomen in the initial phase of DKA. Dehydration causes tachycardia, delayed capillary refill time, poor skin

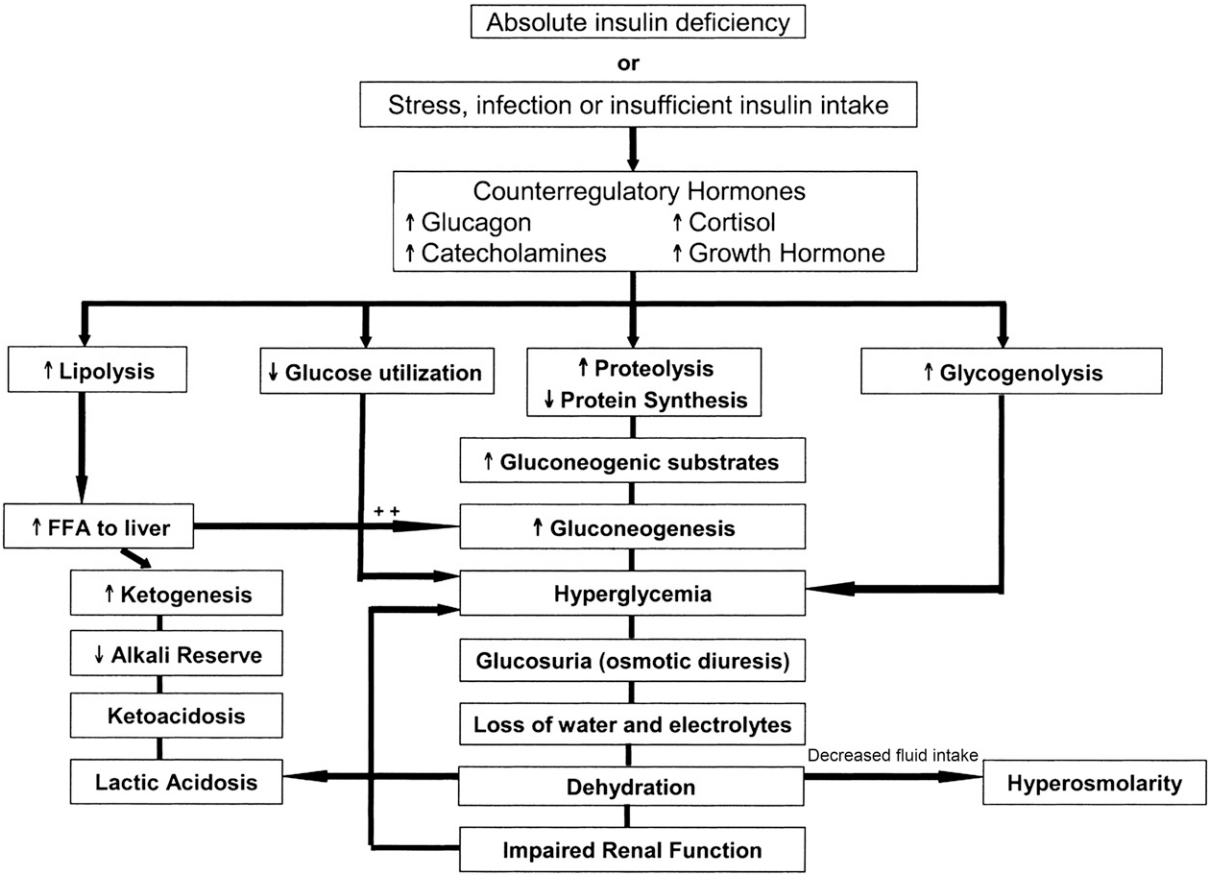

Figure 1. Pathophysiology of diabetic ketoacidosis. (Reprinted with permission from Wolfsdorf JI, Glaser N, Agus M, et al. ISPAD Clinical Practice Consensus Guidelines 2018: diabetic ketoacidosis and the hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2018;19[suppl 27]:155–177.)

turgor, and dry mucus membranes. Ketoacidosis stimulates both central and peripheral chemoreceptors that control respiration, resulting in Kussmaul respiration (rapid fast deep breathing) in an attempt to decrease P_{CO_2} and compensate for the metabolic acidosis. In addition, ketoacidosis may result in a fruity odor to the breath. Despite severe dehydration, children generally maintain their blood pressure, likely due to increased plasma catecholamines and increased release of antidiuretic hormone in relation to high serum osmolality. (2) Eventually, when compensatory mechanisms are overwhelmed, children with severe DKA may present with hypotension, shock, and altered mental status.

The most feared complication of DKA is severe neurologic injury and development of cerebral edema. Warning signs and symptoms of developing brain injury include headache, bradycardia, irritability, increased drowsiness, altered mental status, cranial nerve palsies, new abnormal neurologic signs on examination, hypertension, unresponsiveness, and coma. The Glasgow Coma Scale (GCS) (Table 1) may be used to provide more objectivity when assessing mental status. (14) Recent studies have used multiple scores or a change in GCS score to assess decline in mental status in children with DKA and have shown an association between GCS score and cerebral edema on neuroimaging. (13) However, the GCS is limited because it has not been well validated as a predictor of short- or long-term outcome in pediatric DKA and, thus, should be used in conjunction with a complete neurologic examination. (13)(14)

In addition to the aforementioned history and physical examination findings, it is important to investigate potential precipitants of DKA, including, but not limited to, intercurrent infection, malfunctioning insulin pump, ingestion of medications or substances, and pregnancy.

Laboratory/Diagnostic Findings

Laboratory values necessary for diagnosing DKA are hyperglycemia (blood glucose level >200 mg/dL [>11.1 mmol/L]), venous pH less than 7.3 or serum bicarbonate level less than 15 mEq/L (<15 mmol/L), ketonemia (blood β-hydroxybutyrate concentration ≥3 mmol/L), and ketonuria. (1)(2)(3) The severity of DKA is categorized as mild (pH <7.3 or serum bicarbonate level <15 mEq/L [<15 mmol/L]), moderate (pH

TABLE 1. **Glasgow Coma Scale**

	SCORE					
	1	**2**	**3**	**4**	**5**	**6**
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	NA	NA
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	NA
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion/withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

NA=not applicable.

<7.2 or serum bicarbonate level <10 mEq/L [<10 mmol/L]), or severe (pH <7.1 or serum bicarbonate level <5 mEq/L [<5 mmol/L]). (15)

Serum electrolytes with calculation of an increased anion gap [Na−(Cl+HCO_3)] greater than 12 ± 2 mEq/L is consistent with DKA. Note that this gap is attributable to the presence of serum ketones and not to other etiologies of anion gap metabolic acidosis (such as lactic acidosis, salicylates). Children will have dilutional hyponatremia due to hyperglycemia, and the calculated corrected sodium value should be considered. Corrected sodium is calculated as measured sodium + 2 ([plasma glucose − 100] / 100) mg/dL. Elevated serum osmolality, blood urea nitrogen concentration, and creatinine level are also consistent with DKA. Due to known urinary losses in the setting of glucosuria, serum calcium, phosphate, and magnesium levels are also important to monitor. In addition, an electrocardiogram should be considered in the setting of high serum potassium levels given the associated risk of ventricular arrhythmia.

A complete blood cell count with leukocytosis is commonly seen and by itself may not indicate infection. Cultures or radiographic imaging for the source of infection should be considered if the clinical history or physical examination findings are suggestive.

Findings on laboratory testing that have been associated with the development of cerebral edema are elevated serum blood urea nitrogen level, severe acidosis, and severe hypocapnia. (2) A failure of the corrected sodium level to rise with treatment, or a further decrease in serum sodium level, has been associated with cerebral edema as well. Additional laboratory tests may be warranted as the clinical examination and history findings dictate.

Timing of Initial and Subsequent Laboratory Evaluation

At the time of initial presentation, blood glucose levels, serum electrolyte levels, pH (via blood gas), and the presence of urine or blood ketones will confirm the diagnosis of

TABLE 2. **Example of 2-Bag Technique for Intravenous Replacement and Fluid Requirement**

PLASMA GLUCOSE, MG/DL (MMOL/L)	BAG 1: 0.9% NACL, PARTS	BAG 2: D_{10} + 0.9% NACL, PARTS	FINAL DEXTROSE CONCENTRATION, %
≥250 (>13.8)	1	0	0
200–249 (>11.0–13.8)	1	1	5
150–199 (>8.3–11.0)	1	3	7.5
100–149 (5.6–8.3)	0	1	10
<100 (5.6)	Assess patient	Treat hypoglycemia	

Each intravenous fluid bag should have equal electrolyte levels depending on institutional preference and protocol (eg, 30 mEq/L [30 mmol/L] of potassium acetate + 10 mEq/L [10 mmol/L] of potassium phosphate). The rate of infusion will depend on the calculated maintenance and replacement of the remaining deficit based on institutional protocol. Each child requires specific individual treatment and assessment, and adjustments to treatments should be made based on careful monitoring. D_{10}=10% dextrose, NaCl=sodium chloride.

DKA. Patients presenting in DKA with a new diagnosis of diabetes should undergo additional laboratory testing to assist with the evaluation of their underlying pathophysiology. This testing is inclusive of hemoglobin A1c, thyroperoxidase antibodies, thyrotropin, free thyroxine, tissue transglutaminases, immunoglobulin A, total immunoglobulin A, islet cell antibody, insulin antibody, and glutamic acid decarboxylase antibody.

Serial laboratory testing with hourly capillary blood glucose concentrations and frequent (every 2–4 hours) serum electrolyte, blood gas, blood urea nitrogen, calcium, magnesium, phosphate, and β-hydroxybutyrate levels should be performed. Note that capillary blood glucose levels may be inaccurate in the presence of poor peripheral circulation and severe acidosis, thereby limiting this collection method in measuring extremely high blood glucose concentrations. In these circumstances, capillary samples may need to be cross-checked against venous glucose samples. (2)

Radiologic Findings

Head computed tomography can be used to evaluate the brain parenchyma for radiologic signs of cerebral edema (Fig 2). In some cases, the radiation exposure of computed tomography may outweigh the benefit of the study. Magnetic resonance imaging may be valuable to identify cerebral edema in the setting of acute alteration in mental status for ischemic stroke, dural sinus thrombosis, and other associated neurologic injuries. Imaging is an adjunct to clinical examination findings and should not delay emergency treatment.

TREATMENT

Initial treatment of DKA should follow the guidelines of Pediatric Advanced Life Support. Management should be in a center with expertise in managing pediatric DKA. If a child needing emergency care does not present to a center with this expertise, consultation with an expert in the management of pediatric DKA is strongly recommended. Initial testing as described in the Laboratory/Diagnostic Findings subsection may be necessary, and thorough clinical examination and history are important in determining precipitating factors, estimating the severity of dehydration, and assessing mental status.

After initial life support, a child with DKA should receive care in a unit that has both experienced nurses and physicians trained in the serial monitoring and management of DKA in children and adolescents. Within this care arena, providers should follow a protocolized approach for pediatric DKA management. In addition, to provide the best and safest care, the medical team needs to have access to a laboratory that can provide frequent and timely measurements of biochemical variables. Patients with severe DKA inclusive of prolonged duration of symptoms, compromised circulation, or depressed level of consciousness should be considered for treatment in an ICU (pediatric if available) or in a unit with equivalent staffing and resources. (2)

The overall goals of treatment are to correct dehydration and acidosis and reverse ketosis, with gradual correction of hyperosmolality and hyperglycemia. Initial management includes fluid resuscitation with isotonic fluid. Note that fluid replacement should begin before starting insulin therapy. In children with hypovolemic shock (inadequate tissue perfusion from decreased intravascular volume), fluid resuscitation with isotonic saline (0.9% sodium chloride), and 20-mL/kg fluid boluses should be rapidly infused, with reassessment after each bolus. (2) In children with dehydration without shock, initial fluid resuscitation should begin with an isotonic saline fluid bolus, typically 10 mL/kg over 30 to 60 minutes. Calculation of fluid administration, including replacement from losses and maintenance fluid requirements, should be estimated over 24 to 48 hours, and 0.45% to 0.9% saline, or a balanced salt solution, may be used (Ringer's lactate, Hartmann solution, or Plasma-Lyte). (2)(13) The International Society for Pediatric and Adolescent Diabetes Consensus Statement has been recently modified to reflect that 0.45% to 0.9% saline may be used based on findings from the PECARN DKA FLUID Study. (2)(13) Patients with DKA are typically 5% to 10% dehydrated. The

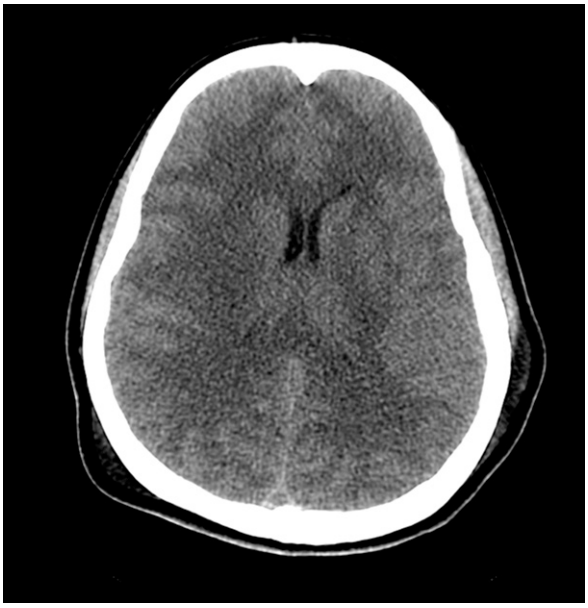

Figure 2. Computed tomographic scan showing effacement of the cerebral sulci consistent with cerebral edema.

mean time to correction of DKA was approximately 12 hours in a study of 635 patients. (16) Thus, although calculations of fluid administration should be made to replace fluid over 24 to 48 hours, most patients will correct before the 24- to 48-hour mark and will be able to restore the remaining fluid deficit via enteral replacement.

As noted previously herein, initiation of insulin therapy should follow initial fluid replacement. Initial insulin infusion rates of 0.05 to 0.1 U/kg per hour are recommended depending on the clinical scenario and degree of hyperosmolality. A continuous insulin infusion should be maintained until resolution of DKA. A small randomized controlled trial in children younger than 12 years showed that low-dose insulin (0.05 U/kg per hour) was comparable with higher dose (0.1 U/kg per hour) in terms of rate of glucose decrease and resolution of acidosis but did not suggest that higher-dose insulin was harmful. (17) The expected rate of decrease of blood glucose is 36 to 90 mg/dL per hour (2–5 mmol/L per hour). Dextrose may be added to the intravenous fluid if the rate of decrease drops faster than expected. Table 2 shows an example of the 2-bag technique for intravenous fluid replacement. If the blood glucose level falls more quickly than expected and DKA has not resolved despite the addition of up to 12.5% of dextrose peripherally, then a reduction in the insulin dose delivered should be considered. When the patient has corrected and is no longer in DKA, has improved sensorium, and is able to tolerate oral intake, he or she should be transitioned to oral food and subcutaneous insulin.

Potassium should be replaced as soon as the patient has urine output and laboratory values confirm that the child is not hyperkalemic. Despite initially normal or elevated levels of serum potassium, children with DKA typically have a total body deficit of potassium with intracellular potassium depletion due to shifts of potassium to the extracellular space and loss due to vomiting and osmotic diuresis. If children are hypokalemic on presentation, potassium should be replaced, and as long as the patient has adequate renal function, rehydration fluid should contain potassium. Depletion of intracellular phosphate is also seen in DKA due to losses from osmotic diuresis, and severe hypophosphatemia should be treated. Bicarbonate administration is not recommended because this has not shown benefit in the resolution of DKA, and bolus administration has been historically associated with worse outcomes. Administration of bicarbonate potentially may cause harm due to paradoxical central nervous system acidosis. Therefore, bicarbonate administration should be reserved for the treatment of severe hyperkalemia or severe acidosis (pH ≤6.9) causing impaired cardiac contractility. (2) Hyperchloremia may develop in children who undergo replacement with fluids containing large amounts of chloride. This can contribute to persistent hyperchloremic metabolic acidosis, large base deficit, and low serum bicarbonate level. Hyperchloremia should be considered in a child with low β-hydroxybutyrate levels with a non–anion gap metabolic acidosis and a low serum bicarbonate level.

Children with severe DKA and children at high risk for cerebral edema should be cared for in an ICU. Children who are severely obtunded or unconscious and cannot protect their airways should be intubated. In most circumstances, intubation should be avoided if possible because pharmacologic sedation and the subsequent elevation of P_{CO_2} in a child with cerebral edema may progress to cerebral herniation. Central venous catheters should be avoided due to the high risk of thrombosis in these patients. If the clinical scenario deems a central venous catheter necessary, it should be removed as soon as the patient status allows. Repeated neurologic examinations (hourly, or more frequently as clinically indicated) should be performed to assess for the development of cerebral edema (new or worsening headache, bradycardia, vomiting, change in neurologic status, new cranial nerve palsies, abnormal pupillary response, hypertension, hypoventilation). (2) Treatment of cerebral edema should be started as soon as there is clinical suspicion and should not be delayed because of neuroimaging. Hypotension, hypoxia, and excessive fluid administration should be avoided. The head of the patient's bed should be elevated to 30°, and hyperosmolar agents (mannitol or hypertonic saline [3%]) should be administered. Hypertonic saline is increasingly used in many institutions; however, controversy remains, and data are lacking to definitively dictate whether mannitol or 3% hypertonic saline is preferable in this patient population.

The mortality from DKA in children is low, and the major cause of mortality and morbidity is cerebral injury. DKA associated with central nervous system complications include neuronal changes resulting from hypoglycemia and hypoxia, dural sinus and basilar artery thrombosis, intracranial hemorrhage, cerebral infarction, and cerebral edema. Note that DKA is associated with a prothrombotic tendency in children. This is due to a combination of altered clotting factor activity and serum hyperosmolarity, which potentiate the risk of thromboembolic events, including the previously mentioned cerebral thrombosis or cerebral infarction. (18)(19)

Clinically apparent cerebral edema occurs in 0.5% to 0.9% of patients with DKA, with high mortality. Alteration in mental status (defined as a GCS score <14) occurs more commonly and has been associated with findings of cerebral

edema on neuroimaging. Neuroimaging studies in children with DKA without overt clinical signs have shown that evidence of cerebral edema occurs more frequently than clinically suspected. The pathogenesis of cerebral edema is incompletely understood, and controversy exists about whether intravenous fluid administration rate and content contribute. Historically, abrupt changes in serum osmolality have been implicated in the development of cerebral edema. (20)(21) Newer theories suggest that factors intrinsic to DKA, including cerebral hypoperfusion with reperfusion injury, neuroinflammation, osmotic shifts, and vasogenic edema, play a role, as does increased permeability of the blood-brain barrier, which can worsen during treatment. (22) Meticulous monitoring of clinical and laboratory response to treatment should be provided with goals to correct rehydration, gradually improve hyperglycemia and hyperosmolality, and reverse acidosis and ketosis. A causal relationship has not been determined between the administration of hypotonic fluid or a rapid reduction in serum osmolality and the development of cerebral edema. However, caution and gradual reduction in hyperglycemia and hyperosmolality are recommended. Additional research is needed to clearly define the pathogenesis of cerebral edema and whether specific treatment interventions contribute to the development of cerebral edema.

Survivors of cerebral edema after DKA have significant morbidity. Even children treated for DKA who did not have clinically apparent neurologic injury during treatment demonstrate memory deficits after recovery, suggesting that subclinical changes may contribute to morbidity. Additional reports suggest that children with altered mental status at presentation without overt clinical or radiologic findings during treatment for DKA score worse on cognitive testing, and structural and functional changes on magnetic resonance imaging persist for up to 6 months after treatment. These data suggest that measures aimed at prevention, including earlier diagnosis in patients with new-onset T1DM and improved diabetes compliance and prevention of recurrence by identifying and addressing precipitating factors, play a key role in morbidity and mortality. (2)(23)(24)

In conclusion, children with DKA should be cared for in a center with expertise in managing these ill patients. Most cases of DKA are seen in children with a new diagnosis of T1DM. Recurrent DKA is more common in children with psychosocial concerns, including difficulty with access to health care, challenging social situations, and poor compliance. Treatment goals are to correct dehydration, acidosis, and ketosis and, if possible, to avoid the development of cerebral edema. Mortality is low but morbidity, especially long-term morbidity, is not well delineated, and the underlying mechanism of neurologic injury requires additional study. Prevention via earlier diagnosis and improved diabetes compliance should be targeted.

Summary

- On the basis of class D evidence and consensus, children and adolescents with diabetic ketoacidosis (DKA) should be cared for in a center experienced in the treatment of DKA.
- On the basis of class A evidence, goals of treatment in children with DKA are to correct dehydration, correct acidosis, and reverse ketosis.
- On the basis of class A evidence, fluid replacement (deficit and maintenance) should be calculated and replaced over 24 to 48 hours.
- On the basis of class B evidence, insulin therapy should start at a rate of 0.05 to 0.1 U/kg per hour after starting fluid replacement.
- On the basis of class B evidence, bicarbonate administration is not recommended except for severe hyperkalemia or severe acidosis with impaired cardiac output.

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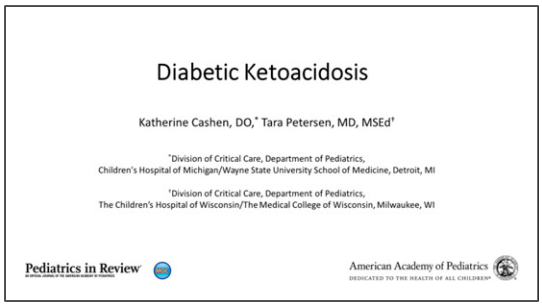

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1. A 16-year-old girl with type 1 diabetes mellitus (T1DM) is seen in the clinic for recurrent episodes of diabetic ketoacidosis (DKA). She is 5 ft 6 in (168 cm) tall and weighs 100 lb (45.4 kg). She is a well-adjusted honor roll student, and her teachers say she is a very pleasant person and gets along very well with her peers. Which of the following is most likely to be associated with her recurrent episodes of DKA?
    A. Fear of weight gain.
    B. Inadequately prescribed dosing of insulin.
    C. Low socioeconomic status.
    D. Misunderstanding insulin administration instructions.
    E. Underlying immune disorder predisposing to infection.
2. A 3-year-old girl with new-onset T1DM is admitted to the hospital with a diagnosis of DKA. On initial evaluation in the emergency department she was assessed to be severely dehydrated. Her parents report that she has been ill for a week. Laboratory studies show mild acidosis, high urine specific gravity, glucosuria, and ketonuria. Which of the following factors is least likely to place this patient at risk for cerebral edema?
    A. Age of 3 years.
    B. Mild acidosis.
    C. New onset of T1DM.
    D. Prolonged nature of her illness.
    E. Severe dehydration.
3. A 10-year-old girl with no significant medical history is brought to the emergency department because of vomiting, abdominal pain, polyuria, polydipsia, dehydration, and weight loss. She is diagnosed as having DKA. In addition to blood glucose, blood ketone, and serum electrolyte levels, which of the following laboratory studies is not recommended as part of the initial routine evaluation of this patient?
    A. Erythrocyte sedimentation rate.
    B. Glutamic acid decarboxylase antibody level.
    C. Hemoglobin A1c level.
    D. Islet cell antibody.
    E. Thyroperoxidase antibodies.
4. A 15-year-old boy, with a history of vomiting and 10% dehydration is brought to the emergency department by ambulance after his parents called 911 when they noticed that he has been progressively becoming more "lethargic" for the past few hours. On arrival at the emergency department he is noted to have clinical signs of dehydration. He is sleepy but arousable and responds to painful stimuli. His initial laboratory evaluation is significant for a blood glucose level of 250 mg/dL (13.9 mmol/L) with serum pH 7.05 and a bicarbonate level of 4 mEq/L (4 mmol/L). A normal saline bolus is started, and a computed tomographic scan of the brain is ordered. Which of the following is the most likely finding to be seen on computed tomographic scan in this patient?
    A. Brain atrophy.
    B. Dural thrombosis.
    C. Effacement of cerebral sulci consistent with cerebral edema.
    D. Intraparenchymal hemorrhage.
    E. Ischemic stroke.

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5. A 14-year-old girl with known T1DM has been noncompliant with her insulin dosing regimen. She is brought to the emergency department in DKA. Bicarbonate therapy should most likely be considered in which of the following clinical and laboratory findings?
    A. Serum pH 7.2, potassium level of 5 mEq/L (5 mmol/L), and normal cardiac output.
    B. Serum pH 7.0, potassium level of 6 mEq/L (6 mmol/L), and decreased cardiac output.
    C. Serum pH 6.7, potassium level of 7 mEq/L (7 mmol/L), and decreased cardiac output.
    D. Serum pH 7.1, potassium level of 4.5 mEq/L (4.5 mmol/L), and normal cardiac output.
    E. Serum pH 7.0, potassium level of 6 mEq/L (6 mmol/L), and normal cardiac output.

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