Current Diagnosis and Treatment of Hyperglycemic Emergencies

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KEYWORDS

• Diabetic ketoacidosis (DKA) • Hyperosmolar hyperglycemic state (HHS)
• Hyperglycemic crisis • Insulin therapy • Electrolyte management

KEY POINTS

- Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are 2 hyperglycemic crises frequently encountered in emergency departments.
- DKA, characterized by hyperglycemia, ketonemia, and anion gap metabolic acidosis, results from absolute or relative insulin deficiency and counterregulatory hormone excess.
- HHS, characterized by hyperglycemia, hyperosmolarity, and profound dehydration without significant ketoacidosis, results from prolonged poor glycemic control and inadequate hydration.
- The management of both DKA and HHS hinges on treatment of precipitating illnesses, fluid resuscitation, and correction of hyperglycemia, acidosis, and electrolyte abnormalities.

INTRODUCTION

Hyperglycemia is a common occurrence in emergency department patients. As the number of new cases of diabetes mellitus increases worldwide, emergency providers are frequently faced with hyperglycemic patients and challenges surrounding their care. DKA and HHS are the most feared and life-threatening hyperglycemic emergencies in diabetes. Both of these diseases are associated with uncontrolled diabetes mellitus and may lead to significant neurologic morbidity and death. Early diagnosis and management in an emergency department is paramount to improve patient outcomes. The mainstays of treatment in both DKA and HHS are aggressive rehydration, insulin therapy, electrolyte management, and discovery and treatment of any underlying precipitating events.

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Emerg Med Clin N Am 32 (2014) 437–452
http://dx.doi.org/10.1016/j.emc.2014.01.004 **emed.theclinics.com**
0733-8627/14/$ – see front matter

EPIDEMIOLOGY

The prevalence and financial burden of diabetes are tremendous and rising. Approximately 10% of the US population lives with diabetes, and approximately 2 million Americans are diagnosed with diabetes yearly.[1] It is projected that by the year 2050, up to 1 in 3 American adults will be diabetic.[2] An estimated 10% of health care dollars are spent treating diabetes and its complications, and 20% of health care dollars are spent caring for diabetics overall; in 2012, the direct medical costs of treating diabetes totaled $176 billion.[3]

The incidence of DKA has been estimated in older studies to range from 4 to 8 episodes per 1000 patient admissions for diabetes.[4] In 2009, DKA accounted for approximately 140,000 hospitalizations.[5] In the United States, DKA accounts for more than $1 billion in hospital costs per year.[6] The incidence of DKA is much higher among young children and persons of lower socioeconomic status. There is often low family income, poor parental support and patient education levels, and less health insurance coverage with decreased access to care, all contributing to poor compliance and high rates of recurrent DKA.[7]

The incidence of HHS is more difficult to quantify because there have been no population-based studies, but it has been estimated to account for approximately 1% of diabetic admissions.[8] This number is likely an underestimation. The mortality in HHS is much higher, however, ranging from 10% to 20%, compared with 1% to 5% in DKA.[8,9]

PATHOPHYSIOLOGY

DKA and HHS are both characterized by hyperglycemia, which stems from insulin resistance or deficiency of insulin secretion from the pancreas. In DKA, the driving force is insulin insufficiency and a subsequent increase in insulin counterregulatory hormones (ICRHs), which prevents the body from metabolizing carbohydrates.[10,11] Insulin normally stimulates the transference of glucose from the bloodstream into tissues of the body, where it is needed for energy, glycogen storage, and lipogenesis. Insulin also inhibits hepatic gluconeogenesis, preventing further glucose production by the body.[12] When insulin is absent, hepatic gluconeogenesis continues, yet glucose cannot move into the cells and instead builds up in the bloodstream. This elevated glucose leads to osmotic diuresis and dehydration.

In DKA, metabolism shifts from normal carbohydrate metabolism to a state of fasting fat metabolism. There is an increase in the aforementioned ICRHs: glucagon, catecholamines, cortisol, and growth hormones.[13] These stress hormones stimulate lipolysis, which leads to free fatty acid oxidation into the ketone bodies, acetone, acetoacetate, and β-3-hydroxybutyrate, the last being the primary contributor to the resultant metabolic acidosis.[8] The body can initially buffer mild ketonemia, and this results in a mild anion gap with a normal blood pH. Once ketonemia reaches excess of the body's limits, however, it begins to spill into urine and causes an anion gap acidosis with a drop in pH and bicarbonate levels.[8] Respiratory compensation ensues with rapid deep breathing, called Kussmaul respirations. Ketonemia further leads to nausea and vomiting, often worsening dehydration.[8] The course of DKA is usually a quick progression, often occurring in hours to days.

DKA occurs more frequently in type 1 diabetes mellitus; however, it can also occur in non–insulin-dependent (type 2) diabetes mellitus. It is growing increasingly common in type 2 diabetes mellitus, which is thought due to an acute halt of insulin secretion by temporary pancreatic beta islet cell dysfunction and temporary insulin resistance. The condition often resolves after treatment of the acute DKA episode, and patients may