Chapter 34

Hyperglycemia, Hypoglycemia, and Acute Diabetic Emergencies

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Hyperglycemia in the ICU

Hyperglycemia is a commonly encountered metabolic disturbance in the ICU. Three major clinical scenarios are patients who are known to have diabetes mellitus (DM), patients with undiagnosed DM, and patients who suffer from stress-induced hyperglycemia. Mechanisms that explain the underlying pathophysiological process include, but are not limited to, insulin resistance, absolute or relative insulin deficiency, impaired glucose metabolism, and the effects of nutritional supplements or stress-induced hormones such as glucagon and corticosteroids. Early research on patients with cardiovascular disease with hyperglycemia suggested that insulin might itself be atherogenic, considering that most patients with diabetes were at high risk for vascular complications. Currently it is believed that hyperglycemia, more than insulin resistance or hyperinsulinemia of diabetes, is the reason for exaggerated inflammatory and oxidative reactions leading to endothelial disruption and cardiovascular damage.

Initial studies on the effects of hyperglycemia on critically ill patients were focused on DM and its effect on the outcome of coronary artery disease and cardiac surgery patients. It was repetitively shown that short- and long-term mortality rates following myocardial infarction are significantly higher when hyperglycemia is present, with or without diagnosed DM. It is now believed...
that hyperglycemia, rather than the presence of DM, influences morbidity and mortality risk. Stress-induced hyperglycemia (SIH) is also associated with poor outcomes and increased rates of congestive heart failure and cardiogenic shock, especially when it is encountered during a perimyocardial infarction hospitalization. Regardless of the cause of high blood glucose (BG) levels, patients with BG levels of 200 mg/dL or more have greater mortality risk at 180 days.

Hyperglycemia has repeatedly demonstrated worsening morbidity and mortality rates in all types of ICUs, even if patients had modest elevations of mean glucose levels during ICU stay. Poorly controlled hyperglycemia during cardiac surgery was associated with higher incidence of deep wound infections and subsequently higher mortality rates in the ICU. Further, hyperglycemia, specifically on the first and second postoperative days, was found to be the single most important predictor of serious infection and complications. In other studies, general surgery and medical ICU patients also displayed increased infection rates with hyperglycemia. Additionally, intravenously administered insulin infusions aimed at keeping patients in normoglycemic ranges independently reduced mortality and deep wound infections in the cardiac surgery population. The Joint Commission and the Surgical Care Improvement (SCIP) Project of the Centers for Medicare and Medicaid now include perioperative BG control in their quality measures for cardiac surgery patients; recently they linked it to pay for performance.

Treatment of stress-induced hyperglycemia (SIH) gained popularity after it was recognized that the presence of hyperglycemia, even without diabetes, contributed to poor outcomes in the ICU. After an acute injury, hyperglycemia is likely to be the result of both decreased glucose uptake due to peripheral insulin resistance and increased endogenous glucose production. Stress-induced hyperglycemia, compared with diabetic hyperglycemia, was shown to indicate a higher risk of poor outcome in hospitalized patients, including increased length of stay and even mortality. Stress-induced hyperglycemia differs from DM, and the long-term management of each will vary significantly. Documenting admission hemoglobin A1C levels will be most useful in differentiating SIH from DM. The American Diabetes Association (ADA) 2010 Standards of Medical Care recommend that BG goal ranges for SIH patients and diabetic patients should differ, since the acute hyperglycemia and mortality relationship might be altered according to preexisting hyperglycemia.

Stress-induced hyperglycemia and gestational diabetes (GD) have similarities. Both SIH and GD are due to a temporary disorder of glucose homeostasis. Patients with GD should be screened regularly, even after pregnancy, for detection of type 2 DM. Patients with SIH during an acute illness should also be considered a population at increased risk for developing diabetes, and ideally their hemoglobin A1C levels should be monitored as follow-up.

After discovery of the adverse outcomes associated with acute hyperglycemia, studies investigated tight glucose control in the ICU. One of the largest and best known studies in this group was published in 2001 by Van den Berghe and colleagues from Belgium. They investigated 1,548 patients, the majority of whom resided in the surgical ICU following cardiac surgery. These patients were randomized to receive tight versus conventional glucose control regimens, where the range for tight or intensive BG control was defined as 80 to 110 mg/dL and the conventional group was treated if BG was more than 200 mg/dL to maintain BG of 180 to 200 mg/dL. The investigators reported a significant reduction in numerous morbidities and in mortality in the intensive BG group. They defined hypoglycemia as BG less than 40 mg/dL and reported low rates of hypoglycemia without adverse effects.

Other randomized controlled studies that followed the first Van den Berghe study failed to show the same mortality benefit, but they consistently demonstrated hypoglycemia as a side effect of tight glycemic control (TGC). These conflicting results of TGC in the ICU led the critical care community to debate what the goal blood glucose target should be during an ICU stay. The largest trial of TGC in ICUs was published as the NICE-Sugar (Normoglycemia
in Intensive Care Evaluation–Survival Using Glucose Algorithm Regulation) trial. The study randomized 6,104 patients in 42 centers to receive conventional versus intensive insulin therapies (IIT). The results indicated that both mortality and incidence of hypoglycemia were significantly higher in the IIT group.

Intensive insulin therapy (ie, tight glucose control) was evaluated in the subgroup of critically ill patients with severe sepsis or septic shock. As the role of corticosteroid therapy was recognized in the Surviving Sepsis guidelines, treatment of septic shock with steroids became more common. One of the side effects of steroid therapy is hyperglycemia, so the COITSS study investigators evaluated effects of IIT in 509 septic shock patients treated with steroids. Compared with conventional insulin therapy, IIT failed to improve in-hospital mortality among these patients. The SepNet study group reported similar results in their 537-patient multicenter randomized trial, in which the investigators assigned patients to receive either conventional or intense insulin therapies and randomized the choice of resuscitation fluids for severe sepsis. The investigators concluded that critically ill patients with sepsis who received IIT were in significant danger of hypoglycemia.

Considering these and other studies, in a 2009 consensus statement, the American Association of Clinical Endocrinologists and the ADA recommended that insulin therapy for persistent hyperglycemia in the ICU be initiated at a threshold BG level of 180 mg/dL or more. Once insulin therapy is initiated, a BG range of 140 to 180 mg/dL is recommended for the majority of critically ill patients. Intravenous insulin infusions are the preferred method of insulin administration in ICU patients, and BG is monitored frequently to prevent hypoglycemic events (Table 1 on page 657).

Recent work in this arena has focused on glucose fluctuations or glycemic variability as opposed to a defined target BG range. Several studies report that glycemic variability is associated with poor prognosis. In vitro and animal studies demonstrate that oscillating glucose is worse than a stable constant high glucose, specifically in activating pathways leading to long-term diabetic complications. Glucose fluctuations increase oxidative stress, increase mitochondrial superoxide production, and cause vascular inflammation. In humans, increased free radicals and endothelial dysfunction and risk of hypoglycemia were reported with higher glycemic variability. Further details of these concerns are beyond the scope of this chapter, but future work aiming therapy at limiting glycemic variability may help define better glycemic management strategies for our patients.

### Table 1.

<table>
<thead>
<tr>
<th>Target range</th>
<th>ICU Patients</th>
<th>Non-ICU Patients</th>
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<tbody>
<tr>
<td>140-180 mg/dL</td>
<td>Intravenous</td>
<td>Subcutaneous</td>
</tr>
<tr>
<td>&lt;180 mg/dL</td>
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**ACCURACY OF METHODS FOR BLOOD GLUCOSE MEASUREMENT IN CRITICALLY ILL PATIENTS**

The conflicting results of studies of hyperglycemia management in the ICUs raised concerns regarding methods of BG measurement in critically ill patients. Most ICUs use nurse-driven insulin protocols. In ICUs, BG levels are usually measured via bedside glucometry devices, which are point of care testing (POCT) devices. Glucometers have several advantages over laboratory testing: they require only a tiny amount of blood, provide fast results to the bedside care provider, and offer a significant reduction in the costs of frequent BG sampling compared with laboratory analysis.
Studies that assessed the accuracy of bedside glucometers in ICU settings revealed conflicting results.\textsuperscript{5-7} Capillary and arterial blood samples for BG levels vary both in POCT devices and during blood chemistry analysis. In most reports, capillary blood samples did not correlate with arterial blood sample measurements. In the majority of studies, capillary blood sample measurements tended to falsely overestimate BG levels; therefore, they can either mask a true hypoglycemic event or lead to increased doses of insulin therapy thereby increasing risk of hypoglycemia.\textsuperscript{58} Other reports suggest that in circulatory shock with hypoperfused capillary beds, increased tissue glucose extraction can result in lower glucose values in capillary than venous samples; in that situation, an underestimation of glucose would cause incorrect diagnosis of hypoglycemia.\textsuperscript{59} When patients require vasopressor support during hypotensive stages of their illnesses, it is prudent to avoid using capillary sampling for glucose monitoring.\textsuperscript{50}

Whole BG levels differ from serum samples because the glucose in plasma is approximately 11% higher than in whole blood given that water content in plasma is higher than in erythrocytes. Therefore, the International Federation of Clinical Chemistry and laboratory medicine protocols recommend a multiplier of 1.11 for conversion of glucose in blood to plasma.\textsuperscript{51} In consideration of this fact, some POCT devices are being integrated with this correction factor so that they “self-correct” to promote more accurate BG results.\textsuperscript{52}

Anemia is reported to cause incorrect glucose measures by glucometry.\textsuperscript{53} Glucometers are programmed to assume a normal hematocrit of 40%, and the internal calculation of BG assumes constant displacement of plasma by red blood cells in the sample. Anemic samples containing fewer red blood cells will have less displacement because of an erroneous ratio concentration. This results in a systematic glucose overestimation for anemic samples, which may mask hypoglycemia. When the hematocrit approaches 30%, this error is likely attenuated.

**HYPOGLYCEMIA IN THE ICU**

In many studies on tight glycemic control, intensive treatment of hyperglycemia in ICUs resulted in frequent iatrogenic hypoglycemia. Hypoglycemia is defined as a BG level less than 70 mg/dL. Although, according to many studies, severe hypoglycemia starts at a BG level of 40 mg/dL, it is established that cognitive impairment begins within the 50 to 70 mg/dL BG range.\textsuperscript{54,55} Besides the most severe complications of hypoglycemia (eg, seizure, death), there are subtle signs of hypoglycemia such as impaired judgment, headache, confusion, and fatigue that might be more challenging to detect in an ICU setting. In multiple trials, hypoglycemia was associated with worse outcomes and higher operative mortality.\textsuperscript{56} Even a single episode of severe hypoglycemia can increase the risk of mortality during an ICU stay.\textsuperscript{57}

Studies of predisposing risk factors for hypoglycemia in critically ill patients have been performed during the past decade. The common risk factors reported are severe sepsis and septic shock, inotropic support, decrease of nutrition without adjustment of insulin infusion, insulin therapy, preexisting diabetes, mechanical ventilation, increased severity of illness, and continuous venous hemofiltration with bicarbonate-based substitution fluid (Table 2 on page 659). Although no studies have looked specifically into type and route of insulin administration and its correlation with hypoglycemia, subcutaneously administered insulin is speculated to cause stacking of repeated insulin doses, potentially creating risk of hypoglycemia. This is why short-acting analog insulin has replaced regular insulin for subcutaneous therapies.\textsuperscript{58} How mechanical ventilation contributes to development of hypoglycemia in the ICU is not clear but it is hypothesized to be due to the effects of sedation by directly or indirectly increasing risk of hypoglycemia. The fact that more women than men have hypoglycemic episodes was attributed to gender, and it was shown that women have a lower counterregulatory threshold for hypoglycemia than men, especially in the elderly population.\textsuperscript{59}
Table 2.

Risk Factors for Hypoglycemia in the ICU

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tbody>
<tr>
<td>Use of continuous venovenous hemofiltration with bicarbonate-based substitution fluid</td>
</tr>
<tr>
<td>Gastric residual removal during enteral nutrition without adjusting insulin administration</td>
</tr>
<tr>
<td>Lowering or stopping nutrition without adjusting insulin administration</td>
</tr>
<tr>
<td>Severe sepsis or septic shock</td>
</tr>
<tr>
<td>Simultaneous use of insulin and octreotide</td>
</tr>
<tr>
<td>Inotropic support</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
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<tr>
<td>Tight glycemic control range</td>
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<tr>
<td>Repeated doses of subcutaneous insulin</td>
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<tr>
<td>History of diabetes mellitus</td>
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<tr>
<td>Renal failure</td>
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<tr>
<td>Liver failure</td>
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<tr>
<td>Medications associated with hypoglycemia: oral antidiabetic agents, β-blocking agents, quinine, aspirin/acetysalicylic acid, trimethoprim/sulfamethoxazole, pentamidine, disopyramide</td>
</tr>
<tr>
<td>Medications associated with hyperglycemia: catecholamines, glucocorticoids</td>
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</tbody>
</table>

Table 3.

Distinguishing Features of Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic Syndrome

<table>
<thead>
<tr>
<th>Diabetic Ketoacidosis</th>
<th>Hyperosmolar Hyperglycemic Syndrome</th>
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</thead>
<tbody>
<tr>
<td>Type 1 &gt; type 2 DM patients</td>
<td>Type 2 DM patients</td>
</tr>
<tr>
<td>Younger patients</td>
<td>Elderly</td>
</tr>
<tr>
<td>Symptoms within 24 hours of ketoacidosis</td>
<td>Symptoms develop over days to weeks</td>
</tr>
<tr>
<td>Abdominal pain and nausea</td>
<td>Prominent mental status changes</td>
</tr>
<tr>
<td>BG &gt;250 mg/dL</td>
<td>BG &gt;600 mg/dL</td>
</tr>
<tr>
<td>Presence of ketones, fruity breath</td>
<td>Osmolarity &gt;320 mOsm/kg</td>
</tr>
<tr>
<td>Ketoacidosis, pH &lt;7.3 (anion gap metabolic acidosis)</td>
<td>Absent or mild ketoacidosis</td>
</tr>
</tbody>
</table>

BG, blood glucose; DM, diabetes mellitus.

ACUTE DIABETIC EMERGENCIES

Hyperglycemic emergencies of diabetes can be divided into diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic syndrome (HHS). The pathogenesis of both of these hyperglycemic disorders lies between complete or relative insulin deficiency and unopposed counterregulatory hormones, most importantly glucagon. Both conditions are characterized by hyperglycemia, which triggers osmotic diuresis and leads to severe dehydration and hyperosmolarity (Table 3 on page 659). The actual level of hyperglycemia is less important than the cause or associated abnormalities of the elevated BG levels. With the initiation of osmotic diuresis, loss of potassium, phosphate, magnesium, chloride, and sodium is promoted. Fluid depletion during DKA is estimated to be around 5 to 7 L, whereas this number is typically higher than 9 L during HHS. Significant volume depletion triggers a renin–angiotensin–aldosterone cascade, causing more potassium depletion. Before initiation of either DKA or HHS treatment, one must measure the potassium level and initiate treatment if hypokalemia is present in order to avoid dangerous worsening of hypokalemia; insulin treatment should be deferred until serum potassium is more than 3.5 mEq/L. In DKA, with the help of glucagon, the release of fatty acids results in ketone production, which in turn causes an anion gap metabolic acidosis via depleting buffer capacity. Hyperosmolar hyperglycemic syndrome differs from DKA with the absence of ketones, likely secondary to the presence of scarce amounts of insulin which prevents ketone production by blocking fatty acid oxidation.

Worldwide, infection is the most common precipitating factor for both DKA and HHS, being responsible for half of the cases. Other predisposing conditions known to trigger DKA or HHS include cardiovascular
events, trauma, surgery, pregnancy, substance abuse, and noncompliance with treatment.

Diabetic ketoacidosis is a triad of hyperglycemia, acidosis, and ketonemia. Diagnostic criteria are defined by the ADA as BG more than 250 mg/dL, pH less than 7.3, serum bicarbonate less than 18 mEq/L, anion gap more than 10, and ketone presence in the serum. The majority of DKA cases occur in patients with type 1 DM; however, patients with type 2 DM, especially African Americans and other ethnic minorities, are now being recognized to suffer from this condition. The presentation of DKA, unlike HHS, usually occurs within 24 hours of ketoacidosis occurrence. Patients complain of polyuria, polydipsia, weight loss, vomiting, and, specifically for DKA, abdominal pain. Physical exams of these patients reflect signs of hypovolemia. Fruity odor in the breath and Kussmaul respirations may be observed. Confusion can also be present, although it is more common with HHS.41

Only about 1% of diabetes-related hospital admissions are attributed to HHS but it has a higher mortality than DKA: up to 15% for HHS versus up to 2% for DKA. Higher mortality is likely due to underrecognition and more frequent presence of severe comorbidities. Hyperosmolar hyperglycemic syndrome can be diagnosed with the presence of hyperglycemia with BG more than 600 mg/dL and hyperosmolarity, in which serum osmolarity is usually 320 or higher. Serum bicarbonate level and pH are expected to be within normal limits. Hyperosmolar hyperglycemic syndrome tends to occur in the elderly population, with presenting symptoms that are very similar to those of DKA but that develop over several days to weeks. Altered mental status triggered by severe volume depletion is usually the reason why patients are brought to a physician’s attention.41,62

All critically ill patients who present to ICUs with altered mental status, severe hypovolemia, hypotension, acid-base disturbances with severe electrolyte imbalance, and suspected association with myocardial infarction or infection should be screened for both DKA and HHS. Diagnosis can easily be made via a POCT device to determine BG levels and the presence of ketones, venous and arterial blood gas analysis, and basic metabolic profile for calculation of serum osmolarity, electrolyte imbalances, and anion gap calculation. Electrolyte imbalances must be diligently managed during the care of both of these hyperglycemic emergencies. Total body potassium is severely depleted because of renal losses, which can be as high as 3 to 5 mEq/kg in DKA and 4 to 6 mEq/kg in HHS. Despite this, the initial potassium value might be within normal ranges because of extracellular potassium shifts under the influence of pH and osmolarity changes. Regardless, before initiation of insulin treatment for either hyperglycemic state, a normal potassium level should be established to avoid development of severe hypokalemia, and additional potassium repletion will likely be required during ongoing therapy. Sodium and chloride are also severely lost during osmotic diuresis. It is well described that hyperglycemia lowers the serum sodium concentration to maintain normal osmolarity. It has been recommended to correct for this by adding 1.6 mEq of sodium for every 100 mg of glucose over the 100-mg glucose level; however, for BG values higher than 400 mg/dL, a correction factor of 2.4 seems more accurate than 1.6.62,63 Depletions of phosphorus, calcium, and magnesium are usually masked by initial hemococoncentration, and they should be expected to become apparent as hypovolemia is corrected. Elevated hemoglobin and leukocytosis can also be present. Mild leukocytosis of 10,000 to 15,000/mm³ can be attributed to stress and dehydration. White blood cell counts of 25,000 and higher should be handled as bacterial infections until proven otherwise.64,65

**MANAGEMENT OF HYPERGLYCEMIC EMERGENCIES**

The treatment goals for both DKA and HHS are correction of hypovolemia, reestablishment of normoglycemia, correction of electrolyte imbalances, and treatment of predisposing factors. Patients with altered mental status, oliguria, severe hypokalemia, hypothermia, or other significant associated comorbidities such as cardiovascular incidents, recent surgery, infection, or stroke should be treated in ICUs during management of these complex disturbances. With suspected diagnosis (positive
insignia via bedside glucometry and urine dipstick) of DKA or HHS, treatment should be initiated promptly with fluid replacement, without waiting for further laboratory results. The rate of volume repletion depends on the patient's cardiovascular status as well as severity of clinical presentation. Metabolic disturbances should be corrected at a more conservative rate than volume replacement since rapid correction can harm the patient. The end point of therapy is resolution of hyperosmolarity (in HHS) and metabolic acidosis (in DKA) with correction of anion gap after proper management of hyperglycemia, hypovolemia, and associated metabolic imbalances.

**Volume Repletion During Hyperglycemic Emergencies**

The initial choice for fluid replacement should be 0.9% sodium chloride (ie, normal saline [NS]). One liter of NS should be initiated immediately with suspicion of DKA or HHS. After clinicians determine a calculated corrected serum sodium level, the choice of crystalloids can be altered. If corrected serum sodium is less than 135 mEq/L, replacement can be continued with NS. But if corrected serum sodium is more than 135 mEq/L, clinicians should consider changing to 0.45% sodium chloride (ie, half-normal saline); by the time the corrected sodium is in high normal range, it needs to be changed to half-normal saline. Fluid replacement should be monitored frequently and tailored individually for each patient. Usually it is recommended to give the first 2 L in 2 hours, the next 2 L in 2 to 6 hours, and an additional 2 L in the following 6 to 12 hours. This algorithm usually replaces 50% of the fluid deficit in the first 8 to 12 hours, and then the rest should be administered within the following 12 to 16 hours. Fluid administration may need to be more judicious in those with renal or cardiac impairment, and these patients must be monitored carefully. Volume replacement alone decreases serum glucose concentration by up to 50 mg/dL by restoring renal perfusion and glycemic diuresis. It is imperative to check BG levels frequently to monitor the rate of correction. Dextrose should be added to replacement fluids when the BG level drops to 250 to 300 mg/dL.

**Insulin Treatment**

Insulin administration increases utilization of glucose in peripheral tissues and decreases hepatic gluconeogenesis by opposing counterregulatory hormones. Fluid administration should always be the first-line therapy, and insulin therapy should be started after initial fluids are given. A dose of 10 to 15 U of regular insulin should be followed by a continuous insulin infusion of 0.1 U/kg/h to achieve a steady decrease in serum glucose of 50 to 75 mg/dL/h. It is imperative that potassium levels be aggressively monitored during this treatment as both fluid and insulin therapy cause rapid decreases in potassium levels and predispose the patient to dysrhythmias.

Although initial volume replacement reduces side effects of the counterregulatory hormones, insulin also helps by decreasing fatty acid production, decreasing ketonemia, and correcting acidosis. Initial serum potassium concentration should be measured before initiation of insulin therapy; insulin administration should be deferred until potassium levels are corrected since insulin treatment can trigger life-threatening hypokalemia from extracellular to intracellular shift. The ADA recommends that patients whose initial potassium levels are between 3.3 and 5 mEq/L should receive potassium at 10 to 15 mEq/L/h in their replacement fluids for at least 4 hours, presuming urine output is maintained.

Continuous low-dose IV insulin therapy (0.1 U/kg/h) continues to be a fundamental therapy for hyperglycemic states, since it allows for a steady decrease in blood glucose along with volume replacement. Intramuscular or subcutaneous administration of regular insulin should be avoided in early treatment since absorption might be affected by severe volume depletion in these hyperglycemic states. Continuous insulin therapy should be maintained until the anion gap is corrected, even after resolution of hyperosmolarity, since it may take longer to resolve than hyperglycemia in DKA states. Therefore, 5% dextrose should be added to replacement fluids to continue insulin infusion; ongoing recovery should be monitored via anion gap measurements.
Morbidity, Mortality, and Complications Associated With Hyperglycemic Emergencies

Among predisposing factors for DKA and HHS, myocardial infarction and infection contribute the most to mortality. Excluding the predisposing causes, cerebral edema due to rapid correction of hyperosmolar states, refractory shock due to severe volume depletion, vascular thrombosis, and superimposed infections can significantly influence the outcome of patients during these diabetic emergencies. Electrolyte imbalances during treatment include hypoglycemia, hypokalemia, and hypophosphatemia, and all can affect morbidity and mortality rates. Mismanaged hyponatremia can complicate treatment with seizures, altered mental status, or life-threatening cerebral edema. Acute respiratory distress syndrome can be observed during management of these states. Although the pathogenesis of acute respiratory distress syndrome with DKA or HHS is not clear, it is believed to be caused by excessive volume replacement. Mucomycosis, a rare opportunistic fungal infection, can complicate recovery from DKA by either pulmonary or rhinocerebral mucomycosis. New onset of fever during the recovery of either hyperglycemic state mandates a thorough examination of sinuses and nasal turbinates, since prognosis of mucomycosis is very poor even with antifungal therapy and debridement.

- Current American Association of Clinical Endocrinologists and ADA consensus statement on hyperglycemia in the ICU: In a 2009 consensus statement, the American Association of Clinical Endocrinologists and ADA recommended that insulin therapy for persistent hyperglycemia in the ICU be initiated at a threshold BG level of 180 mg/dL or more. Once insulin therapy is initiated, a BG range of 140 to 180 mg/dL is recommended for the majority of critically ill patients. Intravenous insulin infusions are the preferred method of insulin administration in ICU patients.

- Risk factors for hypoglycemia in the ICU: Severe sepsis, shock requiring vasoactive agent support, decreases of nutritional input without adjustments in insulin infusion, treatment with insulin, preexisting diabetes, mechanical ventilation, increased severity of illness, and continuous venovenous hemofiltration with bicarbonate-based substitution fluid are the leading risk factors for hypoglycemia in the ICU.

- Diagnostic criteria for DKA: DKA is a triad of hyperglycemia, anion gap metabolic acidosis, and ketonemia with diagnostic criteria defined by the ADA as BG greater than 250 mg/dL, pH less than 7.3, serum bicarbonate less than 18 mEq/L, anion gap more than 10, and ketone presence in the serum.

- The main differences between DKA and HHS and their treatment endpoints: HHS can be diagnosed with BG more than 600 mg/dL and a serum osmolality typically 320 mOsm/kg or greater. Serum bicarbonate level and pH are expected to be within normal limits. Hyperosmolar hyperglycemic syndrome tends to occur in the elderly population, with presenting symptoms that can be traced back over several days to weeks, unlike the early presentation of DKA. Treatment goals for both DKA and HHS include correction of hypovolemia, reestablishment of normoglycemia, correction of electrolyte imbalances, and treatment of predisposing factors. The end point of therapy is resolution of hyperosmolality in HHS and resolution of metabolic acidosis in DKA after proper management of
hyperglycemia, hypovolemia, and associated metabolic derangements.

REFERENCES


