Hyperglycemic Crises

Headache
WEAK
Blurry Eyesight
Cramps
DIZZY
Tired
Nausea
THIRSTY

Hyperglycemic Crises
A NATIONAL EPIDEMIC

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Purpose and Goals

Hyperglycemia occurs from time to time in all people with diabetes. However, at times, hyperglycemia can lead to acute, life-threatening complications known as hyperglycemic crises. This course is designed to educate healthcare professionals about the emergencies associated with hyperglycemic crises, including causes, diagnosis, treatment, and prevention of hyperosmolar hyperglycemic state (HHS) and diabetic ketoacidosis (DKA).

Instructional Objectives

Upon completion of this course, the learner will be able to:
1. Define hyperglycemic crises.
2. Describe the pathophysiology of HHS.
3. List specific risk factors for HHS.
4. Interpret diagnostic findings related to HHS.
5. Outline the treatment practices for HHS.
6. Paraphrase the pathophysiology of diabetic ketoacidosis (DKA).
7. List specific risk factors for DKA.
8. Interpret diagnostic findings related to DKA.
9. Outline the treatment practices for DKA.
10. Relate the nurse’s role in caring for patients with diabetic complications.
11. Summarize the complications of HHS and DKA.
12. Recognize the importance of patient education in diabetes management.

Introduction

Mrs. Canales, a 69-year-old woman, has been a patient of Dr. Reimer for the past 20 years. During that time, he has treated her for hypertension and diabetes, for which she takes sliding scale insulin. Despite these two serious conditions, Mrs. Canales has remained active and independent, even after her husband passed away.

A few days ago, she saw Dr. Reimer for a rash on her leg, which the doctor diagnosed as poison ivy. Mrs. Canales, an active grandmother, had walked with her grandchildren down some forest trails and came into contact with the irritating plant. The doctor prescribed a topical ointment and oral steroid and told Mrs. Canales to follow up with him if she did not see improvement within 1 week.

Mrs. Canales called Dr. Reimer’s office 3 days later and spoke with Jane, the office nurse. Mrs. Canales told Jane that the rash had changed in appearance; although the rash was lighter in color, her leg was now oozing and appeared swollen and “shiny.” While questioning Mrs. Canales about the rash, Jane ascertained that Mrs. Canales also began feeling unwell in the past day. Mrs. Canales reported she has not felt quite like herself and has been unable to drink any fluids, even though she feels thirsty.

Jane asked Mrs. Canales to hold while she spoke with Dr. Reimer. Upon hearing of her status, the doctor quickly picked up the phone and asked Mrs. Canales to check her blood glucose.

A few minutes later, Dr. Reimer told Mrs. Canales that he was calling the paramedics to transport her to the emergency room. When Jane asked the doctor about his decision, he told her he suspected Mrs. Canales was experiencing a hyperglycemic crisis.

The term hyperglycemic crises refers to two acute, potentially life-threatening metabolic complications of diabetes: hyperosmolar hyperglycemic state, or HHS, and diabetic ketoacidosis, or DKA. According to the Centers for Disease Control and Prevention, DKA is responsible for about 500,000 hospital stays per year, and that number is increasing. Most patients diagnosed with DKA are between 18 and 44 years of age, and two thirds have Type 1 diabetes; about 34% have Type 2 diabetes. The incidence of HHS is difficult to determine, due to lack of studies and comorbidities; the number of hospital admissions for HHS is less than 1% of all diabetes-related admissions.

The life-threatening nature of these crises cannot be understated. DKA is the most common cause of death in children and adolescents with Type 1 diabetes. Although mortality of young adults with DKA is less than 1%, mortality of elderly patients and patients with concomitant illnesses is greater than 5%. Mortality attributed to HHS is higher, with rates reported between 5% and 20%.

True mortality data for HHS and DKA are difficult to interpret because of the presence of coexisting disease states in those who suffer hyperglycemic crises. Some sources cite a 15% mortality rate, others 30–40%. Age, degree of dehydration, hemodynamic instability, underlying precipitating causes, and degree of consciousness are powerful predictors of mortality. Although data vary regarding mortality rates for hyperglycemic crises, one point is certain: Before the discovery of insulin in 1922 mortality rates were greater than 90%.

DKA and HHS are complex conditions, and often nurses have difficulty identifying the conditions and discriminating between the two. Because the clinical features of DKA and HHS overlap, many nurses confuse the two conditions and often think they are the same. Although DKA and HHS are similar in many ways, they are two distinct diagnoses with important differences.

Nurses are at the front lines of assessment, treatment, and prevention of both of these serious metabolic disturbances. This course defines these two conditions, explains the diagnostic and treatment differences between them, and offers information for preventive care.
**Hyperglycemia (HHS)**

HHS is a life-threatening condition related to severe hyperglycemia (serum glucose greater than 600 mg/dL). A basic knowledge of the pathophysiology of HHS is needed to be able to recognize symptoms and understand treatments for HHS.

**Pathophysiology**

The pathophysiology of HHS is complex, but a basic understanding is needed if nurses are to identify and treat their patients effectively. The mechanism of action of insulin is key to understanding the pathophysiology of HHS. The ingestion of food triggers a rise in blood glucose, which subsequently triggers a release of insulin. Insulin-sensitive tissues take up the glucose during meals, using the glucose for energy. Between meals, insulin secretion decreases, and glucose uptake decreases in most tissues. The brain and the kidneys, however, take up glucose regardless of how much glucose is in the bloodstream.

A deficiency of insulin causes a rise in serum glucose and an increase in counterregulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. When glucose and hormones increase in the vascular space, an osmotic shift of fluid from the intracellular space to the vasculature takes place to maintain homeostasis. In addition to this fluid shift, osmotic diuresis occurs, which causes electrolyte imbalances, particularly sodium and potassium deficiencies. For this reason, illness and other physiologic stressors that cause dehydration exacerbate the hyperosmolar, hyperglycemic state. In HHS, total body water deficits can reach 9 L, or 15% of a person’s total body weight.

In HHS, acidosis does not take place. The reason for this is not clear, but it is believed that the body has enough insulin to prevent the breakdown of fats, even though not enough insulin is present to aid peripheral tissues in the use of glucose. Normal pH is an important factor that distinguishes HHS from the other hyperglycemic crisis, DKA.

**Risk Factors and Causes**

HHS occurs more frequently in older adults. The average age of onset of patients with HHS is 60 years old, although with the increase in incidence of Type 2 diabetes the cases of HHS in young people may increase. Nursing home residents and older adults who live alone or without a caregiver are at increased risk due to advanced age and comorbidities that predispose them to dehydration.

HHS is most commonly occurs in patients with Type 2 diabetes, although people with Type 1 can suffer from HHS. In fact, patients with new onset Type 2 diabetes are at increased risk for HHS because they are not aware that they need to monitor their serum glucose. HHS can also occur as a result of other illnesses, such as pancreatitis and stroke. In general, conditions that predispose a person to reduced insulin activity or reduced fluid intake (i.e., dehydration) place people at risk for HHS; infection, diarrhea, vomiting, and fever are common ailments that can induce dehydration and subsequent HHS. The elderly are also at risk because their impaired thirst mechanism leaves them vulnerable to dehydration.

Most cases of HHS result from acute febrile illnesses, such as infection. In fact, more than 60% of HHS cases are caused by infections, such as pneumonia and urinary tract infections. Other conditions that may trigger HHS include burns, Cushing syndrome, heat stroke, congestive heart failure, and renal insufficiency. Certain medications can also increase the risk for HHS, including chemotherapy agents, corticosteroids, furosemide (Lasix), phenytoin (Dilantin), mannitol, and conventional and atypical antipsychotics, among others. Total parenteral nutrition can also place a patient at risk for HHS.

Disorders that impair communication, mental function, and mobility increase the risk for dehydration. For this reason, the elderly, who often suffer a reduced thirst drive, and those with cognitive impairment, who may not be able to request fluids, are at increased risk for HHS. Bedridden patients are also at risk for HHS because their fluid intake is restricted.

**Signs and Symptoms**

HHS typically develops over a period of days or weeks. People with HHS can experience dry mouth and extreme thirst (except perhaps in the elderly) because of the profound dehydration related to the condition. Patients may have warm, dry skin and experience mild confusion, lethargy, polydipsia, polyuria, visual changes, and weight loss. Symptoms can mimic cardiovascular accident and include seizures, sensory deficits hemiparesis, and aphasia. Patients may also be comatose.

**Diagnosis**

To diagnose HHS, symptoms are reviewed and stat labs are drawn and analyzed. Obtain the following labs: serum glucose; plasma osmolality; basic metabolic panel, which includes electrolytes, blood urea nitrogen (BUN), and creatinine; urea nitrogen; complete

<table>
<thead>
<tr>
<th>Lab</th>
<th>HHS</th>
<th>DKA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum glucose</td>
<td>&gt; 600 mg/dL</td>
<td>&gt; 250 mg/dL</td>
</tr>
<tr>
<td>Plasma osmolality</td>
<td>&gt; 320 mOsm per kg</td>
<td>Variable</td>
</tr>
<tr>
<td>Serum ketones</td>
<td>Minimal to absent</td>
<td>&gt; 5 mEq/L</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>&gt; 15 mEq/L</td>
<td>15–18 mild DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10–14 moderate DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 10 severe DKA</td>
</tr>
<tr>
<td>pH</td>
<td>&gt; 7.3</td>
<td>7.25–7.3 mild DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7–7.24 moderate DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 7 severe DKA</td>
</tr>
<tr>
<td>Anion gap</td>
<td>Variable</td>
<td>&gt; 10 mild DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 12 moderate DKA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 12 severe DKA</td>
</tr>
</tbody>
</table>

Table 1: Diagnostic Labs for HHS and DKA
blood count with differential; serum acetone; and arterial blood gas. The doctor may also order a hemoglobin A1c to assess the patient’s blood glucose over time.

Hemoglobin and hematocrit concentrations may be elevated falsely because of hemococoncentration; anemia should be suspected in a patient with a normal hematocrit on examination. Electrolyte disturbances may be present, particularly regarding potassium and sodium. Pseudohyponatremia is common due to the osmotic effect of glucose drawing water into the vascular space, causing an artificially low sodium. Serum potassium may be elevated due to an extracellular shift caused by insulin deficiency. However, total body potassium likely will be low. See Table 1 for critical lab values related to HHS.

Remember that often another event, such as a heart attack or an infection, can cause the hyperglycemic crisis. Therefore, other diagnostic tests may be performed to identify the triggering condition. Cardiac enzymes may obtained to rule out myocardial infarction. Creatine phosphokinase (CPK) should also be checked to rule out rhabdomyolysis.

Blood cultures should be collected to rule out bacteremia. Urinalysis is helpful to reveal specific gravity for hydration status and provide ketones and glucose levels. If the triggering event is a urinary tract infection, a urinalysis can confirm the etiology of the crisis. Gross proteinuria suggests the presence of underlying renal disease, which may be the triggering condition.

If the patient presents unconscious, a lumbar puncture may be needed to collect cerebral spinal fluid and analyze it for cell count, glucose, and protein. A culture of the fluid may be performed to rule out a central nervous system infection. Toxicology studies also may be indicated.

Imaging studies are often ordered, such as chest radiograph (CXR) to screen for pneumonia or an abdominal series if abdominal pain or vomiting is present. Keep in mind that the initial CXR may be falsely negative for pneumonitis due to the state of severe dehydration. A CT scan of the head may be indicated in those with neurologic changes.

The Meaning Behind the HbA1c

Hemoglobin A1C (HbA1c) is a measure of the average plasma glucose concentration over a period of time. A HbA1c may provide information about the patient’s overall compliance with diabetes management in the past few months. It can also help the physician trace the origin of the hyperglycemic crisis.

HbA1c was discovered in 1958 and was associated with diabetes in 1969. In 1976, experts proposed using the HbA1c values for monitoring degrees of control of glucose metabolism in patients. In general, the higher the HbA1c value, the higher the average serum glucose level has been over the past 120 days or so. Two individuals that have the same average blood sugar can have HbA1c values that differ by as much as 1%, but in general the healthy range is 4% to 5.5%. These percentages correlate with serum glucose levels. For example a HbA1c of 10% correlates with an average blood glucose of 275 mg/dL over the past 120 days.

The following list shows the average blood sugar and its equivalent HbA1c.

<table>
<thead>
<tr>
<th>HbA1c%</th>
<th>Glucose mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>100</td>
</tr>
<tr>
<td>5</td>
<td>135</td>
</tr>
<tr>
<td>6</td>
<td>170</td>
</tr>
<tr>
<td>7</td>
<td>205</td>
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<td>8</td>
<td>240</td>
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<td>9</td>
<td>275</td>
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<td>10</td>
<td>310</td>
</tr>
<tr>
<td>11</td>
<td>345</td>
</tr>
<tr>
<td>12</td>
<td>380</td>
</tr>
<tr>
<td>13</td>
<td>415</td>
</tr>
</tbody>
</table>

Laboratory results may differ depending on the analytical technique used (electrophoresis, chromatography, immunoassay), the age of the subject, and biological variations among individuals.

Treatment

Although severe cases of HHS are treated in the intensive care unit, some mild and moderate cases can be treated in nonintensive settings. Treatment of HHS in the adult patient requires correction of dehydration, hyperglycemia, and electrolyte imbalances. Underlying disease processes that may have caused the HHS should be addressed.

The most common lethal errors related to HHS treatment are failure to manage the patient’s airway and inadequate fluid resuscitation. Airway management is a top priority in severe cases of HHS. If the patient is comatose, intubation may be necessary. In the absence of significant heart or renal failure, fluid resuscitation with an isotonic sodium chloride solution, preferably through a central line, should occur, with 1–1.5 L infused during the first hour of treatment. Subsequent fluid choices depend on the patient’s hemodynamics, electrolyte balance, and urinary output. When the patient’s glucose reaches 200 mg/dL, fluids should be changed to D5NS to avoid hypoglycemia. The goal of fluid replacement is half the estimated volume deficit in the first 12 hours of therapy, with the remainder of the fluid volume being replaced in the second 12 hours.

IV insulin infusion is also indicated, though not until after fluid resuscitation has taken place since insulin will exacerbate the hyperosmolar state and further disrupt electrolyte imbalances. Nurses should follow facility protocols regarding insulin infusions. A low-dose insulin infusion of 0.1 units/kg/hour decreases serum glucose by 50–75 mg/dL/hour. If the serum glucose does not decrease by at least 50 mg/dL from the initial value within the first hour, the infusion should be checked to ensure it is properly infusing.

Potassium levels must be addressed to avoid cardiac arrhythmias. Potassium can be added to the fluid replacement to prevent hypokalemia that results from hemodilution.

Frequent reevaluation of lab work must be done while treatment is in progress. Serum glucose should be checked every hour, and electrolytes and blood gases should be evaluated every 2–4 hours. Nurses should be especially watchful for fluid overload, especially in elderly patients and those with concomitant congestive heart failure or renal dysfunction.

Diabetic Ketoacidosis (DKA)

Although similar to HHS, DKA has some distinct differences. Understanding those differences begins with the pathophysiology of the disorder.

Pathophysiology

The mechanism of insulin is the same in DKA as in HHS with an important distinction. In DKA, as in HHS, the body does not have enough insulin to drive glucose into cells for use as fuel. However, in DKA, unlike in HHS, the body burns fat for fuel. An acid called ketones, the byproduct of fat breakdown, builds up in the blood and urine, causing an acidotic state. This ketoacidosis is a primary difference between DKA and HHS.
The hyperglycemic state induces diuresis, which causes fluid losses of about 6 L, resulting in dehydration. However, a fluid shift does not occur in DKA, as it does in HHS, so serum osmolality is not affected. The fluid loss associated with DKA causes electrolyte disturbances, particularly hyperkalemia.

**Risk Factors**

Although DKA can occur in people with Type 2 diabetes, people with Type 1 diabetes have an increased risk of developing DKA. As with HHS, infection is the most common precipitating cause of DKA. Other causes include trauma, myocardial ischemia, surgery, and pancreatitis. People with diabetes who are noncompliant with insulin therapy are also at risk for DKA.

**Signs and Symptoms**

Unlike HHS, which develops over days or weeks, DKA has an acute onset. Signs and symptoms usually begin within 24 hours of the insulin deficiency. Patients experience polydipsia and polyuria, and their breath may have a fruity smell, which is a result of circulating ketones. The patient may experience hypotension, arrhythmias, hyperventilation, and headache. Neurologic symptoms include confusion, drowsiness, lethargy, stupor, or coma.

**Diagnosis**

Diagnostic lab work is the same for DKA as in HHS with a few differences. In the majority of patients with DKA, amylase levels are elevated, usually due to nonpancreatic sources, such as the parotid gland. Lipase may also be elevated in DKA. Therefore, a complete metabolic panel is typically drawn.

Ketonemia usually takes longer to correct than hyperglycemia. Direct measurement of beta-hydroxybutyrate (beta-OHB) in the blood is the preferred method for monitoring DKA and has become more convenient with the development of bedside meters capable of measuring whole-blood beta-OHB. Institutions are moving away from the nitroprusside method that measured acetoacetic acid and acetone because it does not detect the most prevalent beta-OHB, which during therapy, is converted to acetoacetic acid, which may lead clinicians to believe that ketosis has worsened.

**What Is the Anion Gap?**

One differing aspect between HHS and DKA is the anion gap. In DKA, the patient will have an increased anion gap. So what is the anion gap? The anion gap is a direct measure of the difference between the positively charged ion (cation) sodium (Na+) and the sum of the negatively charged ions (anions) bicarbonate (HCO₃⁻) and chloride (Cl⁻), expressed as the following equation:

\[
\text{Na}^+ - (\text{HCO}_3^- + \text{Cl}^-)
\]

A normal result is 12 mmol/L. If the result of the equation is greater than 12, it is indicative of an anion gap acidosis. Conversely, if the result is less than 12, it is deemed a nonanion gap acidosis.

**Treatment**

As in HHS, treatment for DKA in the adult patient is aimed at correcting hypovolemia and electrolyte imbalances by fluid and electrolyte replacement; restoring hemodynamic stability; correcting hyperglycemia; and maintaining normal glucose metabolism. In addition, eliminating acidosis is a priority treatment in DKA. Also prevention of complications, identifying comorbidities, and frequent monitoring are essential.

**The Nurse’s Role**

Initial interventions include establishing lines for medication and fluid administration. Because insulin has a dedicated line, and patients will often need electrolyte replacement along with fluids, antibiotics, and other medications, nurses will likely need multiple lines. Nurses will place one or more large-bore peripheral IVs, or the physician may insert a central venous access device. In severe dehydration, a central venous pressure (CVP) line may be started. If continuous blood draws are anticipated, an arterial line should be inserted.

Draw diagnostic labs and place the patient on a cardiac monitor immediately. Insulin therapy, correction of acidosis, and volume expansion decrease serum potassium concentrations, which can cause cardiac arrhythmias. A Foley catheter is placed to monitor urine output and the patient’s response to fluid therapy.

After initial interventions have taken place, nurses need to continuously assess their patients and follow through with necessary interventions. Evidence-based practice dictates that, in both DKA and HHS, labs should be drawn every 2 to 4 hours to monitor serum electrolytes, glucose, BUN, creatinine, osmolality, bicarbonate, and venous pH. If you have a baseline arterial blood gas, and the patient is hemodynamically stable, you can feel comfortable in switching to venous blood gases since venous pH is only 0.02–0.03 units lower than arterial pH.

Monitor blood glucose levels by performing hourly blood glucose checks, if the continuous blood glucose monitoring is not available. Patients who are edematous, in shock, or on vasopressors often have inaccurate fingersticks, so glucose monitoring with blood from an arterial line may be needed. False high blood glucose values can be found in people with low hematocrits, high bilirubin, and severe lipidemia. False low values are found with high hematocrits and hypoxia. Remember, if the point of care measurement does not correlate with the patient’s clinical status, send a blood sample to the lab for analysis. If blood glucose levels decrease too rapidly after administration of IV insulin, a condition called relative hypoglycemia may occur. Symptoms include diaphoresis, tremor, or pallor.

If too much fluid has been administered too quickly, especially in the presence of renal or heart failure, the patient may experience signs of overload, including jugular vein distention, pulmonary crackles, dyspnea, peripheral edema, tachypnea, and tachycardia with a bounding pulse present. If you note signs of fluid overload, notify the physician, who may order a change in fluids or a diuretic.

Throughout treatment, carefully monitor electrolytes. Clinical signs will alert you to the patient’s electrolyte status. Insulin brings glucose and potassium back into the cells, which can result in a sudden decrease in serum potassium. If the patient is hypokalemic, look for nausea and vomiting, diaphoresis, and muscle weakness and cramps, paralysis, ectopic beats, hypotension, flattened T wave and presence of U wave on the ECG monitor, fatigue, lethargy, increased sensitivity to digoxin, and shallow respirations. IV potassium may need to be administered to correct the potassium deficit.

If the patient has an elevated potassium level, look for tachycardia, bradycardia, a peaked T wave and ectopic beats on the ECG monitor, hypotension, nausea and vomiting, diarrhea, intestinal colic, hyperactive bowel sounds, paresthesias, or muscle cramping.

If the patient has a low sodium level, look for nausea and vomiting, diarrhea, hyperactive bowel sounds, tachycardia, hypotension, lethargy or stupor, confusion, seizures, muscle wasting, and coma. If the patient’s sodium...
level drops below 120 mEq/L, initiate seizure precautions.

Because serum magnesium levels are poor indicators of total body magnesium, in the presence of hypokalemia a low magnesium level should be presumed and treated unless the patient is in renal failure. Symptoms of low magnesium mostly relate to neurological changes, such as irritability, tetany, delirium, and convulsions.

Phosphate levels should also be monitored. Phosphate replacement may be considered when the patient’s serum phosphate level falls below 1 mEq/L and when cardiac and skeletal muscle weakness and respiratory depression are a concern. Because phosphate replacement can cause hypocalcemia with no signs or symptoms of tetany, serum calcium levels should also be monitored.

Monitor respiratory status. Metabolic abnormalities can affect the respiratory system profoundly. Continuous pulse oximetry and respiratory rate monitoring is helpful in addition to cardiac monitoring. Vital signs also should be continually monitored. Notify physician if respiratory distress, hypotension, severe bradycardia or tachycardia, or other cardiovascular abnormalities occur.

Patients with profound neurological deficits should receive hourly neurological assessments. Hypovolemia can cause neurological impairments, so observe your patient for signs of low volume, such as flushed or dry skin, tenting of the skin, dry mucous membranes, and sunken eyes. Monitor the patient for polyuria, oliguria, tachycardia, and hypotension. Measure intake and output carefully and record your patient’s weight daily.

If the patient experiences a change in level of consciousness, keep the patient NPO (nothing by mouth). Because a change in level of consciousness is often accompanied by a loss of controlled swallow, elevate the head of the bed or keep the patient side-lying to prevent aspiration. The doctor may order insertion of a nasogastric tube if refractory vomiting occurs and the risk for aspiration is increased. Patients who are alert and oriented may also be kept NPO to better control blood glucose levels. In these cases, water and ice chips are usually given.

After the patient’s blood glucose has stabilized for 12 to 24 hours, the insulin infusion may be stopped and subcutaneous (SQ) insulin started. In a patient with Type 1 diabetes, infused insulin is cleared from the body in as little as 5 minutes. Basal insulin is often given approximately 3 hours before the infusion is discontinued to provide continuous insulin coverage. SQ rapid-acting insulin must be given 30–60 minutes before the IV insulin is discontinued. Be sure when transferring patients from intensive care to general medical units that the insulin doses are clear to the receiving nurse to prevent medical errors. Be aware that discontinuing enteral feedings can cause hypoglycemia in some patients; be sure to check blood glucose hourly if enteral feedings are stopped suddenly.

DKA and HHS pose a risk to patient’s skin, too. Hyperglycemia depresses or stops phagocytosis, which is the body’s ability to engulf bacteria and other foreign bodies. For this reason, skin care is of utmost importance. The patient will most likely be restricted to bed, and if small wounds are not prevented, the chances of infections are heightened greatly.

### Insulin Tip

Consider the effectiveness of insulin before you administer SQ doses. Insulin sensitivity can be calculated by adding up the patients total daily insulin requirements and dividing that amount into 1,700 for patients with Type 1 diabetes or into 1,500 for patients with Type 2 diabetes. Using this formula, one unit of insulin can be expected to drop blood glucose approximately 30 points.

### Complications

The most common complications of hyperglycemic crises are treatment related and include hypoglycemia, hypokalemia, and hyperglycemia (if SQ insulin is not coordinated properly with the cessation of IV insulin). Some patients can develop hyperchloremia as a result of fluid replacement.

Because of the massive fluid losses related to HHS, hypotension with acute circulatory collapse is a common cause of death. Along with hypotension, the severe dehydration of HHS leads to hyperviscosity of the blood, which can cause thromboses and disseminated intravascular coagulation (DIC). If no contraindications are present, preventative low-dose heparin should be started.

Cerebral edema is a serious complication that occurs mainly in children and teens, although some cases have been reported in young adults. Cerebral edema usually occurs as a complication of DKA, but some fatal cases have been associated with HHS. The mechanism of cerebral edema is not clear, but it is thought to occur as a result of rapid lowering of glucose levels, which in turn causes a rapid drop in plasma osmolarity. Brain cells, which trap osmotically active particles, absorb water and swell during rapid rehydration. Children who present with DKA and have low carbon dioxide (CO2) and high BUN should be monitored for signs of cerebral edema, such as headache, lethargy, seizures, and alternating levels of consciousness. An MRI can confirm the presence of cerebral edema.

If cerebral edema is suspected, mannitol or a hypertonic solution may be ordered. Treatment for seizures should avoid phenytoin, which inhibits the release of endogenous insulin and has been associated as a triggering factor of HHS. Death usually occurs due to uncal herniation, and it may occur so rapidly that papilledema is not found. Mortality rates may be as high as 90% in patients with cerebral edema. Avoid administering bicarbonate unless the child’s blood pressure cannot be maintained because of severe acidosis or if blood potassium is high enough to affect the heart. Of patients diagnosed with cerebral edema, 57% will recover completely, 21% will survive with neurologic sequelae, and 21% will die.

Fever is not normally present in DKA unless there is underlying infection. However, a rare but peculiar complication called malignant hyperthermia–like syndrome has occurred in obese African American teenagers. Although rare, nurses should monitor for signs of hyperthermia, even if the patient has not received anesthesia. If the underlying cause of HHS was something that produces fever, such as infection, antibiotics should be administered immediately after the appropriate cultures are performed.

The rapid changes in fluid levels may occasionally result in respiratory distress, particularly in cases of HHS that is occurring due to pancreatitis, myocardial infarction, pulmonary emboli, or pneumonitis. The rapid rehydration integral to the treatment of hyperglycemic crises may cause pulmonary edema. Nurses should continue to monitor pulmonary status, and, if respiratory failure occurs, mechanical ventilation may be needed.

Acute kidney injury may occur when dehydration is so severe the kidneys are compromised. Symptoms of acute kidney injury include confusion and edema. In a few cases, dialysis may be necessary if fluid resuscitation is ineffective in restoring kidney function.

### Patient Education

Preventing hyperglycemic crises is easier than curing it, and patient education is key to prevention. Before you begin educating your patients on diabetes and general health
management, assess their background knowledge first. Ask them what they know about diabetes, blood glucose monitoring, insulin, and symptoms of hypoglycemia. Assessing what they know first will guide you in your education efforts so you can fill in the gaps in their knowledge.

Teach as you go. When in the room performing an assessment, ask your patients what time they take their medications at home and whether they eat a bedtime snack. Ask them if they can recognize when they have a low blood sugar and how they treat it. Before meals, ask them to demonstrate to you how to perform a fingerstick glucose test. Gently correct them if they are performing the test incorrectly. Discuss appropriate food choices with them, and refer them to resources for diabetic diets, such as the American Diabetes Association website.

Discharge planning is vital for diabetic patients. Studies show that if a referral to a doctor is made before discharge, the likelihood that the patient will keep the appointment increases. In some cases, transportation to the appointment may be needed for the patient to comply. Case managers and social workers can help with this process.

Make sure your patients know that their blood sugar may not return to “normal” for 4–6 weeks after the crisis. Help them devise a plan to manage their blood glucose monitoring schedule and meet targeted goals of blood glucose levels. Recognition of hypoglycemia and hyperglycemia should be emphasized.

In cases where the HbA1c is greater than 6.5%, make sure that a referral to a doctor is arranged. For patients whose HbA1c is 5.7–6.4%, discuss lifestyle interventions, such as weight loss or exercise, and let them know of their risk for hyperglycemic crises and need for close monitoring. The use of illicit drugs, particularly cocaine, has been shown to increase the chance of readmission with DKA. Include any referrals to counseling or an addiction treatment program if illicit drug use is suspected.

Discuss the importance of diabetes management in the presence of illness. Because even a mild illness, such as a cold, can cause an increase in serum glucose, patients should prepare for “sick days” when they need to monitor their serum glucose more closely.

The following guidelines can help patients avoid a hyperglycemic crisis during times of illness:

- Keep a sick-day notebook and record your blood sugar and ketone levels. Also keep a list of emergency numbers in the notebook for easy reference.
- Check your blood sugar every 3–4 hours. If your sugar is above 250 mg/dL, check your urine for ketones.
- Drink plenty of noncaloric fluids, such as water and diet sodas. Avoid caffeine.
- Maintain your normal meal plan, if possible. If you cannot eat regular meals, try to maintain your normal calorie intake by eating regular (not sugar-free) gelatin, crackers, broth, juice, and applesauce.
- Check your temperature regularly.
- Continue taking your insulin, even if you cannot eat. The body fights illness by releasing hormones, which can increase glucose levels. Always check with your doctor first to see what is right.
- If you have vomiting and diarrhea for more than 6 hours; moderate to large amounts of ketones in your urine; a blood sugar higher than 180 dm/dL or lower than 70 mg/dL; or if you have been sick for a couple days and aren’t getting better, call your physician.

Some patients transition from the hospital to a skilled nursing facility after discharge to adjust to their new regimen. Nurses at these facilities should reinforce the educational concepts related to diabetes management. Educating patients and caregivers at all levels of care will help decrease the incidence of hyperglycemic crises.

Putting It All Together

So what happened to Mrs. Canales, who was introduced at the beginning of this course? After arriving at the emergency department, a blood glucose test revealed her serum glucose was 688. Her vital signs were blood pressure 100/60, pulse thready at 108, respiratory rate 22, O2 sat 93%, and temperature 101.2°F. The nurse drew labs, including a complete blood count, a complete metabolic panel, and blood cultures. Arterial blood gases were also taken, showing a pH of 7.34, pCO2 of 32, HCO3 of 20, and a pO2 sat 88%. A Foley catheter was placed and a urine sample sent to the lab. A ketone dipstick revealed an absence of ketones. Slight discoloration with the leukocyte estrase strip was noted.

Mrs. Canales was arousable, but she seemed to be in a daze, staring into the corner and then falling back to sleep. When the nurse asked Mrs. Canales to state her name, she appeared startled and jumped.

While performing her assessment, the nurse noted redness, warmth, and swelling to Mrs. Canales’s right lower extremity. The nurse marked the area of redness with a pen to show the area of concern. Both of Mrs. Canales’s legs had healing marks and scratches, and her right posterior calf had an open sore, about the size of a quarter, that was oozing clear, serous secretions. Pedal pulses were present, but her right foot appeared shiny. The skin on Mrs. Canales’s back was red and flakey, and her lips were dry, her eyes were sunken, and the skin on her forearms was tenting.

When the lab results came in, they showed an elevated white blood cell count, an increased hemoglobin and hematocrit, a potassium of 3.6, a sodium of 149, a creatinine of 1.4, and a serum osmolarity of 338. Preliminary blood cultures revealed gram positive cocci. A CT scan was negative, and a portable CXR showed no acute infiltrates.

The nurse and emergency department team determined the patient had HHS. The doctor ordered IV fluids and insulin, and Mrs. Canales was transferred to the intensive care unit for treatment of HHS and right lower extremity cellulitis.

Later tests showed her HbA1c was elevated, and Mrs. Canales was diagnosed with Type 2 diabetes. After transfer to the general med-surg floor, the nurses routinely educated Mrs. Canales about her insulin schedule, discussing actions and side effects of the medication. The diabetes educator visited Mrs. Canales to discuss diabetes management, so another occurrence of hyperglycemic crisis could be avoided.

Upon discharge, Mrs. Canales was armed with educational materials and a new glucose meter, and her follow-up appointments with Dr. Reimer were scheduled. Mrs. Canales was forewarned that it may take several weeks for her blood glucose level to stabilize, and she was reminded to monitor her blood glucose carefully and follow her doctor’s prescribed regimen. The hospital’s case manager set up home health visits to assist Mrs. Canales with wound care and ensure compliance with her diet and medications.

Quick action and critical thinking helped Mrs. Canales avert more serious consequences of HHS. Minutes are crucial during hyperglycemic crises, and nurses are relied on to provide evidence-based care and follow through with patient education. Understanding the pathophysiology of each type of crises and their related treatment will help nurses treat a crisis that is already underway and educate their patients to help them avoid these life-threatening conditions.
Resources

For more information on hyperglycemic crises and underlying diabetes, visit the following websites:

American Dietetic Association: http://www.eatright.org


American Diabetes Association: http://www.diabetes.org

References and Suggested Readings


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