Emergency Management of Hypoglycemia and Hyperglycemia in Diabetic Patients

*See online here*

There are two common hyperglycemic emergencies encountered in diabetic patients; namely, diabetic ketoacidosis and hyperosmolar hyperglycemic state syndrome. Diabetic ketoacidosis is responsible for more than 110,000 hospital admissions per year in the United States. The estimated mortality of diabetic ketoacidosis is 2%. The hyperosmolar hyperglycemic state has a mortality rate of 20%. Because of the hyperglycemic crisis, every future doctor should have the essential information about these emergencies.

**Evaluation of Hyperglycemia**

Patients with diabetic ketoacidosis present with severe hyperglycemia, metabolic acidosis, and increased total body ketone concentration. Hyperosmolar hyperglycemic state syndrome is characterized by severe hyperglycemia, hyperosmolality, dehydration, and altered mental status without significant ketoacidosis.

All patients with severe hyperglycemia should undergo assessment and stabilization of their airway, respiratory and circulation. Their hemodynamic status should be assessed and stabilized. Any patient with an altered mental status should receive a bolus dose of naloxone to reverse the potential effects of an opioid overdose.
If the patient is in a coma, intubation should be considered. When intubation is considered, succinylcholine should be avoided as it is associated with hyperkalemia. Patients with diabetic ketoacidosis usually have hyperkalemia; however, the total potassium content of the body is decreased.

During the immediate assessment of hyperglycemic patients, all patients should be put on oxygen, oxygen saturation should be assessed, and cardiac monitoring should be initiated. A common, yet fatal, cause of diabetic ketoacidosis and hyperosmolar hyperglycemic state syndrome is the myocardial infarction; therefore, a 12-lead electrocardiogram is needed.

A finger-stick glucose test is the first step in the management of a patient in an altered mental state. Once the diagnosis of hyperglycemia is confirmed, further management is indicated. During the initial evaluation, one should obtain a complete medical history and perform a thorough physical examination. A complete blood count, basic metabolic panel, urinalysis, and venous or arterial blood gas should be ordered. The most common causes of hyperglycemia include infection, infarction ‘stroke or myocardial’, pregnancy complications, drug abuse including cocaine, and lack of insulin therapy in a diabetic patient.

Serum effective osmolality should be assessed in patients with severe hyperglycemia and dehydration. Cardiac markers, such as troponin and CK, should be evaluated to exclude the possibility of myocardial infarction.

**Therefore, the initial evaluation of hyperglycemia in diabetic ketoacidosis patients can be summarized in the following:**

- Secure airway, breathing, and circulation.
- Administer naloxone and thiamine to all patients with altered mental status.
- Obtain finger-stick blood glucose, measure peripheral oxygen saturation, perform cardiac monitoring, perform ECG, and assess vital signs.
- Obtain blood samples for a complete blood count, arterial or venous blood gases, and a metabolic panel.
Treatment of Hyperglycemic Crisis

If the patient is critically ill, the following treatment regimen should be followed:

- Administer normal saline bolus dose of 1 L followed by 500 ml/hour.
- Administer insulin regularly in a bolus dose of 0.1 units per kg, and then start continuous infusion at the rate of 0.1 units per kg per hour.
- If serum phosphate levels are below 1.5 mg/dL, start K2PO4 at the rate of 0.5 ml/hour.
- If the pH is below 6.9, bicarbonate should be administered.

Patients with diabetic ketoacidosis, who are not critically ill, should receive intravenous fluid therapy according to the degree of dehydration, and insulin therapy should be started at the rate of 0.1 units per kg per hour without a bolus dose. Bicarbonate and phosphate should not be given to this group of patients.

Potassium replacement therapy might be indicated in patients in a hyperglycemic crisis. The following points summarize the current recommendations for potassium replacement therapy in patients with diabetic ketoacidosis:

<table>
<thead>
<tr>
<th>Serum potassium &gt; 5.3 mEq/L</th>
<th>No additional potassium, but re-check after one hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum potassium 4.0 - 5.3 mEq/L</td>
<td>Add KCL 10 mEq/L/hour</td>
</tr>
<tr>
<td>Serum potassium 3.5 - &lt; 4.0 mEq/L</td>
<td>Add KCL 20 mEq/L/hour</td>
</tr>
<tr>
<td>Serum potassium &lt; 3.5 mEq/L</td>
<td>Hold insulin and add 20 to 60 mEq/L/hour and initiate continuous cardiac monitoring</td>
</tr>
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Most patients in a hyperglycemic crisis need to be admitted to the hospital for inpatient management and are rarely discharged home from the emergency department.

Hypoglycemia Emergencies

Hypoglycemia is defined as a blood glucose level below 60 mg/dL. It is diagnosed after identification of the three components of the Whipples triad that entails:

- Symptoms consistent with hypoglycemia
- Low plasma glucose
- Relief of the symptoms after raising the serum glucose

Epidemiology

- Severe hypoglycemia is associated with significant morbidity and is responsible for 3% of the diabetes-related deaths among the diabetic population.
- It is more common among elderly diabetic females.

Etiology

Oral anti-diabetic drugs

- Metformin, alpha-glucosidase inhibitors, and thiazolidinediones typically do not cause hypoglycemia. Sulfonylureas and meglitinides increase insulin secretion and can cause hypoglycemia. Because the half-life of most of the sulfonylureas
is 14 to 16 hours, the risk of hypoglycemia is quite high in patients taking these drugs.

Exogenous insulin or endogenous insulin

- Exogenous insulin is another common cause of hypoglycemia. It can be given in error or an attempt to self-harm.
- Insulinoma is a tumor of the beta-island cells of the pancreas which is characterized by an elevated level of endogenous insulin and hypoglycemia.
- Poor dietary habits, such as missed, delayed or inadequate meals may enhance the effects of antidiabetic drugs and thus trigger hypoglycemia. This may be worsened by conditions such as malabsorption syndromes.

Alcohol

Hormone deficiency

- A lack of counter-regulatory hormones, such as cortisol, glucagon, and epinephrine, leads to the inability to respond to low glucose levels.

Pathophysiology of hypoglycemia

- Low glucose from the above causes lead to the activation of the central and autonomic nervous system; this leads to low glucose within the body’s system.
- The brain depends on glucose supplied to it as it does not contain cells that can make glucose; thus, neuroglycopenia quickly sets in and presents with confusion, lack of concentration, irritability, hallucinations, coma and maybe death.
- The body responds by activation of the sympathoadrenal system triggering the onset of features, such as sweating, palpitations, tremulousness, and anxiety.

Hypoglycemia unawareness

- It represents a state of reduced sympathetic and neuronal response to hypoglycemia and thus reduced warning signs that can alert the patient of hypoglycemia.
- This state arises from autonomic neuropathy that is even more common amongst diabetics.
- It may raise the chances of cerebral complications of hypoglycemia.

Treatment of Hypoglycemia

The management of hypoglycemia is largely **dependent on the severity and on whether the patient can tolerate oral intake or not.**

Mild hypoglycemia (self-treated)

Most patients are able to eat; thus, 15 grams of simple carbohydrates should be administered orally followed by a snack made up of complex carbohydrates.
Severe

This includes patients who are unable to tolerate oral intake. Treatment involves the establishment of intravenous access and administration of dextrose 50% in water as a single ampule and then re-evaluated to check if the symptoms have resolved and the blood glucose levels are above 60 mg/dL.

Patients who do not have intravenous access should receive glucagon 1 mg subcutaneously or intramuscularly which must be followed by oral glucose.

If the patient’s symptoms have resolved and he or she is no longer hypoglycemic, the patient should be instructed to eat a full meal; otherwise, the treating doctor should repeat the previously mentioned algorithm.

If the patient’s symptoms and blood glucose levels are not corrected after two rounds of this algorithm, **continuous infusion of dextrose should be initiated.**

Disposition of Hypoglycemic Patients

The **patient can be discharged home** from the emergency department if the episode of hypoglycemia was:

- Isolated
- Completely and rapidly reversed without the need for continuous dextrose infusion
- Accidental
- And not caused by an oral hypoglycemic agent that has a long half-life, such as sulfonylureas or long-acting insulin

In addition to the following criteria, the patient should have an uneventful 4-hour observation period at the emergency department and has no comorbid conditions that might affect the intake of medication and food. Also, the patient understands the nature of the hypoglycemic episode, why it happened, and how to prevent it in the future.

It is important that the person can monitor blood glucose levels at home and has a close follow-up appointment with a primary care provider. If the patient is discharged home from the emergency department after meeting all of the above criteria, the insulin dose that he or she usually takes should be reduced by 25% for the first 24 hours.

References


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