



## Hyperglycemic Crisis: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State

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### ABSTRACT

The two most severe metabolic consequences of diabetes mellitus (DM) are diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemia state (HHS). Type 1 and type 2 diabetics are both susceptible to these illnesses. Hyperglycemia, ketone body production, and metabolic acidosis are symptoms of DKA. Hyperosmolality, high blood glucose increase, and little to no ketosis are signs of HHS. Despite significant advancements, it has been difficult to come to a consensus on the diagnostic criteria and course of therapy for both disorders. Another difficulty is that there is a large overlap between these two extremities of the hyperglycemia crisis continuum. A thorough biochemical and clinical patient evaluation, along with prompt diagnosis and therapy, has long been recognized as being essential for symptom relief. Intravenous insulin, fluid replacement, and concurrent management of the triggering causes all form the cornerstones of therapy. Large-scale research that contributes to the definition of how hyperglycemic crises should be handled is lacking globally. A thorough analysis of the pathogenesis, diagnosis, and treatment of DKA and HHS will be provided in this article.

**Keywords:** Diabetic ketoacidosis, Hyperglycemic hyperosmolar state, Ketone bodies, Metabolic consequences.

### INTRODUCTION

#### Hyperglycemic Crises

Uncontrolled diabetes mellitus is a metabolic emergency known as a hyperglycemic crisis that can cause serious morbidity or even death [1]. Hyperglycemic crises are severe, acute, metabolic complications of diabetes that include:

- Hyperosmolar hyperglycemia state
- Diabetic ketoacidosis [2]

These illnesses necessitate immediate medical attention since, if neglected, they can result in catastrophic complications like coma or even death [3].

Even though DKA and HHS are sometimes treated as distinct conditions, they are just two points on a spectrum of hyperglycemic emergencies brought on by poorly managed diabetes.

- In both type 1 and type 2 diabetic individuals, DKA and HHS can occur.
- While HHS is more frequently documented in adult and elderly patients with type 2 diabetes (T2D), DKA is more common in young persons with type 1 diabetes (T1D).
- Features of the two illnesses with ketoacidosis and hyperosmolality may coexist in many people [4].

#### Definitions

##### Diabetic ketoacidosis

The biochemical trio of hyperglycemia, ketonemia, and large anion gap metabolic acidosis makes up DKA.

##### Hyperglycemic hyperosmolar state

HHS has been used to replace the words “Hyperglycemic hyperosmolar non-ketotic state” and “hyperglycemic hyperosmolar non-ketotic coma” to emphasize that:

- The nitroprusside technique can be used to detect mild to varied levels of clinical ketosis in the hyperglycemic hyperosmolar condition.
- Changes in consciousness can frequently exist without coma [5].

#### Pathophysiology

The primary cause of HHS and DKA is inadequate insulin action. A rise in the levels of counter regulatory hormones such as growth hormone, catecholamines, glucagon, and cortisol accompanies the lack of insulin action. The two elements lead to hyperglycemia [6].

Another way to conceptualize DKA and HHS is as occurring on a spectrum of disease symptoms. Absolute insulin insufficiency and deep ketosis and acidosis, or DKA, are at one extreme of the range. Patients with type 1 diabetes are more likely to experience DKA because they have absolute insulin insufficiency due to  $\beta$ -cell death. The other extremity of the spectrum is severe hyperglycemia in the absence of acidosis and ketosis [6].

Patients with type 2 diabetes who still generate enough endogenous insulin to prevent ketosis but not enough to regulate hyperglycemia are more likely to experience this. As the parallel suggests, patients may present with different signs of both conditions. For instance, a DKA patient might have used enough insulin to inhibit ketosis to some extent, but their level of hyperglycemia may still be

rather high. Depending on their ability to manufacture insulin and the degree of concomitant conditions like dehydration, patients with HHS may also exhibit variable degrees of mild acidosis and ketosis [6].

Hyperglycemia results from a lack of glucose utilization in insulin-dependent tissues, including muscle and fat. An insulin shortage causes this. Insufficient insulin also causes a rise in hepatic gluconeogenesis, which in turn causes hyperglycemia. This is a shared mechanism in HHS as well as DKA [6].

Without the ability to use glucose, the body has to find other sources of energy in order to survive. The absence of insulin not only causes hyperglycemia but also accelerates the breakdown of triglycerides in adipose tissue into free fatty acids, which then go to the liver, where they are transformed into the keto acids acetone, acetoacetate, and  $\beta$ -hydroxybutyric acid. The unopposed counter-regulatory hormone action brings further increases in the liver's synthesis of glucose and the breakdown of triglycerides [6].

Unrestrained ketone body creation can result in a significant spike in the production of ketoacids. When there is a significant increase in the generation of ketoacids, metabolic acidosis occurs, which leads to DKA. In HHS, there is still enough insulin present to impede the onset of metabolic acidosis by suppressing ketosis [8].

Another typical finding in DKA and HHS is dehydration. Glycosuria occurs when blood glucose levels are higher than the renal threshold ( $\sim 180$  mg/dl). Osmotic pressure causes uncontrolled diuresis as a result. Patients often complain of polyuria and polydipsia prior to this. Significant electrolyte loss—particularly potassium depletion—may ensue. Excessive dehydration and volume constriction may exacerbate hyperglycemia [8].

## Diagnosis

### *Signs and symptoms*

Compared to HHS, DKA develops more quickly. DKA can sometimes occur within a short period of time after the precipitating cause [7].

Classic hyperglycemia symptoms, such as polyuria, polydipsia, weakness, and alterations in mental status, are present in both metabolic diseases. Additionally, the patient's breath may have a fruity odor. Additionally, patients with HHS and DKA frequently exhibit dehydration-related symptoms, including dry mucous membranes, poor skin turgor, tachycardia, hypotension, and increased capillary refill with severe dehydration. Unconsciousness may result from DKA if it continues to worsen and is not treated [8].

### *Laboratory findings*

Patients with suspected DKA or HHS should have their BG, blood urea nitrogen, serum creatinine, serum ketones, electrolytes, anion gap, osmolality, urine ketones, and arterial blood gases evaluated in the initial laboratory evaluation [9].

Most individuals with DKA who present had plasma glucose levels of 14 mmol/L or above. Meanwhile, the majority of T1D patients with such a plasma glucose level do not experience ketoacidosis. On the other hand, patients with a plasma glucose level below 14 mmol/L may have ketoacidosis. A plasma glucose level of 34 mmol/L or above is arbitrarily one of the diagnostic requirements for HHS since hyperglycemia is typically more severe than it is in DKA. The primary osmole that causes the hyperosmolar condition is glucose [9].

The formula for calculating the elevated serum osmolality is  $(2 \times \text{serum Na}) + \text{serum glucose}$ , where normal values are 290 (SD 5) mmol/kg water. Blood urea nitrogen is freely permeable into and out of the intracellular compartment; hence, it is excluded from the computation of effective osmolality [1,19]. To diagnose HHS, the osmolality must be higher than 320 mmol/kg. However, elevated osmolality in DKA is not unusual. HHS in isolation, will have a pH higher than 7.30, and DKA will have a pH of 7.30 or less. Unless information on oxygen transport is required, measurements of pH and bicarbonate levels can be made using venous blood. It must be kept in mind that mixed acid-base problems cannot be identified in venous blood in the absence of arterial blood gas readings [20]. Although a milder type of DKA may appear with a bicarbonate level between 15 and 18 mmol/L, a lower pH is typically linked to a fall in bicarbonate to 15 mmol/L or less in that condition [9].

Ketone levels in the blood and urine are always moderate to high in less severe DKA. HHS cases may also have trace levels. The use of a reagent strip and a reflectance meter allows for the bedside measurement of blood-hydroxybutyric acid levels [9].

## Treatment

Dehydration, hyperglycemia, ketoacidosis, and electrolyte deficiencies must be adequately corrected for DKA and HHS treatments to be effective [10].

In general, treating dehydration, hyperglycemia, electrolyte abnormalities, and ketone bodies are the main objectives of treatment for managing hyperglycemic crises. DKA and HHS require about 10 to 18 hours and 9 to 11 hours, respectively, to control. Patients admitted with HHS require more intense care and must be watched for the causes of the condition or other complications because HHS is more severe than DKA and is associated with a higher fatality rate [11].

### *Fluid therapy*

Intravenous (IV) fluids are a crucial component of managing hyperglycemic crises. Expanding intravascular volume, restoring renal perfusion, and reducing insulin resistance are all effects of IV fluid therapy alone. This is accomplished by lowering levels of circulating counter-regulatory hormones [12].

The recommended solution is isotonic saline (0.9% NaCl), which is administered initially at a rate of 500 to 1000 mL per hour for the first two to four hours. A study comparing two IV fluid regimens with sodium chloride and lactate ringers revealed no discernible difference in the amount of time it took for DKA to resolve but that the lactate ringers group's time to treat hyperglycemia was noticeably longer [13].

The rate of normal saline infusion should be decreased to 250 mL/h or changed to 0.45% saline (250–500 mL/h), depending on the serum sodium concentration and level of hydration, after intravascular volume depletion has been rectified [14].

In order to continue administering insulin until ketonemia is rectified while preventing hypoglycemia, replacement fluids should contain 5 to 10% of dextrose once the plasma glucose level drops below 200 mg/dL (11.1 mOsm/L) [15].

Since many HHS patients may see an improvement in their mental status changes or their resolution with the treatment of fluid deficiencies, adequate fluid resuscitation is particularly crucial in HHS therapy [16].

## Potassium

Both metabolic acidosis and inadequate insulin cause potassium to migrate outside of cells. In DKA, serum potassium levels may be normal or increased, but individuals nonetheless have a depleted body. Similarly, HHS is linked to a decrease in total body potassium levels because of insulin deficiency and elevated plasma osmolality. The total-body potassium deficit has been estimated to be ~3–5 mEq/kg [17,18].

To help potassium return to the intracellular compartment, insulin therapy reduces serum potassium levels. To maintain a level of 4 to 5 mEq/L, potassium replenishment should begin when the serum concentration is below 5.2 mEq/L. Most individuals only require the administration of 20 to 30 mEq of potassium per liter of fluids; however, patients with acute or chronic renal failure need lower dosages. Insulin delivery may cause severe symptomatic hypokalaemia in individuals with admission hypokalaemia and serum potassium levels below 3.3 mEq/L, leading to muscular weakness and an increased risk of cardiac arrhythmias. When treating such patients, insulin therapy should be postponed until the potassium level is above 3.3 mEq/L and potassium replacement should start at a rate of 10 to 20 mEq/hr [19].

## Bicarbonate

It has not been demonstrated that routinely giving bicarbonate to DKA patients can enhance clinical outcomes, including the length of hospital stays, time to resolution, or mortality [20-22].

Clinical guidelines advise the administration of 50 to 100 mmol of sodium bicarbonate as an isotonic solution (in 400 mL of water) until pH is > 6.9, even though no studies have examined the effects of bicarbonate therapy in patients with severe acidosis due to the potential risk of decreased cardiac contractility and arrhythmias. Bicarbonate therapy is not advised for patients with mild DKA who have a pH of >7.0 or who have HHS [24,25].

## Insulin Dosage Plans

The mainstay of DKA therapy is insulin injection, which decreases serum glucose by preventing endogenous glucose synthesis and promoting peripheral consumption. Additionally, insulin blocks the formation of glucagon, lipolysis, and ketogenesis, which reduces the development of ketoacidosis.

Regular insulin is continuously infused intravenously as the preferred treatment. Most treatment plans include giving a 0.1 unit/kg body weight bolus of insulin, followed by 0.1 unit/kg/hr of continuous insulin infusion until blood glucose is less than 200 mg/dL. The dose is now decreased by half (0.05  $\mu$ /kg/hr), the rate is regulated between 0.02 to 0.05  $\mu$ /kg/hr, and 5% dextrose is added to keep the glucose levels between 140 and 200 mg/dL until the ketoacidosis resolves [14].

Numerous studies have shown that the subcutaneous delivery of rapid insulin analogs (lispro and aspart) every 1 to 2 hours is a time-efficient alternative to the IV infusion of conventional insulin [26,27].

Patients are given an initial bolus of 0.2 to 0.3  $\mu$ /kg, followed by 0.1 to 0.2  $\mu$ /kg every 1 to 2 hours, until their blood glucose level is less than 250 mg/dl, respectively. Once DKA has resolved, the dose is subsequently cut in half to 0.05  $\mu$ /kg every hour or 0.01  $\mu$ /kg every two hours [28].

Scheduled subcutaneous insulin enables safe and efficient treatment without the need for intensive care unit care in the emergency room and step-down units. Rapid-acting insulin can also be administered intramuscularly; however, this method is typically more painful than subcutaneous injection and may put patients who are taking anticoagulant therapy at risk for bleeding [29].

It is crucial to remember that individuals with arterial hypotension, severe and complicated DKA, or HHS should not utilize rapid-acting subcutaneous insulin analogs [29].

## Transition to Maintenance Insulin Regimen

The resolution of DKA is indicated by venous pH > 7.30, a normal anion gap, a serum bicarbonate level 18 mEq/L, and a glucose level of less than 250 mg/dL. HHS resolution is achieved when effective serum osmolality < 310 mOsm/kg, glucose level  $\leq$  250 mg/dL (13.8 mmol/L) in a patient who has recovered mental alertness and is regaining mental status [14,15].

The rapid discontinuation of insulin may produce rebound hyperglycemia, ketogenesis, and repeated metabolic acidosis because of the short half-life of insulin (10 minutes). Before stopping the IV insulin infusion, subcutaneous basal insulin (NPH, glargine, detemir, and degludec) should be administered for at least two hours [30].

When utilizing basal insulin analogs (glargine, detemir, degludec), which have a longer delay in commencement of action than NPH insulin, earlier initiation 3 to 4 hours before withdrawal of insulin drip should be taken into consideration. One randomized controlled experiment compared the effects of IV insulin and subcutaneous glargine co-administration to IV insulin alone immediately after the start of treatment for DKA. According to the closure of the anion gap, patients who received glargine had a marginally shorter time to DKA resolution and a shorter hospital stay, but these differences were not statistically significant [31].

Another study discovered that early glargine delivery during treatment reduced the occurrence of rebound hyperglycemia following the cessation of insulin drip [32].

To start a significant insulin dose of 0.5 to 0.6 units/kg (half as basal and half as bolus) may be begun [14].

Patients who have difficulty swallowing should only receive basal insulin, or they can stay on an insulin drip until they are able to eat. Patients with known diabetes can resume their previous insulin regimens; however, if there is a history of frequent hypoglycemia or considerably uncontrolled hyperglycemia before admission, as indicated by admission HbA1c, an adjustment of the prior regimen should be taken into consideration [33].

The ideal insulin regimen for patients with T1D and DKA, as well as for the majority of patients with HHS, is a multi-dose regimen that includes basal insulin and prandial rapid-acting insulin analogs. In a randomized controlled trial, transition regimens of NPH and regular insulin twice daily were compared to glargine once daily and glulisine before meals. While the two groups had similar glycaemic control, the NPH/regular insulin group experienced more hypoglycemia (70 mg/dl) than the glargine/glulisine group [33].

## CONCLUSION

Much work must be done to reduce the prevalence of DKA and HHS and enhance the prognosis of patients with these illnesses. Despite

claims to the contrary, the rate of fatalities brought on by these problems is still far too high. The several causes of hyperglycemic decompensation in diabetic individuals should prompt an early diagnosis and fast treatment by the doctor.

Polydipsia, polyuria, weight loss, indications of intravascular volume loss, changes in mental status, abdominal pain, assessment of blood glucose, BUN, creatinine, anion gap, serum ketones, serum osmolality, urinalysis, and urine ketones are significant clinical signs to watch for in patients with the hyperglycemic crisis. ABG is crucial in the early evaluation of DKA, HHS, or a patient presenting with a mixed picture. The signs and symptoms of DKA and HHS must be understood, but it's crucial to understand when the two conditions coexist because doing so might increase two-fold mortality.

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## CONFLICTS OF INTEREST

The authors declare no conflict of interest about the publication of the manuscript file.

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