Avens Publishing Group
Adv Diabetes Endocrinol
February 2026 Volume:9, Issue:1

Open Access Case Report

Advances in Diabetes & Endocrinology

Management of Hyperglycemic Hyperosmolar State with Hypertension

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Submission: 27 January, 2026
Accepted: 24 February, 2026
Published: 26 February, 2026

Abstract

Management of Hyperosmolar Hyperglycemic State (HHS) with coexisting hypertension is a medical emergency requiring urgent, multidisciplinary care and immediate hospitalization. Treatment focuses on aggressive intravenous fluid resuscitation, careful electrolyte replacement—particularly potassium—and low-dose insulin therapy to gradually reduce hyperglycemia while minimizing the risk of cerebral edema. Concurrently, underlying precipitating factors, most commonly infection, must be identified and treated. Preventive measures against complications such as thromboembolism, including prophylactic heparin, are also essential in the absence of hemorrhagic stroke.

Introduction

Hyperosmolar Hyperglycemic State (HHS), previously known as Hyperosmolar Non-Ketotic Syndrome/Coma (HONK), is a severe, life-threatening complication of diabetes characterized by extreme hyperglycemia, profound dehydration, and marked hyperosmolarity. Blood glucose levels typically exceed 600 mg/dL, with effective serum osmolality greater than 320 mOsm/L. Severe hyperglycemia causes osmotic diuresis, leading to frequent urination, excessive fluid loss, intense thirst, and ultimately severe dehydration. This dehydration results in a high concentration of solutes in the blood, producing hyperosmolarity. Unlike Diabetic Ketoacidosis (DKA), HHS is associated with minimal or no ketone production, and the anion gap is usually normal due to the absence of significant ketoacidosis.

The key differential diagnosis between DKA and HHS are summarized in (Table 1).

Serum osmolality is calculated to estimate blood solute concentration using sodium, glucose, and blood urea nitrogen (BUN). The commonly used formula is Osmolality = $2[Na^+]$ + (Glucose ÷ 18) + (BUN ÷ 2.8), where sodium is measured in mEq/L and glucose and BUN are measured in mg/dL.

The anion gap (AG) is used to assess metabolic acidosis by calculating the difference between measured cations and measured anions in the blood, reflecting the presence of unmeasured anions such as albumin, phosphate, and organic acids. The standard formula is: $AG = (Na^+ + K^+) - (Cl^+ + HCO_3^-)$, with values expressed in mEq/L. A normal anion gap typically ranges from 8–16 mEq/L (or 6–12 mEq/L if potassium is excluded).

Table 1: Diagnostic Criteria used to differentiate between DKA and HHS

Parameter	Normal Range	DKA	HHS
Plasma Glucose (mg/dL)	70 to 110	≥ 250	≥ 600
Arterial pH	7.35 to 7.45	≤ 7.3	≥ 7.3
Serum HCO3- (mmol/L)	22 to 28	≤ 15	≥ 7.3
Osmolarity (mOsm/L)	275 to 295	≤ 320	≥ 320
Anion Gap (mEq/L)	<12	> 12	*Variable*
Serum Ketone	Negative	Moderate to high	None or trace
Urine Ketone	Negative	Moderate to high	None or trace

Case Presentation

A 73-year-old female patient was admitted to the emergency department of N.C. Medical College and Hospital, Israna, District Panipat, Haryana, with a reduced level of consciousness and focal neurological deficits. Prior to admission, she experienced shortness of breath, vomiting, and a seizure episode. She had a known history of diabetes mellitus and hypertension for the past 13 years.

Clinical Examination

On arrival, the patient's vital signs were notable for severe hypertension and signs of poor perfusion. Blood pressure was 220/120 mmHg, heart rate 95 beats/min, respiratory rate 24–28 breaths/min with SpO_2 93% on room air, and Glasgow Coma Scale was 8/15.

Physical examination findings:

Respiratory: Shortness of breath with oxygen saturation of 93% on room air.

Cardiovascular: Pulse 102 bpm, blood pressure 220/120 mmHg.

Neurological: Altered mental status with confusion; focal neurological deficits were present.

Abdomen: Generalized tenderness with a rigid abdomen on palpation.

Capillary blood glucose measurement read "too high" on the glucometer; therefore, a serum glucose test was ordered for accurate assessment.

Key Management Steps:

Initial management included intravenous hydration with normal saline at 60 mL/hour and administration of labetalol 20 mg IV to treat the hypertensive emergency. To control nausea and vomiting,

ISSN: 2475-5591

ondansetron (Emeset) 4 mg IV and pantoprazole (Pantop) 40 mg IV were given. Levetiracetam (Levera) IV was administered for seizure control and prophylaxis.

Laboratory investigations revealed that marked hyperglycemia with a blood glucose level of 658 mg/dL and serum osmolality of 330 mOsm/kg, consistent with hyperosmolarity. Total leukocyte count was significantly elevated at 24,100/mm^3. Serum troponin, renal function tests, and liver function tests were within normal limits, while CK-MB showed mild elevation (34.5 mmol/L; normal <25 mmol/L). Arterial blood pH level was 7.4. Urine analysis demonstrated trace ketones. Based on these findings, a diagnosis of hyperosmolar hyperglycemic state (HHS) was confirmed, and the patient was transferred to the Intensive Care Unit (ICU) for further management.

In the ICU, aggressive hydration with normal saline was continued. A low-dose insulin infusion was initiated using 50 units of rapid-acting insulin in 50 mL normal saline at a rate of 3 units/hour. Given the markedly elevated leukocyte count and the possibility of infection precipitating HHS, intravenous ceftriaxone and amikacin were started empirically.

The first glucose measurement in the ICU after insulin administration was 396 mg/dL, and levels gradually declined with ongoing insulin therapy and fluid replacement, falling to below 250 mg/dL on the second day. Initially, blood glucose was monitored hourly; once levels dropped below 200 mg/dL, monitoring was extended to every two hours. To prevent a rapid fall in blood glucose and reduce the risk of cerebral edema, 5% dextrose infusion was initiated once glucose approached target levels. As insulin therapy caused intracellular potassium shift, serum potassium dropped to 2.7 mmol/L and was corrected with 40 mmol potassium supplementation in IV fluids to prevent hypokalemia. Antihypertensive therapy with tablet telmisartan 40 mg and capsule nifedipine 10 mg was continued.

By the third day, the hyperosmolar hyperglycemic state had resolved; however, the patient remained confused. The primary therapeutic goals were to gradually reduce serum osmolality, control blood pressure, and lower blood glucose by approximately 50 mg/dL per hour. Multiple attempts to perform an MRI scan were unsuccessful due to the patient's non-compliance. Given the hypercoagulable state associated with hyperosmolarity, low-molecular-weight heparin was indicated for thromboprophylaxis; however, its administration was deferred as hemorrhagic stroke could not be excluded in the absence of MRI imaging.

Discussion

Hyperosmolar Hyperglycemic State (HHS) is a life-threatening metabolic complication of diabetes mellitus characterized by severe hyperglycemia, hyperosmolality, and dehydration in the absence of significant ketoacidosis. The underlying pathophysiology results from a relative insulin deficiency combined with increased counter-regulatory hormones such as glucagon, cortisol, and catecholamines. This hormonal imbalance promotes excessive hepatic glucose production and impaired peripheral glucose utilization, leading to extreme elevations in blood glucose levels, often exceeding 600 mg/dL.

The profound hyperglycemia induces osmotic diuresis, causing massive fluid and electrolyte losses. Progressive dehydration leads to reduced renal perfusion, further limiting glucose excretion and worsening hyperosmolality. Unlike diabetic ketoacidosis, some endogenous insulin activity is preserved in HHS, which prevents lipolysis and ketone formation; this explains the absence or minimal presence of ketones in this patient. Neurological manifestations such as confusion, focal deficits, and coma are primarily due to cellular dehydration within the central nervous system caused by elevated serum osmolality.

Management of HHS focuses on four key principles: aggressive fluid resuscitation, gradual correction of hyperglycemia, electrolyte replacement, and identification of precipitating factors. Intravenous isotonic saline is the cornerstone of therapy because restoration of intravascular volume improves renal perfusion and enhances glucose clearance. In this case, normal saline was appropriately initiated to correct dehydration and reduce osmolality.

Insulin therapy is introduced after initial fluid replacement to avoid rapid shifts in osmolality. A low-dose continuous insulin infusion was used to gradually reduce blood glucose by approximately 50 mg/dL per hour, which aligns with recommended guidelines and minimizes the risk of cerebral edema. Once glucose levels approached 250 mg/dL, dextrose infusion was added to prevent an abrupt decline and to allow continued insulin administration for resolution of hyperosmolarity.

Electrolyte monitoring is essential, particularly potassium. Insulin drives potassium into cells, predisposing patients to hypokalemia, as observed in this case. Timely potassium supplementation was necessary to prevent cardiac arrhythmias and neuromuscular complications. Additionally, prophylactic low-molecular-weight heparin was justified because hyperosmolarity and dehydration increase blood viscosity and thrombotic risk.

Infections are common precipitants of HHS due to stress-induced hormone release and insulin resistance. The markedly elevated leukocyte count raised suspicion of an underlying infection; therefore, empirical broad-spectrum antibiotics were initiated early, which is consistent with best practice.

Despite metabolic correction, the patient remained confused, reflecting delayed neurological recovery from prolonged hyperosmolality or a possible cerebrovascular event, warranting neuroimaging. Early recognition and adherence to evidence-based management were crucial in reversing the metabolic derangements and preventing further complications.

The cornerstone of Hyperosmolar Hyperglycemic State (HHS) management is the rapid correction of dehydration, gradual reduction of hyperglycemia, restoration of electrolyte balance—particularly potassium—and identification and treatment of precipitating factors such as infection. Effective management requires a structured and closely monitored approach:

Fluid Resuscitation: Immediate initiation of intravenous isotonic saline (0.9% NaCl) is essential to correct severe volume depletion and improve tissue perfusion. Depending on serum sodium levels and hydration status, 0.45% saline may later be considered.

ISSN: 2475-5591

Electrolyte Correction: Potassium levels must be monitored closely, as insulin therapy and osmotic diuresis can precipitate hypokalemia. Potassium should be added to intravenous fluids when serum levels fall below the normal range (3.5–5.5 mmol/L).

Low-Dose Insulin Therapy: After initial fluid replacement, a continuous intravenous insulin infusion (approximately 0.05 U/kg/hour) is recommended to gradually reduce blood glucose, targeting a decline of about 50 mg/dL per hour. Glucose levels are initially maintained around 300 mg/dL to avoid rapid osmotic shifts and cerebral edema; dextrose-containing fluids are introduced once glucose falls below approximately 250 mg/dL.

Close Monitoring: Hourly blood glucose measurements, frequent electrolyte assessment, and continuous monitoring of vital signs are required, ideally in an intensive care setting.

Identification and Treatment of Precipitating Factors: Infections are the most common triggers of HHS and should be actively investigated and treated with appropriate antibiotics when present.

Thromboprophylaxis: Due to increased blood viscosity and hypercoagulability associated with hyperosmolarity, low-molecular-weight heparin is recommended unless contraindicated.

Although hyperglycemic crises are relatively common in individuals with diabetes, the occurrence of acute strokes in the immediate context of HHS and severe hypertension is uncommon. This case illustrates the complex interplay between type 2 diabetes mellitus, uncontrolled hypertension, seizures, and subsequent cerebrovascular insults. Early recognition of HHS and prompt initiation of evidence-based therapy were critical in stabilizing the patient. Management with isotonic saline, insulin infusion, potassium supplementation, and empirical antibiotics led to gradual improvement in metabolic parameters and mental status within 48 hours.

The underlying pathophysiology in this patient was likely multifactorial. An acute infection probably precipitated severe hyperglycemia, leading to osmotic diuresis, dehydration, and marked hyperosmolarity. These changes, compounded by uncontrolled hypertension, may have contributed to cerebral hypoperfusion and increased risk of stroke.

Conclusion

HHS is a life-threatening complication of diabetes mellitus, and when accompanied by severe hypertension, it may significantly increase the risk of acute stroke. This case emphasizes the importance of proactive metabolic surveillance and strict glycemic and blood pressure control in patients with type 2 diabetes. Successful management of HHS with coexisting hypertension and neurological complications requires urgent, multidisciplinary intervention to restore metabolic stability and prevent long-term morbidity and mortality.

Ethics Approval

This case report is based on a retrospective analysis of anonymized patient data and does not constitute human subject's research. The report was prepared with the approval of the hospital ethics committee.

Conflicts of interest: The author declares no conflict of interest.

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