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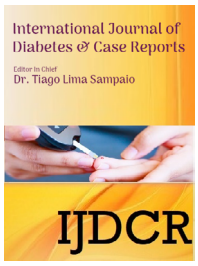


# Hyperosmolar Hyperglycemic Syndrome with Sepsis Secondary to Urinary Tract Infection – A Case Report

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ABSTRACT

**Background:** Hyperosmolar hyperglycemic syndrome (HHS) is a medical emergency and is a serious complication of type 2 Diabetes mellitus that needs immediate medical intervention. HHS is a less common complication of type 2 Diabetes mellitus. This report is of a woman who has attained menopause and has a history of type 2 Diabetes mellitus, hypertension and Urinary tract infection. The main aim of reporting the present case was to highlight the biochemical investigations involved in the diagnosis of HHS with sepsis secondary to UTI.

**Case presentation:** A 65-year-old female patient with a history of type 2 Diabetes mellitus, hypertension and urinary tract infection (UTI) was admitted to the emergency department. She had complaints insomnia for 15 days, epigastric discomfort and nausea for 5 days, vomiting and altered sensorium for one day. All the necessary investigations were done. Ketones were found to be present in the urine which is rare in case of HHS and more common in case of diabetic ketoacidosis (DKA).

**Conclusion:** This case highlights an atypical presentation of insomnia in HHS with sepsis and ketonuria This report also highlights the laboratory differentiators in the diagnosis of HHS from that of DKA.



## Introduction

Hyperosmolar hyperglycemia is characterized by a substantial increase in blood sugar levels, hyperosmolarity, and little or no ketosis and can be life threatening [1]. There are many underlying causes of HHS, but the vital causes include non-compliance to antidiabetic therapy, undiagnosed diabetes mellitus, certain medications, substance abuse and other comorbidities. HHS is characterized by extreme dehydration and absolute or relative insulin deficiency [2]. The fundamental underlying issue is an inadequate supply of insulin. Insulin insufficiency can cause hyperglycemia and impaired glucose absorption, which are both accompanied by elevated levelsof counter-regulatory hormones. It can also stimulate lipolysis and ketogenesis, which can lead to ketoacidosis. Osmotic diuresis, which results in dehydration, will be induced by hyperglycemia and hyperketonemia equally. Many cases of HHS are associated with poor medication adherence, cognitive impairment and poor quality of life. Clinical diagnosis is based on the presence of dehydration and high capillary glucose levels, as well as the presence or absence of ketones in the urine or plasma. The diagnosis is supported by the blood's pH, serum bicarbonate level and osmolality [3]. The effective correction of dehydration, hyperglycemia, ketoacidosis, and electrolyte disturbances is the mainstay of treatment [4].

## Case Report

A 65-year-old female presented to the department of general medicine with complaints of decreased sleep since 15 days, epigastric discomfort and nausea since 5 days, decreased responsiveness and altered sensorium since one day. The patient was apparently asymptomatic 15 days ago. The patient denied abdominal pain, fever, burning micturition, generalized weakness, loose stools, cough or cold. Patient is known case of diabetes mellitus, hypertension since 5 years and urinary tract infection 3 years ago. The patient was on Tab.Telmisartan and Inj. Insulin for hypertension and diabetes mellitus respectively. She is non-alcoholic and has no significant family history. Clinical examination revealed pallor. Vitals on admission were blood pressure of 70/50 mmHg, heart rate of 106 beats/min. On examination patient was pale, drowsy,irritable not obeying commands. Physical examination was positive for mild epigastric and left hypochondriac tenderness without guarding rigidity or distension no palpable organomegaly diffuse rhonchi positive in respiratory system. During the hospital stay patient was found to havesymptoms like Dark color urination, Discoloration of stool, drowsiness, wheezing, crepitus and myalgia.

Laboratory examination revealed Hemoglobin of 11.1 g/dl (reference range: 12-15g/dl), total leucocyte counts of 34,890 (reference range: 4000-11,000), hematocrit of 38.7% (reference range: 35-45%), platelet count of 3.94L (reference range: 1.5L-4.5L), random blood sugar >600mg/dl (reference range: <140mg/dl), Blood urea nitrogen 100mg/dl (reference range:10-45mg/dl), creatinine of 2.8mg/dl (reference range:0.8-1.2 mg/dl), SGOT level of 145 units/L of blood serum (reference range:

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8-45 units/l of blood serum), SGPT level of 30 (reference range: 7-56 units/l of blood serum), Lactate dehydrogenase level of 721 units/l (reference range:140- 280 units/l),D-dimer level of 8.50, Creatinine kinase level of 6775 units/l (reference range: 61- 81 units/l (for females 60-69 years). serum electrolyte levels were as follows- sodium:130 mEq/l, potassium: 7.6 mEq/l, chloride 82 mEq/l, Procalcitonin 5.934.ECG report showed right bundle branch block, USG abdomen report showed grade 1 fatty liver, HRCT chest report showed patchy area of consolidation in dependent segments of left lower lobe and right mild and left minimal plural effusion with sub-segmental atelectasis of the underlying lung, 2D ECHO report showed grade 1 diastolic dysfunction, PML calcified, mild MR no MS, Trileaflet with calcific specks over LCC aortic valve, trivial AR, mild TR, good RV function basal >50% respiratory variation.On the basis of laboratory reports, intensive intravenous fluids and regular insulin were administered, In view of hypotension, inotropes were added. Serum electrolytes and blood pressure were monitored every 6 hours and 2 hours respectively. Serum glucose levels decreased approximately 40mg/dL/hr and was brought to 147 mg/dL 21 hrs after the treatment started however fluctuations were still evident. The laboratory results that confirmed the diagnosis of HHS with sepsis were as follows: Glycated haemoglobin (HbA1C):12.8% (reference range:<5.6%), Ketone bodies and glucose were present in urine, Blood urea nitrogen was found to be 46.72 mg/dL, Osmolality was 344.0 mOsm/kg (reference range:275-295 mOsm/kg), Lactate dehydrogenase level of 721 units/l (reference range:140-280 units/l), D- dimer level of 8.50, total leucocyte count of 34,890 (reference range: 4000-11,000) and hypotension.

After 12 days of hospital stay that included blood glucose control and HHS education, she was discharged without any symptoms. She was advised for insulin therapy (ultra-long-acting insulinand rapid acting insulin) and self-glucose monitoring.

| Lab Parameters | Values |
|---|---|
| Hemoglobin | **9.6 %** |
| Packed cell volume | **38.7** |
| Platelets | **3,94,000** |
| Procalcitonin | **5.394** |
| D-Dimer | **8.50** |
| HbA1C | **12.8%** |
| Osmolality | **344** |
| Random plasma glucose | **457 mg/dL** |
| Mean corpuscular volume | **79.4** |
| Lactate dehydrogenase | **721 U/L** |
| SGOT | **145 U/L** |

**Table 1:** Lab parameters showcasing evidence of HHS with sepsis.

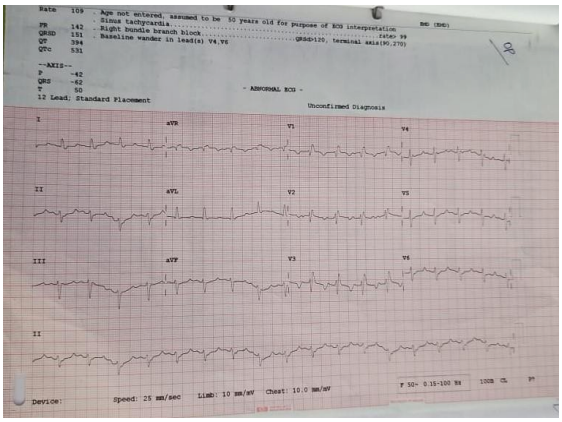


**Figure 1:** ECG abnormalities.

## Discussion

HHS is a significant and perhaps fatal consequence of type 2 diabetes. The death rate in HHS canreach 20%, which is nearly ten times higher than the mortality rate in diabetic ketoacidosis. Age, the level of dehydration, and the existence or absence of additional comorbidities all affect the clinical outcome and prognosis in HHS [5] Extreme hyperglycemia, typically > 600 mg/dL, hyperosmolality, and dehydration are common in HHS patients. Although the true prevalence of HHS is unknown, it is most likely to account for less than 1% of diabetic patients' hospitalizations. Most instances are diagnosed in elderly patients with type 2 diabetes mellitus [6]. As a result of glycosuria and electrolyte loss through urination in a hyperglycemic state, the body becomes hyperosmolar. Osmolality can thus be used to gauge theseverity of HHSand to track how quickly the condition changes in response to treatment while itis in a hyperosmolar state [7]. At a rate of 10 to 20 mL/kg/h during the first hour, isotonic solution hydration is advised. Through a reduction in counter-regulatory hormones and an improvement in renal perfusion, initial hydration typically lowers the glucose levels. As a result of the quick correction in blood osmolarity, it is important to keep an eye on the plasma sodium levels throughout this procedure to avoid complications such cerebral oedema or central pontine myelinolysis. It is necessary to adjust the serum sodium level to represent the actual state of the body since it can vary based on an already high blood glucose level. Water is forced to flow to the extracellular fluid (ECF) by the increased osmolality of the ECF in hyperglycemic conditions, which lowers blood sodium proportionally to the ECF's dilution. When glucose levels fall, this mechanism is called transcriptional hyponatremia and returns to normal. In this case report the patient had a history of insomnia for which the patient was taking Tab. zolpidem5 mg but it was found to be ineffective. Lab parameters like alarming SGOT levels, procalcitonin levels, creatinine kinase levels, lactate dehydrogenase levels and D-dimer indicated septic shock,this might have been caused due to progression of pre-existing urinary tract infection into sepsis which later resulted in septic shock in this patient. ECG report indicated right bundle branch block and HRCT report indicated right mild and left minimal pleural effusion with Sub segmental atelectasis of the underlying lung. These can further lead to increased workload on theheart.

## Conclusion

HHS is a serious complication of type 2 diabetes mellitus. As HHS is a long term complication of diabetes mellitus, in such cases when there is bacterial invasion peripheral nerve damage and reduced blood flow is seen which increases the progression of infection causing complications such as sepsis. The alarming levels of blood glucose paves the way for bacterial growth leading to life threatening consequences as seen in this case which was evident by increased lactate dehydrogenase and D dimer levels. Diabetes mellitus is a chronic condition hence, for better therapeutic outcome it is imperative for the patient to adhere to the prescribed medications. In this case, the patient was poorly adherent to the medications prescribed which might be one of the reasons for progression of HHS. HHS caused metabolic derangements such as hyperkalaemia and hyponatremia which was evident on ECG. Treatment focuses on rehydration with intravenous fluids, intensive insulin therapy and antibiotics to treat the infection causing sepsis. Intravenous fluids are given to replace fluid loses from osmotic diuresis and restore hydration and blood pressure. Insulin is rapidly administered in high doses to rapidly lower glucose levels and prevent further complications. Broad spectrum antibiotics are started immediately based on the suspected infection site and adjusted based on culture results. Medication adherence is imperative for long- term management and prevention of recurrent HHS episodes in such high-risk patients. Once the acute episode is stabilized, patients should be counselled on strategies to improve diabetes control and reduce the likelihood of future episodes. This includes optimizing insulin regimens, adhering toa diabetic diet, exercising regularly, monitoring blood glucose levels frequently and taking all medications as prescribed. Patients who have experienced HHS once are at higher risk for recurrence, so close follow up is essential.

## List of Abbreviation

| | |
|---|---|
| HHS | Hyperosmolar hyperglycemic syndrome |
| DKA | Diabetic ketoacidosis |
| UTI | Urinary tract infection |
| SGOT | Serum Glutamic Oxaloacetic Transaminase |
| SGPT | Serum Glutamic Pyruvic Transaminase |
| ECG | Electrocardiogram |
| HRCT | High-resolution computed tomography |
| USG | Ultrasonography |
| PML | Posterior mitral leaflet |
| MR | Mitral regurgitation |
| MS | Mitral stenosis |
| LCC | left coronary cusp |
| AR | Atrial regurgitation |
| HbA1C | Hemoglobin A1c |
| ECF | extracellular fluid |

**Conflict of interest:** None

**Acknowledgements:** None

**Ethical Consideration:** Not Required

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