

When Blood Turns to Syrup - A Case report

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Abstract: This case presents a 57-year-old female patient with a severe hyperosmolar hyperglycemic state. The initiated rehydration and treatment with an insulin infusion pump led to an extremely rapid decrease in hyperglycemia. Despite the rapid correction of the hyperglycemic state, the patient remained neurologically stable throughout. The condition was successfully managed in the intensive care unit. This was her initial diagnosis of diabetes mellitus.

Keywords: Hyperglycemic hyperosmolar state, Diabetic ketoacidosis, Cerebral edema.

CLINICAL INTRODUCTION AND FINDINGS

We report the case of a 57-year-old female patient who presented to the emergency department via ambulance due to vertigo, dyspnoea, polyuria, and a general decline in condition. According to the patient, the symptoms had only been present for a few hours. The personal medical history revealed a moderate intellectual disability with occasional aggressive outbursts, for which the patient was being treated with Tegretol (carbamazepine) and Truxal (chlorprothixene hydrochloride). Due to her cognitive impairment, the patient resided in an assisted living facility. According to the caregivers, the patient had experienced pronounced polydipsia and polyuria for several days. Owing to her preference for syrup, she reportedly consumed several litres of it over the weekend in response to increased thirst.

Upon admission to the emergency department, the clinical examination revealed a hemodynamically stable, tachycardic (106 bpm), afebrile patient with an oxygen saturation of 92% on 3 L/min oxygen supplementation. The patient was awake but disoriented, with a Glasgow Coma Scale (GCS) score of 14–15.

Initial laboratory findings indicated a severe hyperosmolar hyperglycemic state (HHS) with a blood glucose level of 101 mmol/L (reference range 3.9–5.6 mmol/L) and an serum osmolality of 407 mOsmol/kg (reference range 270–295 mOsmol/kg). Additional findings included dyselectrolytemia with hyponatremia and hyperkalemia, moderately impaired renal function, and mild ketonuria

(Table 1). Blood gas analysis showed a normal pH and partial respiratory insufficiency (pH 7.360, PaO₂ 9.76 kPa [reference 10.70–13.30], PaCO₂ 5.41 kPa [reference 4.67–6.00], base excess -3.1 [reference -3.0–2.0], bicarbonate 22.4 mmol/L [reference 21–26 mmol/L]).

DIFFERENTIAL AND FINAL DIAGNOSIS

The combination of symptoms, including general deterioration, polyuria, and polydipsia, raised suspicion of hyperosmolar hyperglycemic syndrome (HHS). This was later confirmed through laboratory results. HHS must be differentiated from diabetic ketoacidosis (DKA), as both represent serious complications of diabetes mellitus.

HHS typically occurs in older patients with type 2 diabetes and develops over days to weeks. It is characterized by extremely high blood glucose levels (≥ 33 mmol/L), marked hyperosmolality (>320 mOsmol/kg) and severe dehydration. A hallmark of HHS is the absence of substantial ketone body production. Due to the presence of residual insulin, lipolysis and ketogenesis are inhibited. The mortality rate of HHS is 5–20%, which is ten times higher than that of DKA [1].

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Table 1. The progression of blood gas analyses in the first 4 hours after presentation

	Reference	15:46	16:50	20:07
Hemoglobin	120–147 g/l	149		
Leukocytes	3.5–10 10^9 /l	9.0		
Sodium	136–145 mmol/l	125		150
Potassium	3,6–5,1 mmol/l	6.0		4.1
Osmolality	270–295mmol/kg	407		
Blood glucose	3.9 – 5.6 mmol/l	101	82.8	43.9
Creatinine	μ mol/l	108		89
eGFR (CKD-EPI)	ml/min	49		62
CRP	<5,1 mg/l	8.0		8.9
HbA1c (NGSP)	<5.7 (SGED)			13.4%

In contrast, diabetic ketoacidosis (DKA) primarily occurs in children and younger patients with type 1 diabetes. The diagnosis requires the presence of metabolic acidosis, ketonemia, and hyperglycemia. DKA results from an absolute insulin deficiency, leading to uncontrolled lipolysis and ketogenesis, and subsequently to metabolic acidosis. It is noteworthy that the metabolic acidosis is not caused by the ketone bodies produced in the liver but by the free fatty acids in the blood derived from lipolysis. Ketone bodies serve as an alternative energy substrate for cells unable to utilize fatty acids as an energy source (e.g. neurons, erythrocytes).

In approximately 30% of cases, an overlap of the conditions occurs in both type 1 and type 2 diabetes. Typical symptoms include polydipsia, polyuria, weight loss, and fatigue. Specific symptoms of DKA include abdominal pain, nausea, vomiting, and Kussmaul breathing with a fruity odor on the breath. A hallmark symptom of hyperosmolar hyperglycemic syndrome (HHS) is altered consciousness, which may progress to coma [1].

The identification of a trigger is of utmost importance, as infections often act as triggers and must be actively sought. In our patient, nasopharyngeal swabs for SARS-CoV-2, influenza, and RSV were negative. A chest X-ray showed no infiltrates, and a urine analysis provided no evidence of a urinary tract infection. A sonographic examination ruled out postrenal causes of renal insufficiency.

Other common triggers of HHS include acute cardiovascular events, pancreatitis, or medications (e.g. corticosteroids, thiazide diuretics, sympathomimetics, antipsychotics). Poor therapeutic adherence or the initial diagnosis of diabetes are also frequent causes [1], [2].

COURSE AND TREATMENT

The treatment of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic syndrome (HHS) is largely similar. For both conditions, the goals are to restore fluid

and electrolyte balance, normalize serum glucose levels, and address the underlying triggers. Management in both conditions begins with volume correction. After initiating rehydration, potassium levels should be assessed and supplemented if necessary. Only then should insulin therapy for blood glucose control be started. In DKA, due to absolute insulin deficiency, insulin administration should begin promptly. According to the *Guidelines of the Joint British Diabetes Societies for Inpatient Care* (JBDS-IP), insulin in DKA should always be administered via an intravenous infusion pump. A primary intramuscular bolus of insulin is no longer recommended, except when a delay in initiating an insulin infusion is anticipated [3].

In HHS, initial insulin treatment can be deferred. Insulin therapy via an infusion pump should also be introduced during treatment. Rapid correction of hyperglycemic crises should be avoided to minimize the risk of cerebral edema. The reduction in blood glucose levels should not exceed 3–5 mmol/L per hour [3], [4].

It is of paramount importance to maintain continuous insulin administration during the phase of metabolic recompensation. Interrupting insulin delivery can lead to renewed lipolysis and subsequent release of free fatty acids, causing a recurrence or worsening of metabolic acidosis. To achieve this, high-concentration glucose infusions are often necessary to sustain a minimal rate of insulin infusion without inducing hypoglycemia.

The development of cerebral edema is a feared complication of both DKA and HHS, associated with a mortality rate of 20–40% [2]. This complication predominantly occurs in children and adolescents with DKA, affecting 0.3–1% of cases [1].

It is hypothesized that overly rapid correction of the metabolic derangement may contribute to the development of cerebral edema. In adult patients, cerebral edema is extremely rare, but it has been reported in young adults

and individuals with very low body weight [3]. Precise data on its incidence are scarce.

In 2018 Kuppermann et al. published a randomized controlled trial investigating the impact of infusion rate and sodium chloride concentration in intravenous fluids on neurological outcomes in children with diabetic ketoacidosis. Neither the infusion rate nor the sodium chloride content of the intravenous fluids showed a significant effect on neurological outcomes [5].

In general, it is recommended to maintain serum glucose levels at a high-normal range during the first 24 hours of treatment. When glucose levels fall below 14 mmol/L, high-concentration glucose infusions should be initiated in addition to insulin therapy [3], [4].

In our case, intensive rehydration (1 L in the first hour), potassium supplementation (40 mmol/L ad infusion/24 h), and insulin therapy via an infusion pump (Novorapid at 4 units/hour) were initiated in the emergency department. The patient was subsequently transferred to the intensive care unit (ICU). Upon ICU admission (4 hours after initial presentation), the glucose level had already decreased to 43.9 mmol/L (Table 1). Despite the rapid decline, the patient exhibited no neurological deficits.

Under continuous monitoring, we continued the established therapy with insulin and potassium via infusion pump, as well as adequate hydration. Additionally, to prevent an overly rapid reduction in blood glucose levels and thus minimize the risk of cerebral edema, intravenous glucose therapy was initiated. Under this treatment, all parameters improved, and the patient showed no neurological deterioration during therapy.

Given the initially high glucose levels, mild ketonuria, and well-compensated blood gas analysis, we presumed sufficient endogenous insulin production and residual beta-cell function, leading to a diagnosis of type 2 diabetes mellitus. During the course of hospitalization, the HbA1c level was determined to be 13.4%. Since the patient could transition to normal nutrition within one day, insulin therapy was switched to subcutaneous administration, and the patient was transferred to the general medical ward.

On the general medical ward, the insulin therapy was supplemented with Metformin and Sitagliptin based on the suspected diagnosis of type 2 diabetes mellitus. As the patient was of normal weight, further diagnostic workup included the assessment of endocrine autoantibodies (islet-cells antibodies (ICA) and anti-GAD). During the hospital stay, the patient complained of visual disturbances. The consulting ophthalmologists attributed this to the hyperglycemic crisis, with no evidence of diabetic retinopathy.

After 10 days with a positive and stable development of the blood glucose levels the patient was discharged back to her assisted living facility. At discharge, her therapy was adjusted to include Lantus, Novorapid, and Janumet (Metformin-Sitagliptin). Post-discharge endocrine autoantibodies were found to be positive. Consequently, the diagnosis was revised to late-onset autoimmune diabetes.

Abbreviations used in the Article

HHS = Hyperglycemic hyperosmolar state / syndrome

DKA = Diabetic ketoacidosis

GCS = Glasgow coma scale

ICA = Islet-cells antibodies

GAD = Glutamic acid decarboxylase

KEY MESSAGES

- Hyperosmolar hyperglycaemic syndrome (HHS) and diabetic ketoacidosis (DKA) are life-threatening metabolic derangements in diabetes mellitus.
- The first step in treating HHS and DKA is the correction of volume depletion. Initially, 1 litre of crystalloid solution (0.9% NaCl or Ringer's acetate) can be safely administered over one hour.
- In DKA, due to absolute insulin deficiency, insulin therapy should be initiated promptly after potassium levels are checked. In HHS insulin administration can be delayed.
- An initial insulin bolus is no longer recommended unless a delay in starting insulin infusion is anticipated.
- The development of cerebral edema in adults is extremely rare. The pathophysiology remains poorly understood. It is likely that serum glucose can be lowered more rapidly than previously assumed without significantly increasing the risk.

Contributorship

JM wrote the first draft of the manuscript, prepared the tables and is the guarantor. MS revised and helped finalise the manuscript and helped with the paper submission. All two authors were involved in the care of the patient and gave final approval of the manuscript.

Conflicts of Interests

The authors have no conflicts of interests to declare.

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Patient consent for publication

Patient consent was obtained.

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