

Nutritional Management of Severe Diabetic Ketoacidosis in a Newly Diagnosed Type 2 Diabetes: A Case Report

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Abstract: Diabetic ketoacidosis (DKA) is a life-threatening metabolic emergency characterised by hyperglycaemia, ketosis, and metabolic acidosis. While acute management primarily focuses on fluid replacement, insulin therapy, and electrolyte correction, the nutritional consequences of DKA are often underrecognized. Prolonged inadequate intake, infection-related catabolism, and severe hyperglycaemia place patients at considerable risk of malnutrition and muscle loss. This case report describes the nutritional management of a 53-year-old female admitted with seizure-like activity and severe hyperglycemia (>800 mg/dL) who was subsequently diagnosed with diabetic ketoacidosis and de novo diabetes mellitus. A structured nutrition care process was implemented throughout her hospital stay, involving nutrition assessment, enteral nutrition support, followed by transition to oral diabetic diets with oral nutritional supplementation (ONS), through glycaemic monitoring, and intensive nutrition education, because of which recovery was achievable. Following multidisciplinary management, glycaemic control improved significantly with HbA1c reducing from 17.6% at diagnosis to 10.4% at one month and 5.9% at two months. Nutritional intervention facilitated recovery, improved dietary intake, supported weight maintenance, and contributed to successful transition from insulin therapy to oral hypoglycaemic agents. This case highlights the critical role of clinical dietitians in DKA management and demonstrates how early, individualised nutrition intervention can improve both short-term recovery and long-term metabolic outcomes.

Keywords: Diabetic ketoacidosis, Medical nutrition therapy, Clinical dietitian, Enteral nutrition, Hyperglycaemia, Diabetes mellitus, Case report

1. Introduction

Diabetic ketoacidosis (DKA) and Hyperosmolar state (HHS) appear as 2 extremes in the spectrum of diabetic decompensation, which are serious acute complications of diabetes mellitus resulting from absolute or relative insulin deficiency¹.

This condition is characterised by severe hyperglycaemia, ketosis, metabolic acidosis, dehydration, electrolyte imbalance, and increased protein catabolism. Infection remains one of the most common precipitating factors, particularly in individuals with undiagnosed diabetes².

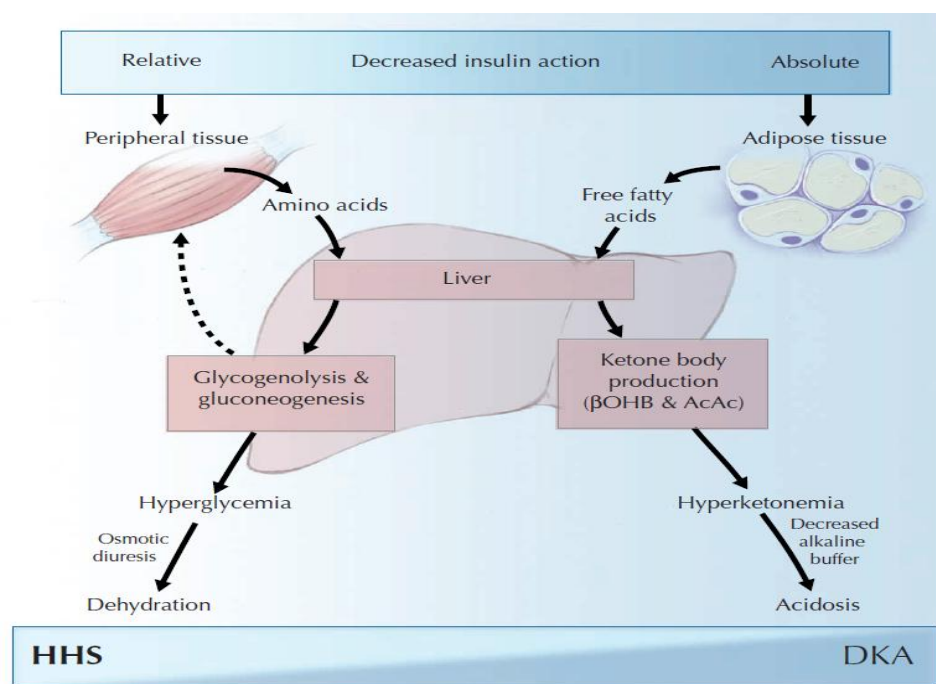


Figure 1: Schematic of the pathogenesis of diabetic ketoacidosis (DKA) and the hyperglycemic hyperosmolar state (HHS). Relative or absolute insulin deficiency stimulates hepatic glucose production, which results in hyperglycemia, osmotic

diuresis and dehydration. In severe insulin deficiency, the liver will augment ketone body production, culminating in hyperketonemia and, eventually, acidosis. $\beta\text{OHB} = \beta\text{-hydroxybutyric acid}$; $\text{AcAc} = \text{acetoacetic acid}$ ².

Beyond its metabolic consequences, DKA has significant nutritional implications. Reduced oral intake, inflammatory stress, accelerated proteolysis, dehydration, and prolonged hospitalization can contribute substantially to nutritional deterioration. Current nutrition guidelines emphasize the importance of early nutrition intervention following hemodynamic stabilization to preserve lean body mass, improve metabolic recovery, and support overall clinical outcomes³.

2. Case Presentation

A 53-year-old female presented to the emergency department (ER) with seizure-like activity involving bilateral upper limbs. No tongue bite, frothing, or upward rolling of eyes was reported. The patient had a known history of hypertension managed with medication.

Anthropometric Assessment

Height: 155 cm, Weight: 62 Kg, Body Mass Index (BMI): 25.8 kg/m², Ideal Body Weight: 53 kgs.

Clinical Findings and Diagnosis

Comprehensive evaluation revealed:

- Diabetic ketoacidosis
- Newly diagnosed Type 2 Diabetes Mellitus
- Bilateral pyelonephritis
- Lower respiratory tract infection
- Anemia
- Acute kidney injury (Stage 1)
- Uterine prolapse with cystocele

Nutritional Assessment

A detailed dietary history revealed significant nutritional compromise before admission. For nearly two weeks, the patient consumed predominantly curd rice and buttermilk due to poor appetite and illness. Estimated intake: Energy: approximately 500 kcal/day, Protein: approximately 7 g/day. This represented substantial inadequacy relative to estimated requirements and increased the risk of muscle wasting during acute illness.

Subjective Global Assessment (SGA) classified the patient as nutritionally stable; however, recent dietary history suggested emerging nutritional risk secondary to acute disease burden and prolonged inadequate intake.

Nutrition Diagnosis

PESS: Inadequate oral intake and altered nutrition-related laboratory values related to acute metabolic stress and lack of diabetes awareness as evidenced by poor intake and hyperglycemia

Nutrition Intervention

The primary goals of MNT were:

- To initiate oral diet at the earliest.
- To provide a low-carbohydrate, high-fibre diet.
- To monitor GRBS.

The long-term goals of MNT were:

- To prevent DKA.
- To prevent muscle loss.
- To provide adequate micro and macronutrients.

Nutrition Requirement

Energy: 1590 kcal/day (25 kcal/kg IBW)

Protein: 80 g/day (1.5 g/kg IBW)

Carbohydrates: 200 g/day

3. Review of Literature

DKA requires urgent medical management to improve the clinical outcome of the patient. As per review of literature approx. 15% of children and 27% of adults hospitalized with acute hyperglycaemic crisis presented with DKA complicated by severe hyperglycaemia and hyposmolality⁴.

Common precipitating causes of hyperglycaemia include skipping insulin or antidiabetic medications, psychiatric illnesses, substance abuse, and infection⁵.

The treatment goals for DKA and HHS are similar and include replenishing intravascular fluid levels, bringing hyperglycemia and hyposmolality to normal levels, correcting ketonemia and electrolyte imbalance, and treatment of precipitating causes⁶.

In addition, continuous monitoring of IV fluid administration rate, urine output, and insulin dosage is necessary to assess response to medical therapy. Hourly monitoring of vitals, as well as mental and hydration status, is also recommended. Measurement of serum glucose, electrolytes, ketones, venous pH, bicarbonate, and anion gap should be done every 2-4 h. As part of the initial evaluation, hemoglobin A1c (HbA1C), complete blood count (CBC) with differential, basic metabolic panel, urinalysis, coagulation profile, cardiac enzymes, and hepatic enzymes are also needed. Chest X-ray, urine and blood cultures, lipase, and ECG can also be performed to identify the precipitating cause. Further testing can be done based on specific cases^{6,7}.

The initial therapy for both DKA and HHS is IV fluids. Patient vital signs, electrolyte levels, and urinary output may dictate fluid therapy⁷.

4. Case Discussion- Medical & Nutritional Management

The patient's attendant mentioned the current complaints with which the patient was brought to the ER of the hospital; they gave a brief history of fever 2 weeks ago, along with intermittent cough with sputum for the past 2 weeks, along with decreased oral intake and generalised weakness.

With the above complaints, the patient was taken to an outside hospital where she was evaluated for hyponatremia with seizures and primary treatment was given, and she was referred to a tertiary hospital for further evaluation.

Radiological investigation: Computed tomography (CT) of the abdomen and pelvis confirmed bilateral pyelonephritis, complete uterine prolapse, and large cystocele.

Urine culture grew *Klebsiella pneumoniae*. An anaemia panel was obtained in view of low haemoglobin, which revealed elevated LDH of 246 U/L and low transferrin saturation of 9.56%. A urine electrolyte assessment was performed to evaluate electrolyte disturbances.

Sputum culture also grew *Klebsiella pneumoniae*.

GRBS was >800mg/dl at the time of arrival to the ER; hence, HAI (Human Insulin) infusion was started at 8ml/hr. Since the patient was in an altered metabolic and sensorium state, a Ryle's tube was placed, and she was shifted to the ICU (Intensive Care Unit) for further medical management.

Primary diagnosis was DKA – de novo DM, AKI with urosepsis and urethral prolapse.

Her initial laboratory studies revealed markedly elevated HbA1C of 17.6%, hypoalbuminemia of 2.3gm/dl, elevated uric acid of 10.6mg/dl, elevated phosphate of 4.8 mg/dl and elevated creatinine of 1.74 mg/dl.

Urine analysis demonstrated 2+ ketones, 2+ blood, plenty of pus cells and granular casts. C- reactive protein was significantly elevated at 233mg/dl. CBC showed low haemoglobin of 8.3 gm/dl and leucocytosis with a white blood cell count of 23K, while other parameters were within normal ranges.

The patient was initiated on IV fluids, pantoprazole injection, intravenous antibiotics and other supportive medications.

Her 1st recordable sugars were after 6 hours of hospitalisation, i.e. 585 mg/dl with ongoing HAI infusion. Upon stabilisation, RT feeds were initiated at 50ml/hr with isocaloric supplement which has components of low carbohydrates, myoinositol and GLP-1 which would help to maintain the blood sugar spike.

On day 2 of ICU stay, the shock was resolving, and HAI – 6ml/hr infusion was ongoing along with IV fluids, and RT feeds at 50ml/hr. The patient's clinical stabilisation was of

utmost priority at this point over nutritional management. 3 litres of fluid per day was recommended (IV + RT feeds) in view of anion gap – HAGMA on ABG (arterial blood gases).

From day 3 onwards, her sugars were managed using a combination of insulins, i.e. HAI and Tresiba. GRBS was monitored 5 times a day, and insulin dosage was adjusted according to the sugar scale; slowly, we were now able to focus towards her nutritional stability.

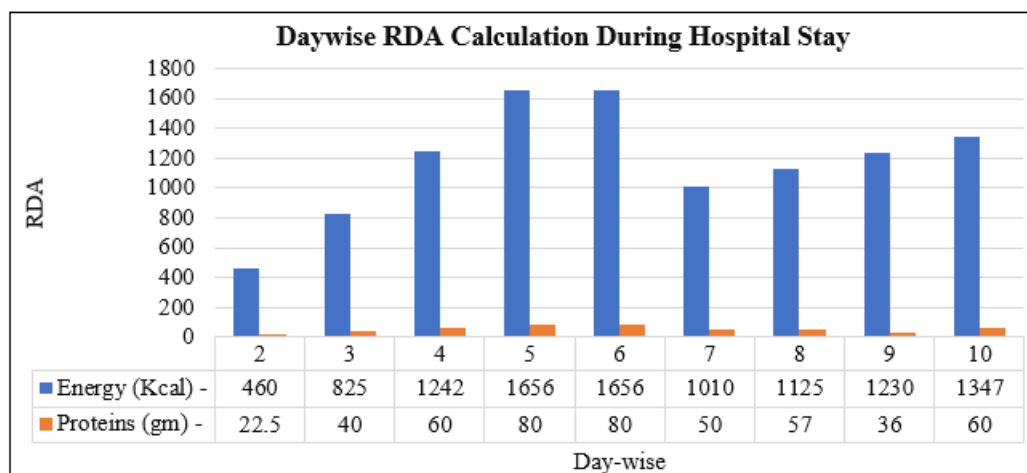
By day 7, the patient was allowed oral sips of diabetic liquids along with 50ml/hr RT feeds; by then, sugars were stabilised and were managed by only a few units of HAI and GRBS was checked thrice a day.

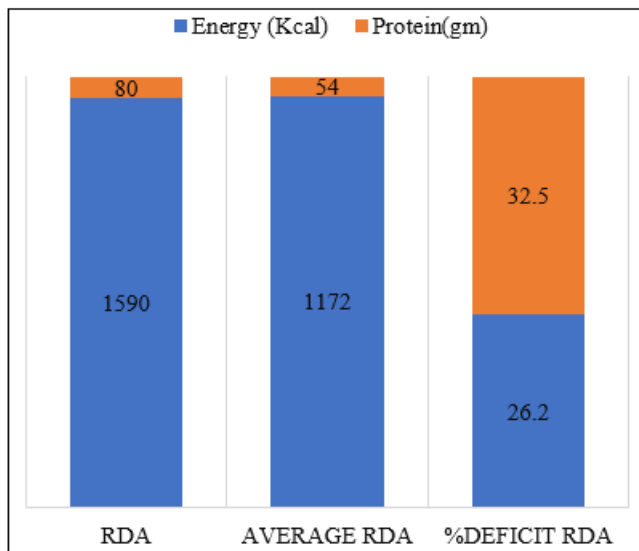
On day 8, she was allowed to have an oral diabetic soft diet along with 50ml/hr RT feeds. Since she was clinically stable hence shifted to the ward.

On day 9, her morning sugars were 106 mg/dl which was managed with OHA (oral hyperglycaemic agents) and insulin. She was able to take 65% of the prescribed oral diet, and the rest was compensated with oral nutritional supplement (ONS) and liquids. Along with her lunch meal, she was also advised to use 4 gm of mulberry leaf extract supplement, which would help to keep her sugars from instant spikes and crashes.

At this point in her medical treatment, she was advised to take OHA – 500mg BD after food along with adequate fluid intake.

Day-wise Nutritive Calculation During Hospital Stay			
Day	Diet	Energy (Kcal)	Proteins (gm)
1	NBM	-	-
2	RT feeds at 50ml/hr	460	22.5
3	RT feeds at 50ml/hr	825	40
4	RT feeds at 75ml/hr	1242	60
5	RT feeds at 100 ml/hr	1656	80
6	RT feeds at 100 ml/hr	1656	80
7	RT feeds 50ml/hr + Oral liquids	1010	50
8	RT feeds at 50ml/hr + Diabetic Soft diet	1125	57
9	Diabetic Soft diet + ONS	1230	36
10	Diabetic normal diet + ONS	1347	60





We tried reassessing her weight, which was difficult as she was unable to stand. Her functional capacity was assessed using handgrip strength, which showed a poor functional state.

Her intake and recovery were closely monitored and cared for, and she was given a strict discharge diet plan along with a food and fluid chart at the point of discharge, which would help a dietitian assess the nutritional adequacy even after discharge, especially in such a vulnerable case so as to know the nutrition delivered daily at least until her 1st follow-up.

This case study report was a huge success only because of the multidisciplinary approach, and moreover, the patient and attendant were active and cooperative enough to adhere to the diet counselling. Post-discharge, the patient's fasting sugars were 170 mg/dl, 170 mg/dl and 224 mg/dl for the first 3 consecutive days and were managed with OHA BD.

After 1 week post-discharge, the patient visited the dietitian's OPD; she was able to walk independently without support, and her functional capacity was assessed and which was noted as average. She lost 2 kgs of weight from the time of admission. Her body composition analysis (BCA) demonstrated: Protein (7.1Kg); Fat-free mass (37.8 Kg), Body fat mass (18.2 Kgs) and total body water (27.9 L), suggesting recovery without significant long-term sarcopenia.

A month later, her HbA1C came to 10.4%, and she was then admitted for her hysterectomy surgery in view of uterine prolapse. Regular follow-up was done telephonically and on an OPD basis very religiously.

Exactly 2 months from her 1st time admission, her HbA1C was marked as 5.9%, which proved that strict adherence and dedication towards diet and consistent follow-up with a dietician made a remarkable difference in the sugar control. A high-protein, low carbohydrate diet with ONS and mulberry leaf extract supplement – a stick per day made all things achievable. A consistent follow-up is essential to attain the impossible goal of glucose control possible.

5. Conclusion

Diabetic ketoacidosis is not only an acute metabolic emergency but also a profound nutritional crisis characterised by severe catabolism, reduced intake, and increased metabolic demand. This case demonstrates that early, structured, and continuous MNT beginning with enteral nutrition during stabilisation and transitioning to individualised oral diabetic diet planning played a pivotal role in recovery.

The integration of nutrition support with medical management contributed to improved glycaemic control, preservation of lean body mass, successful transition to oral therapy, and prevention of recurrent metabolic decompensation. Long-term follow-up further confirmed sustained metabolic improvement and strong dietary adherence.

This case strongly supports the inclusion of clinical dietitians as essential members of the multidisciplinary DKA management team and emphasises that nutrition therapy should be considered a core therapeutic intervention rather than an adjunct in diabetic ketoacidosis care.

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