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Nutrition care in pediatric patients with severe diabetic ketoacidosis, hyperglycemia and suspected type 1 diabetes

Wahyu Dwi Suzanti[1], Sukmawati[2], Suriani Rauf[3], Musyawarah Thahir[4]

[1,2,3]Professional Education of Dietitian, Poltekkes Kemenenkes Makassar, Sulawesi Selatan, Indonesia

[4]RSUD Wahidin Sudirohusodo Makassar, Sulawesi Selatan, Indonesia

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ABSTRACT

Diabetes mellitus (DM) is a chronic disease with an increasing prevalence among children. Type 1 DM, the most common form in pediatrics, results from autoimmune destruction of pancreatic β-cells, leading to absolute insulin deficiency. Diabetic ketoacidosis (DKA) is a life-threatening acute complication that may cause acute kidney injury (AKI), electrolyte imbalance, and respiratory distress. This case study applied the Standardized Nutrition Care Process (SNCP), including nutritional assessment, diagnosis, intervention, monitoring, and evaluation, in a 1-year-3-month-old girl diagnosed with severe DKA, suspected type 1 DM, severe dehydration, community-acquired pneumonia, anemia, hypokalemia, azotemia, KDIGO stage II AKI, and marasmus-type malnutrition. Nutritional assessment showed severe malnutrition (weight-for-length Z-score < −3 SD) with clinical signs of *iga gambang*. Nutrition diagnoses included altered nutrition-related laboratory values associated with endocrine dysfunction, evidenced by HbA1c 11.1%, random blood glucose >200 mg/dL, urea 123 mg/dL, creatinine 0.9 mg/dL, hemoglobin 10.1 g/dL, and estimated glomerular filtration rate (eGFR) 48%, as well as disease-related pediatric malnutrition characterized by low weight-for-length, mid-upper arm circumference (MUAC) 83.3%, inadequate intake (<50% of requirements), and clinical malnutrition signs. Nutritional intervention consisted of a 1,100-kcal diabetic diet with enteral formula via nasogastric tube and chopped porridge orally. The patient's nutritional intake gradually improved to an adequate level during hospitalization, although significant changes in nutritional status were not observed because of the short intervention period. Nutrition education was provided to the family to support long-term management. This case highlights the importance of individualized, gradual, and closely monitored nutritional therapy as part of multidisciplinary care to optimize recovery and prevent further complications in pediatric patients with type 1 DM and severe DKA.

Corresponding Author:

Wahyu Dwi Suzanti,
Professional Education for Dietitians,
Poltekkes Kemenenkes Makassar,
Jl. Wijaya Kusuma No. 46, Kel. Banta-Bantaeng, Kec. Rappocini, Kota Makassar, Prov. Sulawesi Selatan, 90222, Indonesia
Email: wahyudwisuzanti@gmail.com

INTRODUCTION

Diabetes mellitus (DM) is a chronic disease whose prevalence is always increasing significantly worldwide, not only in adults but also in children. Diabetes mellitus is characterized by increased blood sugar levels due to impaired insulin production, impaired insulin function, or both. In children, the most common type of DM is type 1, an absolute insulin deficiency occurs due to damage to pancreatic gland cells by an autoimmune process (Pulungan, A et al., 2019). IDAI data in 2018 recorded 1220 children suffering from type 1 DM. The incidence of type 1 DM in children and adolescents increased approximately 7-fold from 3.88 to 28.19 per 100 million population from 2000 to 2010 (Pulungan, A et al., 2019). One of the complications that occurs in patients with type 1 diabetes mellitus is diabetic ketoacidosis (Nusantara et al., 2023). Data from 2017 showed that 71% of children with type 1 DM were first diagnosed with Diabetic Ketoacidosis (DKA), an increase compared to the proportion in 2015 and 2016, which were 63% each (Ministry of Health of the Republic of Indonesia, 2024).

Type 1 diabetes mellitus impacts children's growth, development, nutritional status, and quality of life. Insulin deficiency causes impaired carbohydrate, fat, and protein metabolism, increasing the risk of weight loss, malnutrition, growth retardation, and acute and chronic complications. Diabetic ketoacidosis (DKA) is an acute complication due to hyperglycemia that is most often found in children with type 1 diabetes mellitus and remains a leading cause of death in children with type 1 diabetes (Kostopoulou et al., 2023). Ketoacidosis is also an early clinical sign in children with type 1 diabetes (Elendu et al., 2023).

DKA can be another complication such as hyperglycemia also induces osmotic diuresis, which causes severe dehydration and electrolyte imbalance (e.g., hyponatremia, hypokalemia, hyperkalemia, hyperchloremia, hypophosphatemia, hypocalcemia and hypomagnesemia), Both hyperglycemia and increased ketone levels worsen osmotic diuresis, resulting in hypovolemia and decreased glomerular filtration rate, which further exacerbates hyperglycemia (Reynaldo, 2022). Hyperglycemia combined with ketoacidosis, triggers the release of proinflammatory cytokines and oxidative stress, which further impairs insulin secretion and sensitivity (Fajriah, 2022; Foti Randazzese et al., 2025).

Management of type 1 diabetes mellitus in children requires a multidisciplinary approach that includes insulin therapy, blood glucose monitoring, medical nutrition therapy, family education, and continuous monitoring of complications. Nutrition therapy plays an important role in meeting nutritional needs, maintaining optimal growth, improving glycemic control, and preventing worsening nutritional status (American Diabetes Association Professional Practice Committee, 2025; Besser et al., 2022). This study aims to describe the implementation of the nutritional care process in pediatric patients diagnosed with Severe Diabetic Ketoacidosis, Hyperglycemia *et causa* suspected type 1 Diabetes Mellitus, Severe Dehydration, *Community Acquired Pneumonia, Anemia, Hypokalemia, Azotemia, AKI KDIGO II, and Marasmus* Type Malnutrition. The results of this study are expected to be the basis for optimizing nutritional interventions to support the success of medical therapy and improve patient clinical outcomes.

This case report provides three main scientific contributions to pediatric nutritional therapy. First, it demonstrates the practical application of the Standardized Nutrition Care Process (SNCP) using the Problem–Etiology–Signs/Symptoms (PES) approach in managing a critically ill child with multiple comorbidities, including severe diabetic ketoacidosis, acute kidney injury, pneumonia, and marasmus. Second, it illustrates how gradual nutritional advancement combined with carbohydrate counting can be safely implemented in a high-risk pediatric patient while minimizing the risk of refeeding syndrome. Third, this report provides practical clinical evidence supporting individualized nutrition therapy as an integral component of multidisciplinary management in pediatric patients with severe metabolic disorders.

Although several studies have discussed nutritional management in pediatric type 1 diabetes mellitus and diabetic ketoacidosis, reports describing the complete implementation of the Standardized Nutrition Care Process (SNCP) in children with severe DKA accompanied by acute

kidney injury, pneumonia, severe malnutrition, and multiple metabolic complications remain limited. Existing literature generally focuses on medical stabilization and insulin therapy, whereas evidence regarding individualized nutritional diagnosis, intervention planning, monitoring, and evaluation is still scarce. Therefore, this case report aims to address this gap by presenting comprehensive nutritional management using the SNCP framework.

RESEARCH METHODS

This research is an analytical observational study with a case study approach that applies the Standardized Nutrition Care Process (PAGT). The study was conducted on a pediatric patient undergoing treatment in the Pediatric Intensive Care Unit (PICU) of Dr. Wahidin Sudirohusodo General Hospital, Makassar, in March – April 2026. The subject of the study was a female pediatric patient and observations were conducted for four consecutive days, from March 31 to April 3, 2026.

Data collection was conducted through a comprehensive nutritional assessment according to the PAGT stages. The collected data included anthropometric data in the form of weight, height, and nutritional status; biochemical data obtained from laboratory tests; clinical data and a nutrition-focused physical examination (*NFPE*); and data on the patient's personal history, medical history, nutritional history, and food consumption patterns. Data were obtained through direct observation, interviews with the patient's family, and a review of medical records. Data analysis was conducted descriptively based on the PAGT stages, which included nutritional assessment, determining a nutritional diagnosis using the *Problem-Etiology-Signs/Symptoms* (PES) format, planning and implementing nutritional interventions, and monitoring and evaluation. The results of the analysis were used to describe the patient's nutritional condition, nutritional problems identified, interventions provided, and changes in the patient's condition during the observation period.

RESULTS AND DISCUSSION

Assessment

Patient An. A, a 1 year 3 months old female, was treated in the Pediatric Intensive Care Unit (PICU) of Dr. Wahidin Sudirohusodo General Hospital, Makassar with a diagnosis of Severe Diabetic Ketoacidosis, Hyperglycemia *and* suspected cause of Type 1 Diabetes Mellitus, Severe Dehydration, *Community Acquired Pneumonia , Anemia, Hypokalemia, Azotemia, AKI KDIGO II, and Marasmus* Type Malnutrition . The main complaint upon admission was shortness of breath since one day before admission which worsened eight hours before treatment, accompanied by weakness. The patient is the second of two siblings, born spontaneously with a birth weight of 3.2 kg, has a history of shrimp allergy, complete immunization history, and was previously diagnosed with Down syndrome at the community health center. The results of nutritional screening using *the Screening Tool for Risk on Nutritional Status and Growth* (STRONGkids) and *the Pediatric Yorkhill Malnutrition Score* (PYMS) showed that the patient was at high risk of malnutrition so a comprehensive nutritional assessment was needed.

Anthropometric data showed a body weight of 7.8 kg, body length of 80 cm, and upper arm circumference (LILA) of 13.5 cm. Nutritional status based on %LiLA of 83.3% was categorized as malnutrition, while the Z-score value showed W/PB -3 SD (malnutrition), W/A -1 SD (normal), and PB/A +1 SD (normal). A focused physical examination of nutrition showed the presence of gambang ribs, a face that looked older than biological age, and a history of weight loss in the past month. The patient's general condition was weak with compos mentis consciousness (GCS E4V5M6). Vital signs included a respiratory rate of 22 breaths/minute, a pulse of 100 beats/minute, an oxygen saturation of 98%, and a body temperature of 36.1°C. The patient also appeared short of breath, weak, and had impaired visual focus. Laboratory results from the referral hospital showed an HbA1c of 11.1%, a random blood glucose of 850 mg/dL, a urea of 123 mg/dL, a creatinine of 0.9 mg/dL, and an eGFR of 48 mL/min/1.73 m^2. Further examination at Dr. Wahidin Sudirohusodo

General Hospital showed a random blood glucose of 238 mg/dL, which still indicated hyperglycemia. A chest radiology examination suggested bronchopneumonia.

Nutritional history showed that before the illness, the patient consumed three main meals a day with two meals of formula milk a day, but the consumption of vegetables, fruits, and vegetable protein was still less varied. The results of the 24-hour intake *recall* before the intervention showed that the patient received enteral nutrition via NGT in the form of formula milk and parenteral nutrition. Enteral nutrition was given in the form of formula milk via NGT with 8 times 30 cc, with 167 kcal of energy, 5.2 grams of protein, 6.2 grams of fat, and 21.8 grams of carbohydrates. Parenteral nutrition 10% dextrose 10cc/hour, which provided an additional 64.6 grams of energy and 16.1 grams of carbohydrates. The total intake recall results were inadequate, with energy 231.5 kcal (40.61%), protein 5.2 grams (18.25%), fat 6.2 grams (32.8%), and carbohydrates 38 grams (47.5%), thus not meeting the patient's energy and nutritional needs. Nutritional support was administered in stages, given the patient's critical clinical condition, to prevent *refeeding syndrome* . Furthermore, the patient's family had not previously received nutrition education, necessitating counseling regarding dietary management for type 1 diabetes mellitus and meeting nutritional needs during treatment and after discharge.

Diagnosis

Based on the results of the nutritional assessment and identification of nutritional problems, the following nutritional diagnosis was obtained: a) NC-2.2 changes in nutrition-related lab values related to endocrine and other organ dysfunction are characterized by an increase in HbA1c examination of 11.1%, GDS >200mg/dl Ureum 123 mg/dl, Creatinine 0.9 mg/dl and a decrease in HGB 10.1 g/dl and e-GFR 48%; b) NC-4.1.5 Child malnutrition related to disease is associated with changes in nutrient absorption characterized by a Z score of BB/PB -3SD, % LiLA 83.3% and the presence of iga gambang and intake <50% of requirements during the last 1 month.

Intervention

Dietary interventions aim to meet and increase nutritional intake according to the patient's nutritional needs to prevent further tissue catabolism. Furthermore, dietary interventions aim to gradually improve nutritional status and prevent further weight loss, considering the patient's condition of malnutrition due to illness. Adequate nutritional intake is expected to aid the healing process, restore body function, and improve muscle mass. Intake is carried out gradually to prevent *refeeding* and improve the patient's ability to accept food, as critically ill patients require prior adaptation before achieving adequate nutritional intake. Another goal of intervention is to help reduce problematic laboratory values (HbA1c and GDS) through carbohydrate *counting* , which calculates the total amount of carbohydrates consumed. This aims to help manage blood sugar by matching the amount of carbohydrates consumed with the insulin dose prescribed by the doctor, thereby avoiding blood sugar that is too high (*hyperglycemia*) and too low (*hypoglycemia*).

The patient was given a dietary intervention for diabetes mellitus, namely a 1100 kcal DM diet. The diet was administered via the NGT route using an enteral formula, given 8x85ml. Basal energy requirements were calculated using *the Schofield eq,* resulting in an energy requirement of 570 kcal. The patient's protein requirement was set at 20% of the total energy, namely 28.5 grams per day, to help the body's tissue repair process. Fat intake was given at 30% of the requirement of 19 grams. In addition, carbohydrate requirements were given at 50% of the total energy, namely 80 grams per day, adjusted to the insulin dose given by the doctor to avoid an increase in blood sugar (*hyperglycemia*) or a decrease in blood sugar (*hypoglycemia*).

In the initial phase of dietary intervention, the target intervention is to meet daily basal metabolic needs. Intervention is provided gradually according to the patient's ability, by increasing initial intake to meet daily basal needs and continuously increasing it according to tolerance until optimal nutritional needs are met, monitoring the patient's progress. Dietary intervention is carried out with the hope that the patient's nutritional needs will be optimally met, thus supporting the healing process and gradually improving the patient's nutritional status.

Monitoring and Evaluation
Anthropometry

Anthropometric data before and after the intervention showed that the measurement results showed that there was no change in anthropometric data before and after the intervention of the patient's weight 7.8 kg, height 80 cm, LiLA 13.5 cm and %LiLA 83.3%. Based on the BB/U indicator, it shows that the patient has a normal weight. Then based on the PB/U indicator, it shows that the patient has a normal height. Meanwhile, based on the BB/PB, the patient was found to be in the category of malnutrition, which indicates that the patient's weight does not match his height. Then the percentage of LiLA obtained results of 83.3% included in the category of malnutrition and the presence of ribs and the overall appearance of the patient was thin. Based on these data, a nutritional diagnosis of NC-4.1.5 Malnutrition was established related to increased nutritional needs due to illness characterized by a Z score of BB/PB -3SD, %LiLA 83.3% and the presence of ribs. This condition is caused by insulin deficiency and poor metabolic control which results in weight loss due to the body being unable to process glucose, thus breaking down fat and muscle reserves, resulting in growth disorders where weight and height do not increase adequately (Padmita Utami et al., 2024; Pulungan, A et al., 2019).

The effectiveness of nutritional intervention was evaluated using predefined monitoring indicators consisting of: (1) achievement of daily energy and macronutrient intake (% of requirements); (2) changes in blood glucose levels; (3) anthropometric measurements including body weight, weight-for-length Z-score, and MUAC; (4) improvement of clinical signs observed through Nutrition Focused Physical Examination (NFPE); and (5) patient tolerance to enteral nutrition without evidence of refeeding syndrome or feeding intolerance.

Table 1 Results of anthropometric data monitoring

Anthropometry	Initial Intervention	End of Intervention
Weight	7.8 kg	7.8 kg
Height	80 cm	80 cm
LiLA (cm)	13.5%	13.5%
LiLA Percentile	83.3% (Malnutrition)	83.3% (Malnutrition)
Nutritional status	BB/U : Normal	BB/U : Normal
	PB/U : Normal	PB/U : Normal
	BB/TB : Malnutrition	BB/TB : Malnutrition

Children with type 1 diabetes mellitus (DM) with diabetic ketoacidosis are complicated by insulin deficiency associated with metabolic control, which significantly disrupts glucose, protein, and fat metabolism. Insufficient insulin leads to uncontrolled hepatic glucose production by the liver and kidneys, impaired peripheral glucose uptake, and increased lipolysis, which contribute to ketogenesis. This insulin deficiency mimics the pathophysiological mechanisms of hypoglycemia, triggering the release of *counter-regulatory hormones* (e.g., cortisol, glucagon, catecholamines, and growth hormone), which increase serum glucose levels by reducing peripheral glucose utilization, promoting hepatic gluconeogenesis, and stimulating glycogenolysis. In addition, lipase activity in adipose tissue increases, breaking down triglycerides into glycerol and increasing levels of free fatty acids, which are converted into ketones, such as acetoacetate and β-hydroxybutyrate, through hepatic ketogenesis. Among these ketones, β-hydroxybutyrate predominates and contributes significantly to the development of metabolic acidosis (Foti Randazzese et al., 2025).

In addition, DKA can have other complications such as hyperglycemia also induces osmotic diuresis, which causes severe dehydration and electrolyte imbalance (eg, hyponatremia, hypokalemia, hyperkalemia, hyperchloremia, hypophosphatemia, hypocalcemia and hypomagnesemia), Both hyperglycemia and increased ketone levels worsen osmotic diuresis, resulting in hypovolemia and decreased glomerular filtration rate, which further exacerbates hyperglycemia (Reynaldo, 2022). Hyperglycemia combined with ketoacidosis, triggers the release of proinflammatory cytokines and oxidative stress, which further impairs insulin secretion and sensitivity (Fajriah, 2022; Foti Randazzese et al., 2025).

Severe DKA can cause various complications, ranging from metabolic and electrolyte changes to life-threatening systemic conditions, which can lead to multiple organ failure and ultimately death if they are not recognized and treated promptly. One of the short-term complications of DKA is *Acute Kidney Injury (AKI) and Acute Respiratory* Distress Syndrome (ARDS) (Foti Randazzese et al., 2025). If this condition persists for a long time, it will accelerate the occurrence of malnutrition and worsen the child's nutritional status. In children with type 1 DM with complex complications such as diabetic ketoacidosis accompanied by hyperglycemia, severe dehydration, AKI, CAP, and hypokalemia, it is very important to carry out adequate nutritional interventions and regular anthropometric monitoring to prevent further decline in nutritional status and help the healing process of the disease.

Biochemistry

Table 2 Laboratory examination

Inspection	Reference Value	Before Intervention	Day 1 intervention	Day 2 intervention	Day 3 intervention
GDS	<140 mg/dl	*03/29/2026* GDS: 238 mg/dl (01.00) GDS: 229 mg/dl (02.00) GDS: 207 mg/dl (03.00) GDS: 194 mg/dl (04.00)	GDS: 130 gr/dl (06.47) GDS: 126 gr/dl (12.25)	GDS: 157 gr/dl (00.55) GDS: 139 gr/dl (06.52) GDS: 174 gr/dl (18.02)	
WBC	4-10 10 ^3/uL	16.28 10^3/ul			No laboratory tests
HGB	12-16 g/dl	10.1 gr/dl			
Creatinine	<1.1 mg/dl	0.9 mg/dl			
Potassium	3.5-5.1 mmol/l	3.3 mmol/l			
Chloride	97-111 mmol/l	116 mmol/l			
SGOT	<38 U/L	220 U/L	No laboratory tests	No laboratory tests	
SGPT	<41 U/L	92 U/L			
Albumin	3.5-5.0 gr/dl	4.1 gr/dl			
urea	10-50 mg/dl	123 mg/dl			
Sodium	136-145 mmol/l	147 mmol/l			
e-GFR	80.4 – 117.8 %	48%			

Source: secondary data

The results of the patient's laboratory examinations were carried out at the beginning of the intervention, during the intervention and at the end of the intervention, but at the end of the intervention there were no further laboratory examinations. The results of laboratory examinations before the intervention showed an increase in HbA1c values of 11.1%, random blood sugar (GDS) of 171 gr / dl and a decrease in HB values of 10.1 gr / dl and Ureum 123 mg / dl, Creatinine 0.9 mg / dl and e-GFR 48%. Based on the data, a nutritional diagnosis of NC-2.2 was established changes in laboratory values related to nutrition related to endocrine and other organ dysfunction characterized by an increase in HbA1c examination of 11.1%, GDS >200mg / dl Ureum 123 mg / dl, Creatinine 0.9 mg / dl and a decrease in HGB 10.1 g / dl and e-GFR 48%.

The first day of intervention there was a drop in GDS examination, at six in the morning it was 130 gr/dl and at twelve in the afternoon it dropped to 126 gr/dl, which was within normal limits. On the second day of intervention, the GDS value increased again at 1 pm at 157 g/dl and fell at 7 am to 139 gr/dl then rose again to 174 gr/dl. This indicates that the GDS value during the intervention was still fluctuating. The criteria used by doctors to diagnose patients with type 1 diabetes include hyperglycemia, plasma glucose ≥ 200 mg/dl, and HbA1c > 6.5% according to the *National Glycohemoglobin Standardization Program* (NGSP) standards in a certified laboratory (Pulungan, A et al., 2019) . This is in line with data obtained from the beginning to the end of the intervention. High HbA1c values (> 15) in patients reinforce poor glycemic control and lack of

blood sugar monitoring. Children with diabetes are at risk of growth disorders due to the disease process or its complications. Poor metabolic control can result in growth disorders where weight and height do not increase adequately. During the intervention, fluctuations in random blood sugar (GDS) may be caused by absolute insulin deficiency due to autoimmune damage to pancreatic beta cells, which makes the body unable to regulate glucose independently. The main triggers include the dawn phenomenon (increased morning growth hormone/cortisol levels), the *Somogyi effect* (*rebound* hyperglycemia after nocturnal hypoglycemia), inappropriate insulin dosage, physical activity, and infection (Islami & Kurniawati, 2022).

Elevated serum urea and creatinine levels are key indicators of impaired kidney function or Acute Kidney Injury (AKI) in patients with type 1 diabetes mellitus (DM) with Diabetic Ketoacidosis (DKA). In patients with type 1 diabetes mellitus (DM), DKA is often the primary cause of sudden onset of AKI. Diabetic ketoacidosis causes severe dehydration and osmotic diuresis, leading to decreased renal (pre-renal) perfusion. A disproportionate increase, with urea levels exceeding serum creatinine values, indicates prerenal azotemia, an early form of AKI in DKA (Chen et al., 2020). AKI in diabetic ketoacidosis is usually transient. However, close monitoring is necessary to prevent permanent kidney damage. Therefore, patients still require adequate nutritional intervention and regular monitoring to help improve problematic laboratory values and support the recovery process.

Clinical Data And Physique Focus Nutrition

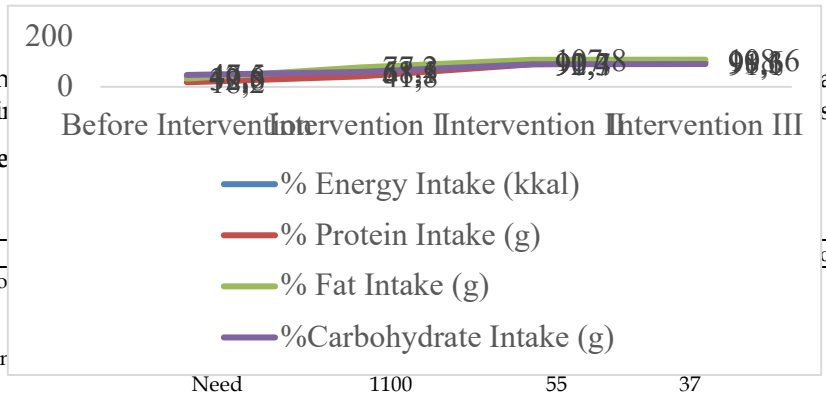
Table 3 Results monitoring physique nutritional and clinical focus

Parameter	Initial Intervention	The day I	Day II	Day III
Awareness (GCS)	15 (E4M6V5) Compost mentis	15 (E4M6V5) Compost mentis	15 (E4M6V5) Compost mentis	15 (E4M6V5) Compost mentis
General Condition	Weak	Good	Good	Good
RR (x/minute)	22x/minute	24x/minute	24x/minute	24x/minute
Pulse (x/minute)	100x/minute	107x/minute	105x/minute	110x/minute
Temperature (°C)	36.1	36.5	36.5	36.7
SpO_2 (%)	98	98	98	98
Gambang Ribs	There is	There is	There is	There is
Congested	There is	Reduce	Reduce	Reduce
Weak	There is	There is	Reduce	There isn't any
The patient's eyes cannot focus	There is	There is	There is	There is

Source: Data Secondary, 2026

Patient physical data is data obtained from the signs and symptoms experienced by the patient. Physical examinations are carried out daily. Based on Table 3, it was found that the patient's physical condition had improved until the last day of observation of the patient's physical condition during the assessment, namely with complaints of rib gambang, reduced shortness of breath, no weakness, the patient's eyes could not focus. The patient experienced shortness of breath and weakness on the first day of intervention and reduced on the following day. During the intervention, the patient still had rib gambang.

A physical examination of the rib cage suggests an additional diagnosis of marasmus. The rib cage is a clinical sign indicating muscle atrophy and loss of subcutaneous fat in the chest, resulting in prominent ribs. In children with type 1 diabetes mellitus, this condition often indicates complications of malnutrition. In children with type 1 diabetes, insulin deficiency is associated with impaired metabolic control, leading to significant disturbances in glucose, protein, and fat metabolism. Insufficient insulin leads to uncontrolled hepatic glucose production by the liver and kidneys, impaired peripheral glucose uptake, and increased lipolysis, which contributes to ketogenesis. Newly diagnosed type 1 diabetes mellitus (DM) patients, as in this case, often result from inadequate nutritional management and uncontrolled diabetes (Foti Randazzese et al., 2025). Overall, the patient's condition demonstrates a complex interaction between disease and malnutrition. This leads to increased metabolic demands and decreased muscle mass and fat tissue. Therefore, patient



managemen apy and adequate nutritional i ss.

Data Intake

					ohydrate (gr)
Befo					38.0
					80.0
					47.5
Ir					82.0
	Need	1100	55	37	140
	% Intake	61.1	41.8	77.2	58.5
Intervention II	Intake	1019.6	50.2	39.4	126.6
	Need	1100	55	37	140
	% Intake	92.7	91.3	107.8	90.4
Intervention I	Intake	1081.6	54.8	39.8	127.5
	Need	1100	55	37	140
	% Intake	98.3	99.6	108.6	91.1

Source: Data Primary, 2026

Patient nutritional intake is data on the patient's daily food and beverage intake orally. Monitoring of patient intake was carried out using *food weighing,* or measuring the patient's initial weight and remaining food weight, and a 24-hour *recall* , which included interviews with the patient's family regarding the patient's daily intake. The diet provided during the intervention was a 1100 kcal DM diet, administered gradually starting at 50% of daily needs until optimal levels were reached. Table 4 shows that the patient's intake increased until it reached optimal levels. The patient was now able to consume 90-110% of daily needs. Further increases in intake are needed, taking into account the patient's clinical physical condition and modifying the food intake to suit the patient's tolerance.

Based on graph 1, it was found that the patient's intake was good, seen from the beginning to the end of the intervention, the patient's intake had reached >80% of the menu plan that had been made. Overall, the monitoring results showed that the nutritional intervention provided was able to increase energy and macronutrient intake gradually over the three days of intervention. The trend of increasing intake reflects a good response to the intervention and serves as a basis for continuing nutrition therapy gradually and adjusting the patient's tolerance to achieve optimal target needs. The nutritional intake intervention was carried out for 3 days, starting from March 31, 2026, to April 2, 2026. Intake was given gradually, taking into account the patient's abilities. On the first day of the intervention, a change in the administration plan was made, which was initially given according to *the basal metabolic rate* (BMR) calculated by *Schofield. eq* with enteral feeding of 8x80 ml to 3x120 ml enteral feeding with minced side dish porridge on the first day of intervention. This occurred due to the improvement in the patient's condition and ability to accept food, so that nutritional intake was provided according to daily needs using the RDA formula with the need for energy 110 kcal, protein 55 grams, fat 37 grams and carbohydrate 140 grams. Nutritional intake continued to increase, reaching optimal levels on the third day of intervention.

Figure 1 Results monitoring intake nutrition

Energy intake in children with type 1 diabetes is recommended using ideal body weight because this is to prevent weight loss and encourage optimal growth and development (Kaushik et al., 2024). Protein intake based on ADA 2013 recommends 15–20% of total daily energy with an individualized approach considering factors such as the patient's metabolic status (e.g., lipid profile, kidney function) and/or food preferences (AMERICAN DIABETES ASSOCIATION, 2013). Fat intake has clinical manifestations of cardiovascular disease that generally appear in adulthood but vascular damage may occur early in patients with type 1 diabetes (Kaushik et al., 2024). A study shows that a high-fat diet and increased energy intake above daily needs impairs metabolic control of diabetes (Mackey et al., 2018). and the relationship between higher HbA1c concentrations and high intake of SFA, MUFA, and total fat (Delahanty et al., 2009). Providing adequate fat intake is done to prevent further complications.

Carbohydrate intake has the greatest influence on blood glucose levels. Carbohydrate monitoring is necessary to control glucose fluctuations, which can have a positive impact on reducing blood sugar levels, reducing HbA1c levels, and preventing further complications (Ewers et al., 2019). *Carbohydrate Counting* is a tool to ensure that carbohydrate intake is in accordance with the insulin dose given. Several studies have shown that *Carbohydrate Counting* has a positive impact on metabolic control, reducing the incidence of hypoglycemia, lowering HbA1c, and providing better blood glucose regulation (Fu et al., 2016; Gökşen et al., 2014; Tascini et al., 2018).

In the intervention, the patient's family was educated about *carbohydrate counting* according to the daily meal allocation. Carbohydrate counting was 20% (28 grams) for breakfast, 25% (35 grams) for lunch and dinner, and 10% (14 grams) for three snacks a day. The *carbohydrate counting approach* is expected to help the patient's family better manage their daily carbohydrate consumption. The education provided to the patient's family demonstrated a positive understanding of the diet and how to implement it. Clinical guidelines for the management of type 1 diabetes recommend adjusting the insulin dose to the overall macronutrient composition of the diet to control blood sugar. Insulin, calculated for carbohydrates, protein, and fat, is effective in controlling post-meal glycemia in patients using insulin (*ADA,* 2020). Patients with type 1 diabetes who consult with a dietitian as part of their treatment plan may have better adherence to the recommended diet and have better glycemic control (Kaushik et al., 2024).

The gradual increase in energy and macronutrient intake was clinically associated with improved dietary tolerance, stabilization of blood glucose levels, reduction of weakness and respiratory distress, and achievement of nutritional intake targets exceeding 90% of estimated requirements. Although anthropometric improvement was not observed because of the short follow-up period, adequate nutritional intake is expected to reduce tissue catabolism, preserve lean body mass, improve immune function, and support recovery during the acute phase of illness.

The findings emphasize that nutritional care should be initiated as early as possible in pediatric patients with severe DKA and malnutrition. Individualized nutritional assessment, gradual advancement of nutritional support, carbohydrate counting, and continuous monitoring of metabolic and nutritional parameters should become integral components of multidisciplinary management. These strategies may reduce nutritional deterioration, improve metabolic stabilization, and optimize recovery in critically ill pediatric patients.

CONCLUSION

A 1 year 3 months old female child with a medical diagnosis of Severe Diabetic Ketoacidosis, Hyperglycemia *et causa* suspected Diabetes Mellitus type 1, Severe Dehydration, *Community Acquired Pneumonia* , Iron Deficiency Anemia dd/ Chronic Disease Anemia, Hypokalemia, Azotemia, AKI KDIGO II, *Marasmus Type Malnutrition* . The established nutritional diagnosis is NC-2.2 changes in laboratory values related to nutrition related to endocrine and other organ dysfunction characterized by increased HbA1c, GDS, urea, creatinine examinations and decreased

e-GFR and HGB. The second nutritional diagnosis is NC-4.1.5 Child malnutrition related to changes in nutrient absorption characterized by nutritional status based on Z score BB/PB and % LiLA malnutrition and the presence of iga gambang and inadequate intake since the last 1 month. The intervention provided was a 1100 kcal DM diet in the form of enteral feeding via NGT and chopped side dish porridge via oral . The patient's intake during the intervention increased to adequate. There was no change in nutritional status due to the relatively short intervention time so that the diagnosis of malnutrition was still established. The patient's family has been provided with education regarding the DM diet and *carbohydrate counting* . Overall, this case emphasizes the importance of proper, gradual, and monitored nutritional management in patients with type 1 DM with complications to support recovery and prevent the risk of further complications. In addition, a multidisciplinary approach is needed for providing nutritional therapy according to the patient's clinical development and metabolic response. Future studies involving larger sample sizes and prospective multicenter designs are recommended to evaluate the effectiveness of individualized nutritional interventions in pediatric patients with severe diabetic ketoacidosis and malnutrition. Comparative studies evaluating different nutritional support strategies and longer follow-up periods are also needed to determine their effects on nutritional recovery, glycemic control, growth outcomes, and long-term clinical prognosis. The SNCP provided a structured, individualized nutritional management approach that supported metabolic stabilization and improved nutritional status in pediatric patients with severe DKA and malnutrition.

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