



Olive oil and Vegetable Extract in Modified Hospital Enteral Formula Improves Glycemic Variability in Critically-Ill Diabetic Ketoacidosis Obese Patient: A Case Report

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ABSTRACT

Background: Severe hyperglycemia in diabetic ketoacidosis may elevate pro inflammatory cytokines, oxidative stress, and metabolic disruptions, impacting the nutritional status of critically ill patients. Diabetes-specific formula (DSF) administration is linked to favorable glycemic control, but research on the role of modified hospital enteral formulas in diabetic critical illness is lacking.

Case Illustration: An obese 29-year-old male at risk of malnutrition, presented to the emergency room with decreased level of consciousness due to metabolic encephalopathy, diabetic ketoacidosis due to suspected type 1 diabetes mellitus, hypertension, and acute kidney injury. Medical nutritional therapy was provided via enteral route according to recent ESPEN, ASPEN and ADA recommendation. The administered enteral formula was a modified hospital-based enteral formula, consisting of a special kidney hospital-based enteral formula mixed with olive oil as source of monounsaturated fatty acid (MUFA) and vegetables as source of fibers. During the first week of hospitalization, the patient's coefficient of variation (%CoV) of glycemic variability ranged between 17–61%, in addition, at the beginning of the second week of treatment there was also an increase in glycemic variability to 53%. This could be influenced by several factors. However, improvement in glycemic variability was observed in the following days. This improvement was in line with the gradual increase in MUFA and fiber intake, which reached its highest intake during the second week of hospitalization.

Conclusion: Hospital-based enteral formula modified with olive oil and vegetable extract can be made to resemble the nutrients composition of diabetes specific formula and has a favorable effect on glycemic variability.

Keywords: critically ill; diabetic ketoacidosis; glycemic variability; modified enteral formula



**Minyak Zaitun dan Ekstrak Sayuran dalam Formula Enteral
Modifikasi Rumah Sakit Meningkatkan Variabilitas Glikemik pada
Pasien Obesitas dengan Ketoasidosis Diabetik Kritis:
Sebuah Laporan Kasus**

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ABSTRAK

Latar Belakang: Hiperglikemia berat pada ketoasidosis diabetik dapat meningkatkan sitokin proinflamasi, stres oksidatif, dan gangguan metabolisme, berkontribusi pada penurunan status nutrisi pasien kritis. Pemberian formula enteral khusus diabetes terkait dengan variabilitas glikemik yang baik. Dalam ketiadaan formula khusus, penggunaan formula enteral rumah sakit yang dimodifikasi bisa dipertimbangkan, meski penelitian mengenai peran formula tersebut pada pasien diabetes kritis belum ada.

Ilustrasi Kasus: Pasien laki-laki obesitas berusia 29 tahun berisiko malnutrisi, datang ke ruang gawat darurat dengan penurunan kesadaran akibat ensefalopati metabolik, ketoasidosis diabetikum suspek diabetes mellitus tipe 1, hipertensi, dan gagal ginjal akut. Terapi medik nutrisi diberikan melalui rute enteral sesuai dengan rekomendasi ESPEN, ASPEN, dan ADA. Formula enteral yang diberikan merupakan formula enteral rumah sakit yang dimodifikasi, terbuat dari formula enteral rumah sakit khusus ginjal yang dicampur dengan minyak zaitun sebagai sumber asam lemak tak jenuh tunggal dan sayuran sebagai sumber serat. Pada minggu pertama, nilai variabilitas glikemik pasien dengan coefficient of variation (%CoV) berkisar antara 17–61%, pada awal minggu kedua perawatan, juga terjadi peningkatan %CoV mencapai 53%. Hal ini dapat dipengaruhi oleh beberapa faktor. Namun, pada hari perawatan selanjutnya ditemukan perbaikan variabilitas glikemik dengan %CoV berkisar antara 8–19%. Perbaikan variabilitas glikemik ini sejalan dengan peningkatan asupan asam lemak tak jenuh tunggal dan serat secara bertahap dengan asupan tertinggi tercapai pada minggu kedua perawatan rumah sakit.

Simpulan: Formula enteral rumah sakit yang dimodifikasi dengan penambahan minyak zaitun dan sayuran dapat dibuat untuk menyerupai komposisi formula enteral komersil khusus diabetes dan juga memiliki efek menguntungkan pada variabilitas glikemik.

Kata Kunci: formula enteral modifikasi; ketoasidosis diabetik; pasien kritis; variabilitas glikemik

INTRODUCTION

Diabetic ketoacidosis (DKA) is one of the acute, emergency complications of diabetes mellitus (DM). In the USA, the incidence of DKA is 61.6 cases per 10,000 hospital admissions,¹⁻³ with mortality rates reaching up to 30% in certain Asian populations, such as Indians.⁴ A study by Venkatesh et al.⁵ showed a sharp increase in the incidence of DKA patients admitted to ICU from 0.97/100,000 in 2000 to 5.3/100,000 in 2013. Additionally, a half of critically ill patients in the ICU had diabetes as comorbidity. A study by Plummer et al.⁶ also showed that 49.8% of critically ill patients had hyperglycemia, 22% were diagnosed with diabetes and 5.5% had previously unknown diabetes. In Indonesia, there is a scarcity of studies regarding the prevalence of DKA in adults.

DKA in critically ill patients has various adverse effects and is associated with long-term mortality and higher costs.^{7,8} In addition to an increase in osmotic diuresis, hyperglycemia and ketoacidosis also can worsen an increase in proinflammatory cytokines and oxidative stress, as well as metabolic derangements. All of which contribute to decrement of nutritional status in critically ill patients.⁹⁻¹¹ Currently there is a paradigm shift in diabetes therapy for critically ill patients from tight glycemic control with intensive insulin therapy to glycemic variability (GV) control and the prevention of hypoglycemia.¹² Studies have shown that high GV in critically-ill patients is associated with an increased risk of mortality and prolonged ICU stay.¹³⁻¹⁵

Administration of diabetes specific formula (DSF) has been shown to be associated with significant reductions in postprandial blood glucose, mean blood glucose levels, HbA1C, and mean insulin dose thus contributing to good glycemic variability.^{16,17} In the absence of DSF, the use of modified hospital-based enteral formula can be considered. However, there are no studies on the role of modified hospital enteral formula in glycemic control among diabetic patients with critical illness. This case report aims to discuss the role of medical nutrition therapy, particularly modified hospital-based enteral formula on glycemic variability on obese critically ill patient with diabetic ketoacidosis.

CASE DESCRIPTION

A 29-year-old male patient presented to University of Indonesia Hospital emergency room with decreased consciousness since one day prior to admission. The patient had been experiencing severe nausea and vomiting with fever and anorexia for five days prior to admission. The patient brought to a primary care and the physical examination showed a high blood pressure. Antihypertensive medication was administered to the patient and then he was discharged. Three days before admission the vomiting was getting worse, the patient was getting weaker and started losing consciousness. The patient was returned to the previous primary care with initial assessment revealing decreased consciousness, high blood pressure, severe hyperglycemia (1030 mg/dL), positive blood ketones (2.3), hyponatremia (108 mmol/L), hyperkalemia (6.64 mmol/L) and decreased kidney function (creatinine 1.69 mg/dL, eGFR 66.5 mL/min/1.73 m²). Fluid resuscitation was performed, and an insulin drip was started at five units per hour. However, the patient's consciousness continued to decrease, prompting a referral to the University of Indonesia Hospital. In the emergency room of the University of Indonesia Hospital, the patient exhibited delirium with Kussmaul breathing. The laboratory workup revealed leukocytosis (11x10³/uL), hyperglycemia (589 mg/dL), hyponatremia (122 mmol/L), hypoalbuminemia (2.4 g/dL), decreased kidney function (blood urea level 278 mg/dL, creatinine 5.74 mg/dL, and eGFR 12.2 mL/min/1.73 m²). Blood gas analysis showed a metabolic acidosis with an increase in anion gap. Chest x-ray examination showed a bilateral perihilar and paracardial infiltrate in both lungs. The patient diagnosed with DKA, type 1 diabetes, pneumonia and acute kidney injury. Fluid resuscitation was continued, with the administration of vasopressor drug. The patient was admitted to critical care unit, intubated, underwent nasogastric tube insertion and was given seven units per hour drip of insulin. The management provided during ICU stay involved a multidisciplinary team consisting of an anesthesiology specialized in critical care, clinical nutrition specialized in critical care, and a team of dietitians and critical care nurses.

On the first and second day of hospitalization the patient remained on fluid resuscitation and vasopressors with unstable hemodynamic. The patient was given one bag of parenteral nutrition containing 30 grams of amino acids and 75 grams of glucose and was fasted enterally due to a dark and black gastric residue. The patient also received sodium correction with 10 mL per hour of hypertonic 3% sodium chloride infusion. Blood glucose levels fluctuated, with a mean random blood glucose level of 330 mg/dL on day one and 173 mg/dL on day two, respectively. The insulin dosage was gradually reduced from 7 units per hour to 2 units per hour. On the second day of hospitalization, the patient was started on an enteral diet with 18x30 mL of clear liquid, while still receiving supplemental two-chamber parenteral nutrition containing amino acids and glucose.

On the third day of hospitalization, nutritional screening and assessment were performed. Nutritional screening was performed with Nutritional Risk Screening (NRS) 2002 tools with a score of 4 indicating the patient was nutritionally at risk and nutritional assessment was then performed. Anthropometric examination revealed a height of 1.7 m, the upper arm circumference of 34 cm, the estimated body weight of 81 kg and BMI of 28 kg/m². The patient's nutritional status was classified as grade 1 obesity according to the Asia Pacific BMI criteria. According to the ASPEN clinical malnutrition criteria, the patient met only one criteria (decreased intake), and was therefore only diagnosed as at risk of malnutrition. On the third day of hospitalization, the patient was still on vasopressor but the hemodynamic was gradually stabilizing. The patient also began receiving hospital-based enteral formula at a rate of 12x30 mL with a density of 1 kcal per 1 mL. The gastric residues had resolved. The mean blood glucose level was 186 mg/dL, and the patient continued to receive a two-unit-per-hour insulin drip.

The medical nutrition diagnosis included grade 1 obesity, risk of malnutrition, severe hypermetabolism, hypoalbuminemia, hyponatremia, decreased consciousness due to metabolic encephalopathy, diabetic ketoacidosis suspected to be related to type

1 diabetes mellitus, hypertension, and acute kidney injury. Medical nutrition therapy was provided based on European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines for critical illness and kidney injury, American Society for Parenteral and Enteral Nutrition (ASPEN) guidelines for critical illness, and American Diabetes Association (ADA) diabetes nutrition guidelines.^{9, 11, 18-20}

The total energy requirement was 2400 kcal (38 kcal per kg body weight, kcal/kg bw) with 1.5 of stress factors, obtained by the Harris-Benedict predictive equation. The protein intake was targeted to achieve 1–1.3 g/kg bw and the target was increased to 1.3–1.5 g/kg bw when the patient went through kidney dialysis. The target for fat intake was 25–30% of total calories with monounsaturated fat (MUFA) intake of 12–15% of total calories. The carbohydrate was targeted to be not greater than 60% of total calories, emphasis on complex carbohydrate and minimum fiber intake of 14 gram per 1000 kcal. The medical nutrition therapy provided was administered via enteral route through a nasogastric tube while gradually discontinuing the parenteral nutrition. The enteral formula given to the patient was a modified hospital-based enteral formula, made by mixing a special kidney hospital-based enteral formula with olive oil as source of MUFA and vegetables (carrots, or pumpkin, or chayote) as source of fiber. Micronutrients given to the patient included 2x 100 mg of thiamin, 2x50 mg of vitamin C, 3x1 tablet of vitamin B complex, and 1x20 mg of zinc and 1x0.5 mg of folic acid. Monitoring included clinical condition, hemodynamics, respiratory status, enteral feeding tolerance and nutritional intake analysis was performed daily with 24-hours food recall.

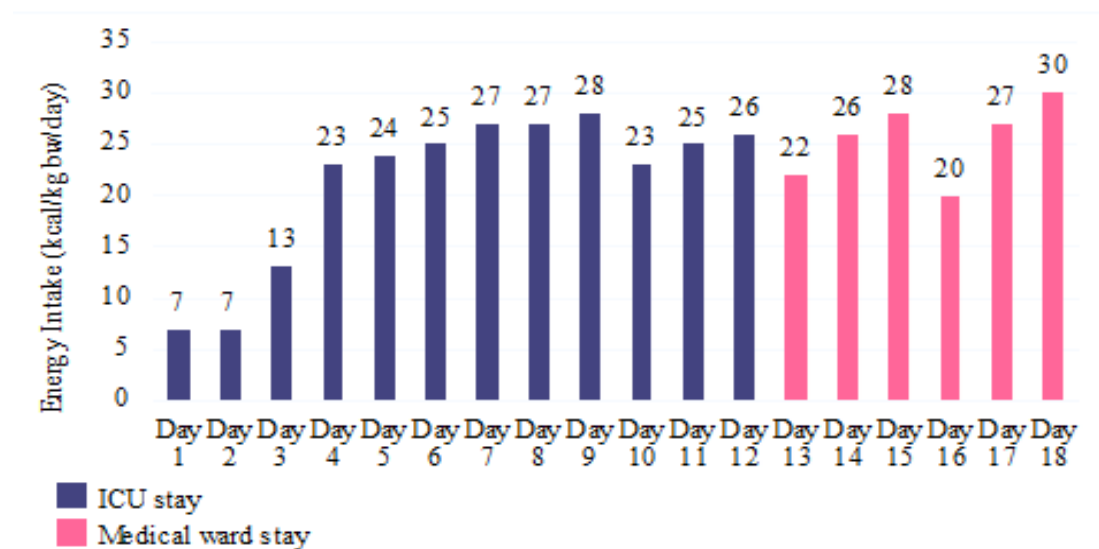
The calorie provision was increased gradually according to the patient's clinical, hemodynamic, respiratory and enteral feeding tolerance. During monitoring in the ICU, the energy intake was targeted at 25–30 kcal/kg bw with energy intake targeted below 70% of predictive equation during the acute phase and gradually achieving 100% of the predictive equation by the end of the first week. The energy provision was then targeted to reach 125–150% of the predictive equation in the rehabilitative period.

On the fourth day of hospitalization, there was an improvement in kidney function with a decreased in blood urea level from 280 to 159 mg/dL and a decreased in creatinine level from 7.38 to 4.32 mg/dL after the patient received supportive hemodialysis (ultrafiltration goal, UFG of 1500 mL). However, on the fifth day of hospitalization there was a further decline in kidney function with blood urea level of 201 mg/dL, creatinine level of 5.07 and eGFR 14.2. The patient continued to receive a full enteral diet with the modified hospital-formula. Overall, during the first week of hospitalization, the patient's level of consciousness began to improve with stable hemodynamic on vasopressor. The patient remained intubated with ventilator support. The nutritional intake was increased with enteral nutrition gradually increased while maintaining the SPN. The patient tolerated the modified hospital formula well, with no gastric residual volume or diarrhea.

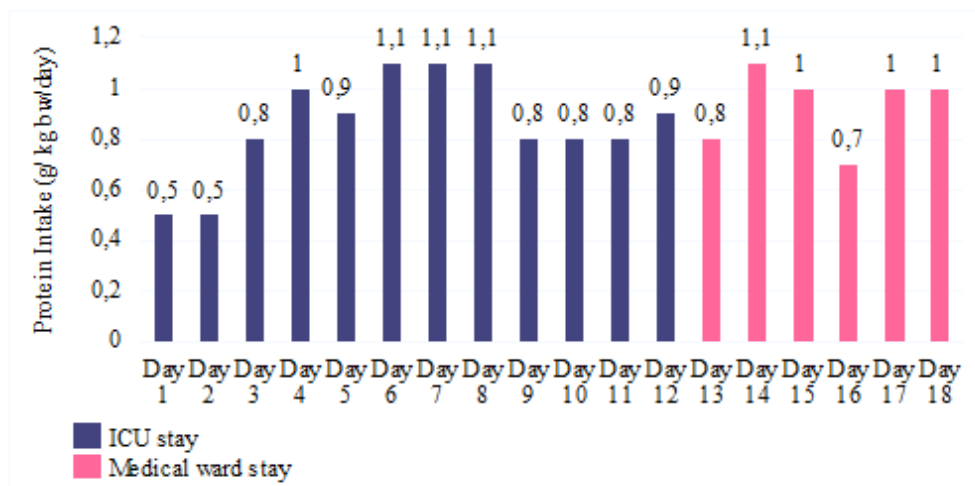
On the second week of hospitalization, the patient was fully conscious, with stable hemodynamic allowing for discontinuation of vasopressor support. The patient was gradually weaned off the ventilator and eventually extubated on the 9th day of hospitalization. Supplemental parenteral nutrition was no longer

given, and the nasogastric tube was removed following extubation. The patients received a full enteral diet with modified enteral formula and gradually transitioned to oral diet with soft food. The patient also received a second supportive hemodialysis session (UFG 1400 mL), during which there was an improvement in kidney function (blood urea level 77, creatinine 2.1, eGFR 41.3). On the 12th day of hospitalization, the patient was transferred to medical ward for further care.

Based on the nutritional analysis, the energy and protein intake during hospitalization was fluctuated with a mean energy intake of 23 kcal/kg bw (7–30 kcal/kg bw) (Graphic 1) and a mean protein intake of 0.9 g/kg bw (0.7–1.1 g/kg bw) (Graphic 2). It was also found that fat intake was 23–30% of total energy, predominantly composed of MUFA that increased gradually until it reached the target of 15% energy requirements. Carbohydrate and fiber intake during monitoring were also aligned with the prescribed target with a mean intake of 59% (52–71%) of total energy and 16 g (0–24 g), respectively. The MUFA and fiber intake can be increased gradually reaching the target especially by administering modified hospital formula with mixed olive oil and vegetables (Table 1).



Graphic 1. Energy intake during hospitalization



Graphic 2. Protein intake during hospitalization

Since the patient was bedridden and unable to stand up, we measured the upper-arm circumference to evaluate the nutritional status. The patient's upper-arm circumference decreased from 34 cm to 32 cm after 2 weeks of hospitalization. Based on the upper arm circumference, the estimated body weight also decreased from 81 kg to 76.6 kg. Physical examination also showed a moderate decrease in muscle mass, as evidenced by a slight depression of temporalis muscle, visible clavicle bone, slightly depressed interosseous muscle, more rounded kneecap, and mild depression on the inner thigh. Therefore, the patient was diagnosed with clinically severe malnourished in the follow-up period. Nonetheless, there was an improvement in functional capacity based on Katz Index, from 1 to 3. In addition, there was also an improvement in kidney function, glycemic variability, and insulin dose during the monitoring period.

The patient's glycemic control fluctuated during the first week of hospitalization with

a coefficient of variation (%CoV) of glycemic variability ranging between 17 and 61%. At the start of the second week of treatment, there was an increase in glycemic variability to 53%. However, in the following days, an improvement in glycemic variability was observed, with %CoV ranging between 8 and 19% (Table 1). This improvement was in line with the gradual increase in MUFA and fiber intake, which reached its highest intake during the second week of hospitalization. The patient also showed an improvement in insulin dose, as the 7-unit-per-hour insulin drip was slowly reduced to 2 units per hour. The intensivist also managed to stop the insulin drip and replaced it with 3x8 units fixed dose of rapid insulin. This patient was successfully transferred from ICU to the medical ward on the 12th day of hospitalization. Finally, the patient was successfully discharged from the hospital on the 18th day of hospitalization, exhibiting improvement in overall condition, functional capacity, kidney function and glycemic variability. (Final outcome)

Tabel 1. Fat, carbohydrate, and fibers intake with glycemic variability and insulin dose during hospitalization

Hospital Days	Fat (% of total calories)	MUFA (% of total calories)	Carbohydrate (% of total calories)	Fibers (g)	Continuous Glucose Monitoring (mg/dL)	Glycaemic variability (%CoV)	Insulin Dose
Day 1	0%	0%	71%	0	589-482-382-324-150-50	61%	Drip 7U/hour
Day 2	0%	0%	71%	0	134-247-216-181-134-124	29%	Drip 4U/hour
Day 3	23%	0%	63%	0	146-166-183-177-212-233	17%	Drip 2U/hour
Day 4	25%	7%	57%	11	277-306-354-289-238-236	16%	Drip 2U/hour
Day 5	26%	5%	59%	22	233-232-225-170-291-318	21%	Drip 2,5U/hour
Day 6	27%	9%	55%	15	287-141-142	44%	Drip 2U/hour
Day 7	28%	9%	56%	22	90-234-186-189	35%	Fixdose 3x8 U
Day 8	29%	9%	56%	22	158-93-275	53%	Fixdose 3x8 U
Day 9	28%	9%	59%	23	154-148-173	8%	Fixdose 3x8 U
Day 10	34%	14%	52%	13	152-155-176	8%	Fixdose 3x8 U
Day 11	25%	13%	63%	15	152-217-162	19%	Fixdose 3x8 U
Day 12	28%	14%	59%	15	196-146-148	17%	Fixdose 3x8 U
Day 13	28%	15%	58%	19	167-158-220	18%	Fixdose 3x8 U
Day 14	27%	15%	56%	24	154-127-134	10%	Fixdose 3x8 U
Day 15	30%	15%	56%	24	125		Fixdose 3x8 U
Day 16	29%	15%	56%	17	174		Fixdose 3x8 U
Day 17	28%	14%	59%	24	142	14%	Fixdose 3x8 U
Day 18	30%	15%	59%	24	139		Fixdose 3x8 U

DISCUSSION

Diabetic ketoacidosis is often the first manifestation of type 1 DM, as in this patient, with the triad of hyperglycaemia, metabolic acidosis and ketosis. The presence of infection as indicated by fever, increased leukocytes, and pneumonia on chest x-ray can trigger the occurrence of DKA.²¹⁻²³ Furthermore, DKA can also cause electrolyte imbalance such as hyponatremia and hyperkalaemia.^{24,25} Renal impairment in the patient can be triggered by several factors including hypovolemia caused by increased osmotic diuresis and emesis, decreased renal perfusion as compensation for

hyperglycaemia, obesity and the critical illness itself.²⁶⁻²⁸

Early enteral nutrition was achieved within 36 hours of admission when the patient got 18x30 mL of clear liquid. Early EN can have a beneficial effect because it supports the integrity of gastrointestinal tract by maintaining intraepithelial cell tight junctions, stimulating gut blood flow and inducing the release of trophic endogenous agents.⁹ However, full energy provision are not recommended in the first 72 hours of the acute phase of critical illness, as endogenous energy production can reach 500-1400 kcal/day during this phase. Provision of energy reaching 100% energy

requirement can result in an overfeeding state and aggravate metabolic impairment. Providing hypocaloric nutrition (below 70% of estimated energy requirements) can be given if energy requirements are calculated using predictive equations. After the third day, the energy delivery can be increased to 80–100% energy requirements.^{17–19} In this patient, the daily intake during the first three days of hospitalization was 7–13 kcal/kg BW, which was less than 70% of the predictive equation target during the acute phase. The patient reached 100% of the energy requirement at the end of the first week of hospitalization. However, during the rehabilitation phase, achieving the energy target of 125–150% of the total energy requirement was more difficult because the patient had started eating orally and had a lack of appetite. The patient was diagnosed with clinically severe malnourished in the follow-up period. This suggests that the provision of energy and protein has not been sufficient to meet the high catabolic process commonly seen in critically ill patient. Some of the obstacles to achieving energy and protein targets include the patient's fasting periods before procedures and the patient's lack of appetite in the rehabilitation phase despite good glycemic variability. In this situation, provision of oral nutrient supplements (ONS) should be considered,¹¹ but it was also difficult because of the patient's lack of appetite which may be caused, among others by the presence of uraemia. In addition, achieving the protein target of 1.3–1.5 g/kg bw was also challenging due to poor kidney function despite the patient receiving supportive haemodialysis. For critically ill patient with acute kidney injury receiving kidney replacement therapy, ESPEN recommends a protein intake of 1.3–1.5 g/kg BW.²⁰ However, in this patient; the kidney function remained poor despite receiving supportive hemodialysis. Therefore, protein provision was gradually increased and only reached a maximum provision of 1.1 g/kg BW. At the end of hospitalization, there was an improvement of kidney function with this moderate protein restriction.

Glycemic variability is defined as fluctuations in blood glucose levels, including upwards fluctuations after hyperglycemic correction

and downward fluctuations in hyperglycemic correction, leading to increased oxidative stress, endothelial dysfunction and vascular damage. Intermittent sharp upwards or downwards fluctuations in blood glucose levels are much more dangerous than constant exposure to persistent hyperglycemia in critically ill patients.^{12,29–31} Exogenous insulin administration is predominantly the current focus for glucose management in critically ill patient. However, the administration of exogenous insulin also contributes to the occurrence of hypoglycemic episodes, thereby increasing glycemic variability.^{29,30}

The administration of diabetes specific formula is associated with a decrease in glycemic load and is expected to reduce insulin requirements, contributing to the improvement of glycemic variability. Meta-analysis by Ojo et al.¹⁶ showed that a diabetes specific formula with high MUFA and high fiber is effective in reducing blood glucose parameters in patients with DM compared to standard formulas. Sanz Paris et al.¹⁷ also showed that provision of DSF with a high MUFA content (more than 20% of total energy from MUFA or more than 40% of total energy from fat) was associated with significant decreased in postprandial blood glucose, mean levels of blood glucose, mean insulin dose, mean triglyceride level and an increase in mean high-density lipoprotein levels. These beneficial effects of MUFA can be achieved through several mechanisms including the anti-inflammatory effects of oleic acid in diminishing insulin resistance by upregulating free fatty acid receptor-4 (FFAR4), enhancing anti-inflammatory macrophages 2 expression, increasing adiponectin and downregulating protein phosphatase 2A which all contributes to promote glucose uptake and enhancing insulin sensitivity.³² On the other hand, the main role of soluble fibers is to improve glycemic control by increasing chyme viscosity, thereby slowing gastric emptying and glucose absorption in the small intestine.³³

In many developed countries, commercial enteral formula including DSF is covered by both private insurance and the national health insurance.³⁴ Unfortunately, in Indonesia, the provision of commercial enteral formula is often

not covered by the national health insurance. In the absence of commercial DSF, our center used a modified kidney hospital-based enteral formula. By adding olive oil as a source of MUFA and vegetables (carrots, or pumpkin, or chayote) as a source of soluble fiber to the enteral formula, our modified enteral formula had a composition that resembled the DSF but with a lower protein content, at a lower price. In terms of glycemic variability, we use a threshold value of 36% for %CoV to determine whether the glycemic variability is favorable or not and improvement was observed in the second week of hospitalization with %CoV ranging between 8–19%.^{35,36}

In the first week of hospitalization, a high %CoV was observed. This could be influenced by several factors, including the presence of stress response in the acute phase, which also plays a role in the occurrence of hyperglycemia, in addition to hyperglycemia caused by the DKA.³⁷ Furthermore, the administration of parenteral nutrition is associated with episodes of hyperglycemia compared to enteral nutrition, thus also linked to an increase in glycemic variability.³⁸ The patient initially received total parenteral nutrition consisting of amino acid and glucose infusion due to the presence of gastric residue at the beginning of the hospitalization. In addition, the administration of insulin can also affect glycemic variability.

At the beginning of the treatment, the patient received a 7 unit per hour of insulin drip, contributing to episodes of hypoglycemia. Study by Hsu et al.³⁹ also indicates that the use of mechanical ventilation is associated with a 1.6-fold increase in glycemic variability. In this case report, the patient received mechanical ventilation until the 7th day of hospitalization. The administration of modified enteral formula was initiated on the third day of hospitalization with initial rate of 12x30 mL (density 1 mL=1 Kcal). Along with the increased amount of enteral nutrition provided as well as the increase amount in total fat percentage, MUFA and fibers content, an improvement in %CoV (less than 36%) was observed, particularly during the second week of hospitalization.

The improvement of glycemic variability in this patient was also accompanied by other positive

impact, including a decrease in insulin dose and a shorter length of stay in the ICU. Study by Kim et al.¹⁵ showed that high GV within 48 hours of ICU admission was associated with prolonged ICU stay (>14 days). This patient was successfully transferred from ICU to the medical ward on the 12th day of hospitalization despite high GV (61%) within 24 hours of ICU admission. This indicates that our nutritional strategies to reduce GV also have favourable effects on ICU length of stay.

Although the patient experienced a decrease in nutritional status during hospitalization, there was an improvement in clinical condition including functional capacity, kidney function, glycemic variability, insulin dose and shorter length of ICU stays (less than 14 days). This case report shows that modification of hospital-based enteral formula can also be made to produce tailor-made formulas adapted to patient needs without relying on commercial formulas. The provision of nutrition continued until the patient was discharged. It was expected that there would be an improvement in nutritional status and insulin administration could be gradually reduced until discontinued. In the future, body composition examinations to assess muscle mass can also be conducted to assess the nutritional status and evaluate the administration of nutritional therapy in critically ill patients.

One limitation of this case report is the single-patient design, which inherently restricts the generalizability of the findings. The unique characteristics of individual patients, including their specific medical history, response to treatment, and comorbidities, may limit the applicability of the results to a broader population. Additionally, the absence of a control group hinders the ability to draw direct comparisons and establish a causal relationship between the modified hospital-based enteral formula and the observed improvements. Further research involving a larger sample size and a comparative study design would be valuable to validate and extend the insights gained from this single-case investigation.

CONCLUSION

Modified hospital enteral formula with olive oil and vegetable extract can be made to resemble the composition of diabetes-specific formula and

has a favourable effect on glycemic variability. Further research is needed to confirm these findings.

CONFLICT OF INTEREST

The authors declare no conflicts of interest related to this manuscript.

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