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Case Report
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Recurrent Diabetic Ketoacidosis in Adolescents with Type 1 Diabetes Mellitus

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Abstract

Introduction: *Diabetic ketoacidosis (DKA) is one of the serious complications that is often found in pediatric cases of type 1 diabetes mellitus (DM). Diabetic ketoacidosis mostly occurs in patients with low glycemic control. Currently, many studies are assessing risk factors for recurrent ketoacidosis, including young age, male gender, patients with comorbidities (psychiatric diseases, alcohol or substance abuse, other chronic diseases), and patients with socio-economic factors. The incidence of recurrent diabetic ketoacidosis in children and adolescents is becoming more frequent. Risk factors that often trigger the incidence of diabetic ketoacidosis are important to understand to reduce the incidence of recurrent DKA.*

Case Presentation: *This study reported a 17-year-old adolescent patient with a history of type 1 diabetes mellitus who had recurrent diabetic ketoacidosis. Patients come with complaints of nausea and vomiting, accompanied by a feeling of weakness. The results of the blood glucose test were 565 mg/dL. In urinalysis, a reduction of glucose was obtained (+3), and ketonuria with urinary ketones was obtained with a result of +3. A blood gas analysis showed a blood pH of 7,367 mmHg with HCO3 of 16.6 mmol/L and PCO2 of 28.6 mmHg. The initial management of the patient was given a loading of 0.9% NaCl fluid as much as 500 cc/hour for 2 hours, then 0.9% NaCl fluid as much as 500 cc mixed with KCL 10 meq at a rate of 75 ml/hour. The patient was also given an insulin drip to correct blood glucose levels in the form of 5 IU of insulin diluted with 0.9% NaCl, as much as 50 cc. During treatment, the patient's condition tends to stabilize, and the patient is discharged from the hospital by continuing with routine treatment with subcutaneous insulin.*

Conclusion: *Diabetic ketoacidosis is one of the complications with high morbidity and mortality, especially in patients with type 1 diabetes mellitus. There are several risk factors associated with recurrent DKA in patients. Known risk factors for DKA allow health workers to prevent the occurrence of a recurrence of DKA.*

Keywords: Recurrent Diabetic Ketoacidosis, Type 1 Diabetes Melitus, Adolescents, Rsik Factors, Therapy

Introduction

Diabetic ketoacidosis (DKA) is one of the serious complications that is often found in the case of children with type 1 diabetes mellitus (DM), where type 1 DM occurs due to absolute or relative insulin deficiency associated with pancreatic B cell destruction. Diabetic ketoacidosis is also the most common cause of death in diabetes mellitus type 1 [1-4]. The incidence of DKA in children varies widely, with the highest incidence found in Finland, which is around 43/100,000, and the lowest incidence found in Japan, which is around 15-2/100,0004. As many as 30% of patients with new-onset type 1 DM experience diabetic ketoacidosis, and as many as 6%–8% of cases of children with type 1 DM experience diabetic ketoacidosis each year. In Indonesia, in 2017, 71% of pediatric patients experienced DKA as an initial clinical presentation of type 1 DM [5, 6].

Diabetic ketoacidosis mostly occurs in patients with poor glycemic control. Relative or absolute insulin deficiency in type 1 DM will reduce the use of glucose in tissues and stimulate the occurrence of lipolysis. Lipolysis, in addition to causing hyperlipidemia, will also lead to the hepatic ketogenesis process, which will cause ketoacidosis. Early signs and symptoms of DKA

include the classic symptoms of DM in the form of polyuria, polydipsia, and weight loss. Patients often experience nausea, vomiting, abdominal pain, tachycardia, hypotension, decreased skin turgor, Kussmaul breathing patterns, and decreased consciousness [7].

Currently, many studies are assessing risk factors for recurrent ketocytosis, including young age, male gender, patients with comorbidities (psychiatric diseases, alcohol consumption or abuse of certain drugs, other chronic diseases), and patients with low socioeconomic factors [8]. DKA predominantly occurs in patients with type 1 diabetes mellitus but is less common in patients with type 2 diabetes mellitus. DKA cases that are diagnosed early in childhood have a greater long-term risk of poor glycemic control, regardless of socioeconomic and demographic risk factors. Interventions aimed at improving the early diagnosis of type 1 DM patients before they develop into DKA are important to reduce the incidence of recurrent DKA [9].

In this case report, the author reports on the case of adolescents with type 1 diabetes mellitus who experience recurrent diabetic ketoasoidosis. The patient received treatment for five days in the pediatric intensive care unit. The incidence of recurrent diabetic ketoacidosis in children and adolescents is becoming more frequent. Risk factors that often trigger the incidence of diabetic ketoacidosis are important to understand. This case report was made to be considered with knowledge about the incidence of recurrent diabetic ketoacidosis in adolescent patients with type 1 diabetes mellitus, so it is hoped that it can identify risk factors and help reduce the re-admission rate of DKA patients, as well as the incidence of illness and mortality of DKA patients in children and adolescents.

Case Presentation

A 17-year-old teenage boy weighing 53 kg and 165 cm tall came to the Emergency Department of Sanjiwani Gianyar Hospital with complaints of nausea and vomiting. The patient complained of vomiting three times, accompanied by complaints of body weakness. Complaints of fever, cough, and shortness of breath were denied. The patient urinated quite a lot two hours before entering the hospital. The patient has a history of type 1 diabetes mellitus and has been undergoing treatment with insulin since elementary school. Patients usually carry out routine control at Prof. Ngoerah Denpasar Hospital. The patient's treatment history was insulin glulisine (16 IU) 3 times a day and insulin glargine (26 IU) 1 time at night. In April 2024, the patient was hospitalized with a diagnosis of diabetic ketoacidosis. The patient had an HbA1c test in April 2024 with a result of 14%, which indicates the possibility that the patient's blood sugar level in the last 3 months was not controlled.

The general examination of the patient appeared weak with compos mentis consciousness. A vital signs examination obtained a pulse rate of 120 x/min with a strong lift, a temperature of 36 oC, a respiratory rate of 16 x/min, a blood pressure of 110/80 mmHg, and an oxygen saturation of 99%. The patient's blood sugar level was 400 mg/dL. In initial management at the emergency room, the patient was given oxygenation with a 2 lpm nasal cannula, then given 0.9% NaCl fluid loading of 500 cc/hour for 2 hours. Supporting examinations performed on patients include a complete blood hematological examination, clinical chemistry, urinalysis, and blood gas analysis examination to confirm the patient's diagnosis.

A complete blood hematological examination (Table 1) showed leukocytosis of 15.08 x 103/uL. Hematocrit was 49.2%, with hemoglobin levels slightly elevated at 16.6 g/dL. Platelet levels were found to be within normal limits at 240 x 103/uL. In the results of the clinical chemistry examination, the result of blood glucose was 565 mg/dL. The patient had increased liver function with the results of SGOT obtained at 87 U/L and SGPT at 76 U/L. The urea level was found to be slightly elevated at 56.6 mg/dL, with a creatinine level of 0.62 mg/dL, which is still within normal limits. Examination of blood electrolyte levels showed hyponatremia with a sodium level of 124 mmol/l. A potassium level of 4.0 mmol/l was found to be within normal limits, and a chloride level of 85 mmol/l was slightly below normal levels. Blood gas analysis (Table 2) showed a blood pH of 7.367 mmHg with HCO3 of 16.6 mmol/L and PCO2 of 28.6 mmHg. The urinalysis (Table 3) showed glucose reduction +3 and ketonuria with urine ketones +3. The initial diagnosis based on the results of the history, physical examination, and support for the patient is diabetic ketoacidosis.

The management given to the patient is treated in the pediatric intensive care unit by providing nasal canal oxygenation at 2 lpm. The patient was given a liquid infusion in the form of 0.9% NaCl, as much as 500 cc, mixed with KCL 10 meq at a rate of 75 cc/hour. The patient was given an insulin drip for blood sugar correction in the form of 5 IU of insulin diluted with 0.9% NaCl at 50 cc, which was dripped at a rate of 5 cc/hour. In addition, patients also receive injections of anti-nausea drugs in the form of ondansentron as much as 4 mg every 8 hours, omeprazole as much as 20 mg every 12 hours, and paracetamol 500 mg if there are complaints of fever or pain. Patients are periodically monitored for blood sugar levels every 3 hours, fluid balance in the form of fluid inlet and outflow, as well as monitoring of signs of consciousness and vital signs. During blood sugar level correction, fluid therapy administered to patients depends on the results of monitoring the patient's sugar level examination. If the patient's blood sugar level is more than 250 mg/dL, the fluid given is NaCl 09%, as much as 500 cc, mixed with KCL 10 mg at a rate of 75 cc/hour. Meanwhile, if the patient's blood sugar level is 150–250 mg/dL, the fluid given is D5 1/2NS, as much as 500 cc, mixed with KCL 10 mg at the same rate, namely 75 cc/hour. If the patient's blood sugar level is less than 150 mg/dL, the fluid given in the form of D10%, as much as 500 cc, is mixed with KCL 10 mg at the same rate, namely 75 cc/hour. The patient's routine insulin injection treatment is delayed until the patient's blood sugar level is stable for 72 hours. For nutrition, the patient was given a high-protein and low-carbohydrate diet.

The patient was evaluated for electrolyte levels, with an improvement in sodium levels of 131 mmol/l, potassium levels slightly below normal of 3.0 mmol/l, and chloride levels within the normal limit of 103 mmol/l. During the 3 days of treatment, the patient experienced improvement. The administration of insulin drips for correction of blood sugar levels was then stopped, followed by the administration of insulin subcutaneously, namely insulin aspart, as much as 16 IU of insulin in the morning, afternoon, and evening before meals, and 26 IU of insulin detemir at night before bedtime. Patients were periodically mon-

itored for blood sugar levels, namely basal blood sugar when they wake up in the morning, then blood sugar before breakfast, lunch, and afternoon, and blood sugar 2 hours after breakfast, lunch, and bedtime. When blood sugar levels are less than 150 mg/dL, insulin administration is delayed.

The results of monitoring the patient's blood sugar level for 24 hours after replacing the therapy with insulin subcutan showed that the patient's blood sugar level tended to be stable below 200 mg/dL. The patient's condition tends to be stable, with compos mentis consciousness and vital signs within normal limits. Patients said there were no complaints. The patient was then allowed to be discharged from the hospital with subcutaneous insulin treatment continued at home, and it was planned for control at the pediatric polyclinic 3 days after treatment.

Table 1: Hematological Examination Result

NAME OF TEST AND INDICATOR	RESULT		UNIT	REFERENCE VALUE
COMPLETE BLOOD COUNT	**08/06/24**	**09/06/24**		
M C V	82.7		fL	80.0 - 100.0
RDW-SD	42.9		fL	35.0 - 56.0
Trombosit (PLT)	240		10^3/uL	150 - 450
M C H	27.8		pg	27.0 - 31.0
RDW-CV	14.2		%	11.5 - 14.5
Bas%	0.0		%	0.0 - 1.0
Hematokrit (HCT)	49.2		%	37.0 - 54.0
Eritrosit (RBC)	5.94		10^6/uL	3.50 - 5.50
Mon#	0.54		10^3/uL	0.12 - 0.80
Neu#	12.29		10^3/uL	2.00 - 7.00
Neu%	81.5		%	50.0 - 70.0
PCT	0.236		%	0.108 - 0.282
Hemoglobin(HGB)	16.6		g/dL	11.0 - 16.0
Lym%	14.9		%	20.0 - 40.0
Bas#	0.00		10^3/uL	0.00 - 0.10
PDW	15.08		fL	9.0 - 17.0
Lekosit (WBC)	3.10		10^3/uL	4.00 - 10.00
MPV	9.8		fL	7.0 - 11.0
Mon%	3.6		%	3.0 - 8.0
Lym#	2.25		10^3/uL	0.80 - 4.00
M C H C	33.7		g/dL	32.0 - 36.0
Eos#	0.00		10^3/uL	0.02 - 0.5
Eos%	0.0		%	0.5 - 5.0
CLINICAL CHEMISTRY				
Blood Glucose	565		mg/dL	80 - 120
SGOT	87		U/L	< 35
SGPT	76		U/L	< 41
Ureum	56.6		mg/dL	18 - 55
Creatinin	0.62		mg/dL	0.2 - 0.7
ELEKTROLIT				
Natrium	124	131	mmol/l	135 – 147
Kalium	4.0	3.0	mmol/l	3.5 - 5.0
Chlorida	85	103	mmol/l	95 - 108

Table 2: Blood Gas Analysis Result

NAME OF TEST AND INDICATOR	RESULT	UNIT	REFERENCE VALUE
BLOOD GAS ANALYSIS			
BE	-9	mmol/L	-2 - +2
AaDO2	15	mmHg	0.0 - 24
tCO2	17	mmol/L	22 - 29
HCO3	16.6	mmol/L	22 - 28
PH	7.367	mmHg	7.35 - 7.45
PO2	99	mmHg	75 - 100
PCO2	28.6	mmHg	35 - 45
SaO2	98	%	94 - 100

Table 3: Urinalysis Result

NAME OF TEST AND INDICATOR	RESULT	UNIT	REFERENCE VALUE
URINALYSIS	08/06/24		
Trichomonas	Negatif		Negatif
Thorax Granuler	Negatif		Negatif
Ca Oxalat	Negatif		Negatif
Bacteri	Negatif		Negatif
Urobilinogen	Negatif		Normal
Triple Phospat	Negatif		Negatif
Epitel Cell	1 - 3	/small field of view	(+) little
Eritrosit	Negatif		Negatif
Lekosit	1 - 2	/large field of view	0 - 5
Fungi	Negatif		Negatif
Uric Acid	Negatif		Negatif
Reduction (Gluc)	+3		Negatif
Sedimen Urine :			
Nitrit	Negatif		Negatif
Thorax Lekosit	Negatif		Negatif
Color	Kuning		Kuning
Specific Gravity (SG)	1.015		1.003 – 1.030
Oval Fat Bodies	Negatif		Negatif
pH/Reaction	5.5		6.0 – 6.5
Bilirubin (Urine)	Negatif		Negatif
Lekosit	Negatif		Negatif
Albumin (Prot. Urine)	Negatif		Negatif
Keton	+3		Negatif
Eritrosit	0 - 2	/large field of view	< 2

Discussion

Diabetic ketoacidosis is one of the complications with high morbidity and mortality, especially in patients with type 1 diabetes mellitus. In young patients with type 1 diabetes mellitus, DKA is the most common complication as a cause of death, with the risk of disease ranging from 1–10% per patient per year. DKA emergencies can lead to fatal advanced complications, with a mortality rate of 5% to 7%. A study conducted in 2006 in 11 Asian countries and the Western Pacific Region stated that the incidence of DKA in pediatric type-1 DM patients occurred at 10 per 100 patients per year. Meanwhile, in Indonesia, in 2017, it was found that 71% of pediatric patients experienced DKA as an initial clinical presentation of type-1 DM. In developing countries, DKA cases are said to be still high. The incidence of DKA in children is actually very varied. DKA in type 1 DM is more often found at a young age, mainly due to late treatment and low socioeconomic status. In type 2 DM, the incidence rate of DKA is much lower than in type 1 DM, which is approximately 25%

of DKA cases. The risk of recurrent DKA in children who have been diagnosed with type 1 DM is 1–10% per year. This case report discusses a 17-year-old adolescent male patient with type 1 diabetes mellitus who has recurrent diabetic ketosis.

Diabetic ketoacidosis (DKA) is biochemically defined as a venous pH <73 or a serum bicarbonate concentration <15 mmol/L and a serum glucose concentration >200 mg/dL (11 mmol/L) along with ketonemia, glucosuria, and ketonuria [10]. DKA is caused by a relative or absolute insulin deficiency and an increase in counter-regulatory hormones that always cause a triad of DKA, namely hyperglycemia, metabolic acidosis, and ketosis (an increase in ketone levels in the blood or urine with serum ketone concentrations > 3 mmol/l) [11]. Insulin deficiency stimulates the formation of counter-regulatory hormones such as glucagon, catecholamine, cortisol, and growth hormone. The counter-regulatory hormone response is also triggered by stress-induced proinflammatory cytokines. They stimulate lipolysis and proteolysis, increase the production of hepatic and renal glucose, and stimulate the oxidation of fatty acids into ketones in the liver. In contrast to physiological fasting, the absence of glucose processing in the citric acid cycle inhibits the processing of ketones for energy. Hyperglycemia in patients will cause loss of fluid volume through urine, extracellular fluid loss, and loss of electrolytes (sodium, potassium, and chloride). DKA are divided into 3 degrees of severity, with mild degrees of venous pH > 72 and < 73 or serum bicarbonate (HCO3) levels < 15 mmol/L. Moderate degrees of DKA venous pH < 72 or serum bicarbonate levels (HCO3) < 10 mmol/L. Severe degree of DKA venous pH < 71 or serum bicarbonate (HCO3) levels < 5 mmol/L [12, 13].

Criteria for the diagnosis of DKA include hyperglycemia (plasma glucose above 200 mg/dL or 11 mmol/L), a venous pH <7.3 or a bicarbonate level <15 mmol/L, and elevated serum or urine ketones. Clinical manifestations of DKA are generally patients with a history of diabetes mellitus with symptoms of polydipsia, polyuria, polyphagia, nocturia, enuresis, and malaise. Other clinical signs and symptoms usually present with signs of dehydration, accompanied by abdominal pain and complaints of nausea and vomiting (which may often be mistaken for gastroenteritis). Patients also have breath signs of an odor of acetone and other ketones. Complaints are accompanied by tachycardia, tachypnea, and often a typical breathing pattern of Kussmaul breathing, which is characterized by rapid and deep breathing as a result of compensation from the body experiencing a state of metabolic acidosis. In more advanced conditions, patients can experience a decrease in consciousness, such as confusion, drowsiness, changes in mental status, and loss of consciousness. In this case, the patient comes up with complaints of nausea and vomiting; he also complains of feeling weak. In this case, the patient has had a history of type 1 diabetes mellitus since elementary school. The patient also said routine control at Prof. Ngoerah Denpasar Hospital

Supportive examinations performed in patients with DKA include blood sugar level checks with sugar levels > 200 mg/dL. A blood gas analysis examination is also important to determine the patient's blood pH and serum bicarbonate levels. In DKA patients, the blood pH will tend to acidosis, which is < 73, or serum bicarbonate levels will be obtained at < 15 mmol/L. A urinalysis examination is carried out to determine the presence of ketonuria in the patient. A complete blood hematology examination is also routinely carried out to find out the presence of an infection that can trigger diabetic ketoacidosis, which is usually characterized by the presence of leukocytosis. However, DKA may also be accompanied by non-specific signs of leukocytosis that do not lead to an infection. In addition, electrolyte examinations are also carried out because DKA can cause disturbances in the body's electrolyte balance. The occurrence of insulin deficiency, hyperglycemia, and acidosis can lead to increased potassium levels. In addition, osmotic diuresis, poor food intake in patients, and vomiting can cause a decrease in potassium levels. 6 There are studies that have found cases of acute kidney failure in pediatric patients with diabetic ketoacidosis, so it is also important to check kidney function in DKA patients. The incidence of acute kidney failure is related to the occurrence of severe dehydration and acidosis. 4,5 In this case, the blood sugar level was obtained at the time of the patient's initial admission to the hospital. DKA patients include checking blood sugar levels with a sugar level > 200 mg/dL. It is also important to check the blood gas analysis to determine the patient's blood pH and serum bicarbonate levels. In DKA patients, the blood pH will tend to acidosis, which is < 73, or serum bicarbonate levels will be obtained at < 15 mmol/L. A urinalysis examination is carried out to determine the presence of ketonuria in the patient. Complete blood hematology examinations are also routinely carried out to find out the presence of an infection that can trigger diabetic ketoacidosis, usually characterized by the presence of leukocytosis. However, DKA may also be accompanied by non-specific signs of leukocytosis that do not lead to an infection. In addition, electrolyte examinations are also carried out because DKA can cause disturbances in the body's electrolyte balance. The occurrence of insulin deficiency, hyperglycemia, and acidosis can lead to increased potassium levels. In addition, osmotic diuresis, poor food intake in patients, and vomiting can cause a decrease in potassium levels. There are studies that have found cases of acute kidney failure in pediatric patients with diabetic ketoacidosis, so it is also important to check kidney function in DKA patients. The incidence of acute kidney failure is related to the occurrence of severe dehydration and acidosis. In this case, the blood sugar level obtained at the time of the patient's initial admission to the hospital was 400 mg/dL.

Complete blood examination results obtained leukocytosis of 15.08 x 103/uL. The blood glucose result of the clinical chemical examination was obtained at 565 mg/dL, which, according to the theory, showed an increase in blood glucose levels > 200 mg/dL. The patient was also found to have slightly elevated urea levels, with creatinine levels still within normal limits. According to the theory, the patient experienced electrolyte balance disorders that indicated hyponatremia, with potassium levels still within normal limits and chloride levels slightly decreasing below normal levels. In this patient's case, the patient's blood pH showed that the patient's blood tended to acidosis with a pH of 7,367 accompanied by a decrease in serum bicarbonate levels, namely HCO3 16.6 mmol/L. The patient also experienced ketonuria, which can be seen from the presence of ketone +3 on the urinalysis examination.

The principles of DKA management include fluid therapy to correct dehydration and stabilize circulatory function, insulin

administration to stop excessive ketone production, treating acidosis and electrolyte balance disorders, overcoming factors that trigger the onset of DKA, and monitoring the complications of therapy. Initial management according to the general handling of emergency patients according to the Pediatric Advanced Life Support (PALS) guidelines includes stabilization of the airway, breathing, and circulation. Fluid correction improves circulation, replaces fluid and electrolyte deficits, and improves renal filtration function to increase plasma glucose and ketone clearance. If shock occurs, 0.9% NaCl or 20 mL/kg BB RL can be given and can be repeated until the shock is resolved. If the shock has improved but circulation has not stabilized, fluids can be given at a rate of 10 mL/kgBB within 1-2 hours. Rehydration fluid administration should begin immediately with isotonic fluids (NaCl 0.9% or near-isotonic fluids such as lactated ringers, RL, or acetic ringers). After initial resuscitation, fluid administration for deficits and housing needs involves selecting fluids that have a tonicity equal to or lower than NaCl 0.9% by adding potassium chloride (KCl), potassium phosphate (KPO4), or potassium acetate. 4,5 In this patient, initial management was carried out according to PALS guidelines by providing oxygenation. Furthermore, the patient was given fluid therapy by giving 0.9% NaCl fluid loading. As fluid therapy, the patient was given NaCl 0.9% (500 cc), which was added with KCl as much as 10 meq.

The therapy in DKA patients that has an important role in controlling blood sugar levels and suppressing the processes of lipolysis and ketogenesis is the administration of insulin therapy. Insulin is given immediately after initial fluid resuscitation, namely after 1-2 hours after administering fluids. The dose of insulin that can be given is 0.05–0.1 U/kg/hour by diluting 50 units of insulin diluted in 50 mL of 0.9% NaCl (1 mL = 1 U) or 5 units of insulin diluted in 50 mL of NaCl (1 mL = 0.01 U). The insulin dose is maintained at 0.05–0.1 U/kg/hour until DKA is resolved (pH >7.30, bicarbonate >15 mEq/L). To prevent blood glucose from dropping too quickly, 5% dextrose can be added to intravenous fluids (5% dextrose added to 0.9% or 0.45% NaCl) if plasma glucose levels drop to 250–300 mg/dL (14–17 mmol/L). If there is a decrease in blood glucose of more than 90 mg/dL/hour (5 mmol/L/hour), the addition of glucose-containing fluids can be considered even if the blood glucose level has not reached <300 mg/dL. In this case, the patient was given an insulin drip for blood sugar correction. The patient was given 5 units of insulin diluted with NaCl 0.9%, as much as 50 cc dripped at a rate of 5 cc per hour. The patient was monitored for blood sugar levels as a consideration for giving dextrose fluid if the patient's blood sugar level decreased rapidly. If blood sugar levels drop between 150 and 250 mg/dL, the patient is given additional D5 ½ NS fluid. If the patient has a decrease in blood sugar levels < 150 mg/dL, the patient is given additional fluid in the form of D10%.

Correction of acidosis in pediatric patients with DKA is usually unnecessary. Bicarbonate administration increases the risk of hypokalemia, secondary hypertonicity, and cerebral edema. Acidosis correction is only performed in severe DKA with a pH <6.9 and life-threatening hyperkalemia. Children with DKA have pseudohyponatremia. Potassium correction is done if there is urine production, which is a sign that kidney function is good [14, 15]. One of the complications that can occur in pediatric patients with DKA is cerebral edema. Cerebral edema is an increase in the amount of fluid in the brain tissue (edema) that causes an increase in brain tissue volume. Edema can be vasogenic due to damage to the blood-brain barrier, cytotoxic edema due to metabolic disorders, or osmotic edema due to hyponatremia.

The main risk factors for DKA recurrence are poor metabolic control or previous episodes of DKA, female gender (peripubertal or adolescent), psychiatric disorders including eating disorders, difficult or unstable family circumstances, limited access to medical services, and use of insulin pump therapy. Only rapid or short-acting insulin can be used in insulin pump therapy, so interruption of insulin delivery can quickly lead to insulin deficiency. Among 1,243 patients in Colorado, the risk of recurrent DKA was eight episodes per 100 patients per year. DKA occurring at a younger age increases the risk of DKA recurrence. Another study mentioned that DKA patients are readmitted more often by males than females [16].

Studies have also shown that poor initial glycemic control and elevated glycated hemoglobin levels (HbA1C > 10.6%) are associated with recurrent DKA in patients. In this case, the patient was known to have had poor glycemic control over the past few months. The patient had an HbA1c level of 14%. In patients with co-morbidities, depression, alcohol, or substance abuse also seem to play an important role, particularly active cocaine use, which is strongly associated with recurrent DKA [17]. In addition, socioeconomic factors (such as ethnic minority status, use of public health insurance, and underinsurance), psychosocial, economic, and behavioral factors (including financial constraints, difficulty in obtaining insulin, and homelessness) have all been reported to be associated with recurrent DKA [18, 19]. Among the known risk factors for DKA, some may be thought to help health workers prevent recurrent DKA. Bradford AL et al. (2017) conducted a study on six potential risk factors for recurrent DKA: age <35 years, history of depression, HbA1C >10.6% at admission, history of substance use or alcohol consumption, ethnic minority status, and self-payment or publicly funded insurance. In this study, there was a significant increase in the odds of recurrent DKA in 4 of the 6 factors, namely age <35 years, history of depression, history of substance or alcohol abuse, and self-pay or publicly funded insurance.

Other studies have suggested that the main cause of recurrent DKA is insulin negligence. Possible reasons for the increase in insulin misuse among the younger generation are eating disorders or weight manipulation (common in adolescent girls), in addition to the desire or need to escape from parents (possibly related to sexual, emotional, or abandonment). Depression, which is 2-3 times more common in people with diabetes, may also play a role. Ultimately, this condition can lead to feelings of resentment and rejection, resulting in a period of rebellion against diabetes [20]. In this case, the patient is an adolescent boy who has been diagnosed with type 1 diabetes mellitus for a long time, namely when the patient was still in elementary school. This may be related to the patient's emotional state and feelings of boredom, which played a role in the occurrence of recurrent DKA in this patient.

Conclusion

Diabetic ketoacidosis is one of the complications with high morbidity and mortality, especially in patients with type 1 di-

abetes mellitus. Diabetic ketoacidosis (DKA) is biochemically defined as venous pH <73 or serum bicarbonate concentration <15 mmol/L, serum glucose concentration >200 mg/dL (11 mmol/L), along with ketonemia, glucosuria, and ketonuria. The principles of DKA management include fluid therapy to correct dehydration and stabilize circulation function, administer insulin, overcome acidosis and electrolyte balance disorders, and overcome factors that trigger the onset of DKA. There are several risk factors associated with the occurrence of recurrent DKA in patients. Known risk factors for DKA allow health workers to prevent the recurrence of DKA.

Research Ethics

Before obtaining patient data through medical records, the authors provided informed consent which was approved by the patient to be reported in this case report.

Conflict of Interest

There is no conflict of interest in this study.

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Authors' Contribution

All authors contributed to the data writing process up to publication.

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