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Case Report

Ischemic-Hemorrhagic Stroke in New Onset Type 1 Diabetes Mellitus with Diabetic Ketoacidosis in a Two-Year-Old Toddler: The First Reported Case in Indonesia

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Abstract: Cerebral edema accounts for 90% of intracerebral complications in diabetic ketoacidosis (DKA). Stroke has been reported to account for 10% of intracerebral complications of DKA in children. It may be underrecognized because its presentation may be subtle. This case report aims to raise awareness of stroke presentation and risk factors in pediatric DKA. A previously healthy two-year-old female presented to the emergency department one day before admission with loss of consciousness and was diagnosed with DKA. The recognition of left hemiparesis was on the 9th day of hospitalization. After 14 days of hospitalization, the patient had recurrent seizures. Computed tomography and angiography revealed subacute ischemic transformative to subacute hemorrhagic stroke. During the follow-up, the symptoms of hemiparesis improved with routine physiotherapy, with some partial palsy of the third cranial nerve remaining. Risk factors for stroke in the patient were the severity of dehydration during DKA, younger age of onset, delayed DKA treatment, and iron deficiency anemia. Suspicion of stroke is necessary even if a subtle neurologic deficit occurs.

Keywords: type 1 diabetes mellitus; diabetic ketoacidosis; pediatric stroke; Indonesia

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1. Introduction

Type 1 diabetes mellitus (T1DM) is one of the most common autoimmune diseases leading to chronic conditions in pediatric patients [1,2]. Its prevalence is expected to increase, affecting 13.5 to 17.4 million people worldwide, including children and adults, by 2040 [1]. The incidence of T1DM diagnosed under four years of age is the lowest in Asia, with a mean incidence of 10.27/100,000 cases/year [3]. In the United States, one-third of T1DM patients are initially diagnosed with diabetic ketoacidosis (DKA) [4]. Meanwhile, in Indonesia, the awareness of T1DM is low; around 71% of T1DM cases were diagnosed at the onset with DKA [5]. Neurological complications of DKA require the presence of severe DKA or chronically poor glycemic control [6]. Those include cerebral edema, which occurs in 6.8 per 1000 DKA cases, and stroke [7]. Stroke, both ischemic and hemorrhagic, is rare, accounting for 10% of intracerebral complications in DKA [6–10]. Recognition may be challenging when symptoms and signs mimic those of cerebral edema [9]. This case presents the first reported case of a toddler with DKA and ischemic hemorrhagic stroke in Indonesia.

2. Case Description

A previously healthy two-year-old Indonesian girl was admitted to another hospital's emergency department (ED) with altered consciousness and decreased appetite one day before the admission. The parents observed that a week before the ED presentation, the patient ate and drank more eagerly than usual and had a weight loss of approximately two kilograms within a month. No significant birth or family history suggested diabetes mellitus or other diseases. Physical examination in the ED revealed a Glasgow coma scale

(GCS) of 8, a temperature of 37 °C, a heart rate of 112/min with signs of poor perfusion, a respiratory rate of 57/min (Kussmaul's breathing), a blood pressure of 141/95 mmHg, a peripheral oxygen saturation of 98%, severe dehydration, and hyperglycemia (bedside blood glucose indicating HI > 600 mg/dL). The laboratory data from the previous hospital was not obtained. The patient was diagnosed with diabetic ketoacidosis (DKA) and shock. She was treated with 20 mL/kg weight of 0.9% saline for one hour and 0.05 IU/kg weight per hour of insulin drip with potassium at the maintenance dose. She was admitted to the pediatric intensive care unit with a femoral central venous catheter (CVC), a nasogastric tube (NGT), and an endotracheal tube insertion. The DKA was resolved within 12 h.

The patient was transferred to another hospital after nine days of hospitalization with no improvement in consciousness (GCS 10) following the regular treatment protocol of DKA. The patient had developed left hemiparesis. Laboratory data revealed iron deficiency anemia (Hb of 8.1 mg/dL, low ferritin level), leukocytosis (leukocyte of 22,000/µL), with an abnormal level of prothrombin time (PT) and activated partial thromboplastin time (aPTT), HbA1C of 13.4%, C-peptide of 0.32 ng/mL, metabolic acidosis with impaired renal and liver function tests, low potassium, and high calcium levels (Table 1). A broad-spectrum antibiotic was administered. The patient was still hyperglycemic after ten days of hospitalization (glucose level around 235–490 mg/dL). On day 14 of hospitalization, she had recurrent tonic-clonic seizures in less than three minutes. A physical examination showed a positive Babinski sign in both legs, no neck stiffness, and an unremarkable Brudzinski sign. Phenytoin via intravenous was administered to treat the seizure. The first electroencephalography (EEG) revealed generalized cortical dysfunction, and no epileptiform wave was detected. She was extubated after 16 days of hospitalization, and her consciousness remained stable in an apathetic state (GCS 12).

Table 1. Laboratory results on day nine of hospitalization.

Laboratory Test	Value	Reference Range
Hematology		
Hb [1]	8.1 g/dL	11.5–14.5 g/dL
Leukocyte	22,500/µL	5000–10,000/µL
Thrombocyte	295,000/µL	150,000–450,000/µL
Coagulation		
pT [1]	9.7 s (L)	10.0–12.7 s
aPTT [1]	21.8 s (L)	23.0–34.7 s
INR	0.9	
D-Dimer	1.93	<0.5 µg/mL
Metabolic		
Blood glucose	482 mg/dL	<200 mg/dL
HbA1c	13.4% (H)	<6.5%
C-peptide	0.32 ng/mL	1.1–4.4 ng/mL
Blood ketone	20 mg/dL (H)	<10 mg/dL
Urine ketone	+1 (H)	Negative
Serum osmolality	321 mOsm/kg	275–295 mOsm/kg
Blood gas analysis		
pH	7.28 (L)	
7.35–7.45	25.8 mEq/L (H)	18.0–23.0 mEq/L
pCO_2	12.0 mm/Hgm (L)	
35.0–48.0 mm/Hgm	197 mmHg (H)	83–108 mmHg
Base excess	−12.9 (L)	−2.0–−3.0 mEq/L
Renal function test		
Urea	109.1 mg/dL (H)	5–18 mg/dL
Creatinine	1.74 mg/dL (H)	0.2–0.5 mg/dL
Liver function test		
AST [1]	442 U/L (H)	0–32 U/L
ALT [1]	390 U/L (H)	0–33 U/L
Electrolyte		
Na [1]	138 mmol/L	136–146 mmol/L
K [1]	2.7 mmol/L (L)	3.5–5.1 mmol/L
Cl [1]	106 mmol/L	98–106 mmol/L
Ca iron [1]	1.53 mmol/L (H)	1.15–1.32 mmol/L

Table 1. *Cont.*

Laboratory Test	Value	Reference Range
Others		
Ferritin	1.24 ng/mL (L)	4.63–204.00 ng/mL
Blood smear	Erythrocyte: normochromic, anisocytosis	Normocytic normochromic
Lipid profile		
LDL [1]	53 mg/dL	<110 mg/dL
HDL [1]	25 mg/dL (L)	>45 mg/dL
Triglyceride	260 mg/dL (H)	<75 mg/dL

[1] Hb: hemoglobin; PT: prothrombin time; aPTT: activated partial thromboplastin time; HbA1c: hemoglobin A1c; AST: aspartate aminotransferase; ALT: alanine aminotransferase; Na: sodium; K: potassium; Cl: chloride; Ca: calcium; LDL: low-density lipoprotein; HDL: high-density lipoprotein.

The patient was transferred to our hospital after 33 days of hospitalization for metabolic control and neurologic assessment. A physical examination in our hospital revealed left hemiparesis with right third cranial nerve (CN) palsy. Laboratory tests showed hyperglycemia (299 mg/dL), thrombocytosis (499,000/µL), abnormal blood coagulation function (pT 10.3 s—reference range 10–12.7 s, aPTT 21.6 s—reference range 21.6 s), and platelet hyperaggregation. Magnetic resonance imaging (MRI) showed sub-acute infarction at the right temporal lobe, cortical hyperintensity at the bilateral frontal-parietal-occipital lobe, and late subacute hemorrhage at the right frontal lobe (Figure 1). Brain computed tomography angiography (CT-A) was performed after 36 days of hospitalization and revealed cortical hyper-density enhanced by contrast-suggestive ischemic transformation to hemorrhagic stroke (Figure 2). During this time, rapid-acting insulin was continued at 0.05 IU/kg/hour per infusion, and 8 mg/kg weight daily of phenytoin was given. The administration of 1 mg/kg of aspirin daily and a routine physiotherapy regimen began. The patient was hyperglycemic during the hospitalization (blood glucose range from 200 to 360 mg/dL); glycemic control was achieved on day 37. On day 38 of hospitalization, oral intake was introduced. Insulin was switched to two international units (IU) of fast-acting insulin per subcutaneous before meals and five IU of long-acting insulin per subcutaneous at night. The second EEG was performed, and severe generalized cortical dysfunction was detected. The patient continued to be treated in the hospital with routine physiotherapy, a controlled diet, and glycemic control. The patient was discharged after 40 days of hospitalization.

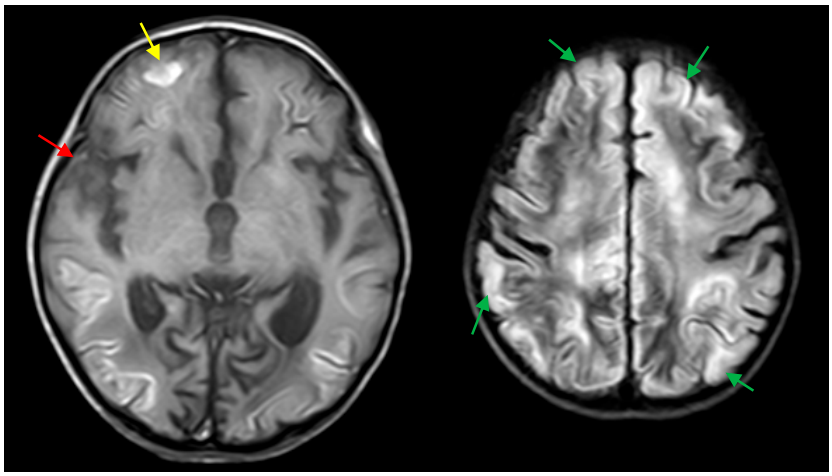

Figure 1. Magnetic resonance imaging (MRI) axial shows: red arrow: subacute infarct on the right temporal lobe; yellow arrow: late subacute hemorrhage on the right frontal lobe; green arrow: cortical hyperintensity on the bilateral frontal-parietal-temporal-occipital lobe.

During follow-up, the patient's condition remained stable three months after hospitalization. She could eat orally and gained about two kilograms in weight. She could respond to sensory stimuli, but her developmental milestones regressed compared to other children her

age. The complaint of left hemiparesis decreased with routine physiotherapy, but her partial palsy of the right third CN remained. Figure 3 shows the timeline of the patient's treatment.

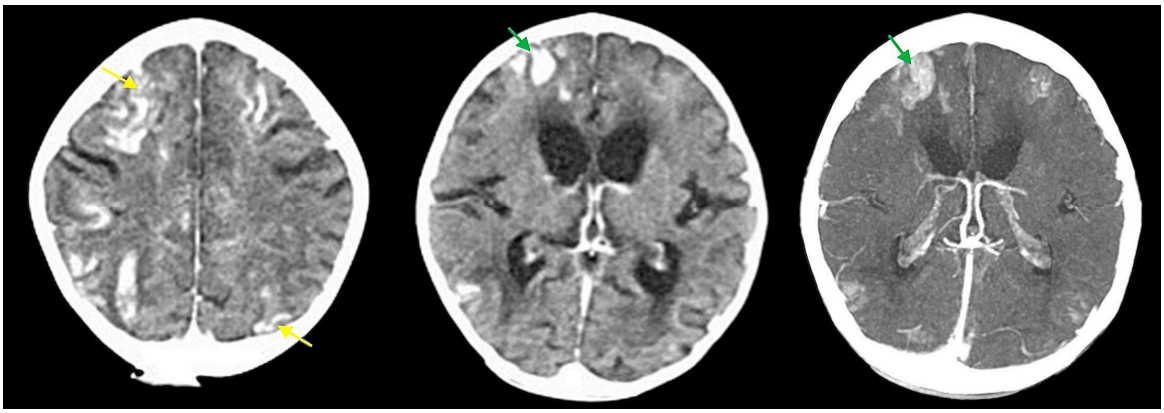

Figure 2. Computed tomography angiography (CT–A) with contrast shows: yellow arrow: hyperdense lesion enhanced with contrast on bilateral frontal-temporal-parietal-occipital lobe suggestive hyper-perfusion post-ischemic region; green arrow: hyperdense lesion suggestive hemorrhagic lesion; no abnormalities of cerebral vasculature were found.

	Treatment
Presentation day	20 ml/kg weight of 0.9% saline, fast acting insulin drip 0.05 IU/kg weight, potassium at maintenance dose. The patient was intubated, central venous catheter (CVC), nasogastric tube (NGT) was inserted.
Day 9 of hospitalization	Fast acting insulin 0.05 IU/kg/hour IV drip, potassium at maintenance dose, broad spectrum antibiotic
Day 14 of hospitalization	Fast acting insulin 0.05 IU/kg/hour IV drip, potassium at maintenance dose, broad spectrum antibiotic, intravenous phenytoin. The patient was extubated after 16 days of hospitalization
Day 33 of hospitalization	Fast acting insulin 0.05 IU/kg/hour IV drip, potassium at maintenance dose, intravenous phenytoin.
Day 36 of hospitalization	Rapid acting insulin 0.05 IU/kg/hour IV drip, potassium at maintenance dose. Per NGT: phenytoin 8mg/kg weight, aspirin 1 mg/kg weight, routine regimen of physiotherapy
Day 38 of hospitalization	Subcutaneous insulin: 5 IU long acting, 2 IU fast acting. Per NGT: phenytoin 8mg/kg weight, aspirin 1 mg/kg weight, physiotherapy, controlled diet
Day 40 of hospitalization	Subcutaneous insulin: 5 IU long acting, 2 IU fast acting. Per NGT: phenytoin 8mg/kg weight, aspirin 1 mg/kg weight, physiotherapy, controlled diet

Figure 3. A treatment summary of the patient during hospitalization. IU: international unit; IV drip: intravenous drip.

3. Discussion

In Indonesia, 1481 pediatric T1DM cases were reported by the Indonesian Pediatric Association in 2023. This number is estimated to be underreported compared to 278,696,000 of the total population in Indonesia [11]. Around 71% of T1DM cases in Indonesia were diagnosed at the onset of DKA; awareness of T1DM is low among health workers [5]. The intracerebral complication is rare, with only 0.3–1% of DKA cases in developed countries with a lower prevalence of DKA [10]. In Indonesia, the prevalence rate has not been reported. Cerebral edema is a more common intracerebral complication that can occur in DKA [9,12,13]. Stroke (ischemic and hemorrhagic) and other cerebral complications should also be suspected [12–15].

Early-onset stroke may be asymptomatic or subtle and occur within the first seven days after admission [16,17]. In this patient, the stroke may have occurred earlier before the ninth day of hospitalization, marked by no improvement in consciousness after the DKA had been resolved and prolonged intubation even with regular treatment of DKA [9,16]. Lack of improvement in consciousness by 24 h should raise suspicion for an intracerebral complication. Left hemiparesis went unnoticed until the ninth day of hospitalization, leading to a delayed diagnosis of stroke. The new-onset seizures on day 14 of hospitalization may be a sign of a new brain sequel, such as the transformation of ischemic to hemorrhagic stroke and the occurrence of hemorrhagic stroke [9,16]. The appearance of the stroke itself can mimic the appearance of cerebral edema. Only a few patients had focal neurological symptoms [9].

DKA patients are prone to an acquired procoagulant process mediated by systemic inflammation and ketosis-induced oxidative stress, characterized by diffuse vascular endothelial damage and coagulopathy [9,18,19]. Decreased coagulation factors (PT and APTT) and elevated thrombocyte count during the hospitalization marked the hypercoagulation process that may mediate the ischemic event [9,14]. Some risk factors that may trigger the stroke are the severity of dehydration during DKA, marked by poor perfusion at the ED and its complications (liver and renal failure), delayed DKA treatment that is associated with the severity of DKA, and younger age at onset [7,9,14,16,20]. Iron deficiency anemia has been associated with childhood stroke by increased bone marrow activation and release of large platelet forms that were seen as thrombocytosis and platelet hyperaggregation in our patient [21].

The hemorrhagic event might be a result of secondary hemorrhagic transformation of cerebral infarcts due to coagulation factor consumption in the procoagulant state or may be induced by the hyperglycemia status since glycemic control was difficult to achieve until the 37th day of hospitalization [7,9,16].

Early imaging is essential to detect stroke onset, particularly in patients who still show no improvement in consciousness once the metabolic treatment starts. Admission to the critical care unit and close monitoring of neurologic status are crucial for a patient with a central nervous system (CNS) complication [9,13,16]. Recanalization therapy (intra-arterial or intra-venous alteplase and balloon angioplasty) may be considered for hyperacute stroke patients, but the complications may outweigh the benefits [9,13,22,23]. Initial treatment using low molecular weight or unfractionated heparin, followed by warfarin therapy, is recommended for high-risk children with post-ischemic stroke [9,24]. The anticoagulant was not given in our case because it had never been used in children. Aspirin as an antiplatelet was given at the lowest dose (1 mg/kg body weight/day) during the 36 days of hospitalization as secondary prevention for the stroke, considering the onset of hemorrhage was in the sub-acute phase [22]. Prevention of DKA is the most effective way to prevent stroke and its sequel [9,24]. Poor outcome in this patient is associated with younger age, DKA severity, late DKA initial treatment, the requirement of intubation, poor metabolic control, delayed stroke recognition, and the presence of hemorrhagic stroke [9,13,14,16,25]. Most DKA-associated strokes have residual neurologic deficits [9,13].

The diagnosis of hyperosmolar hyperglycemic state (HHS) in this patient can be excluded; at the presentation, the patient might be in hyperosmolar DKA due to hyper-

glycemia with a random blood glucose of >600 mg/dL on the first day of hospitalization and Kussmaul respiration [26].

Mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (MELAS syndrome) presentation is similar to our case, with lactic acidosis, recurrent seizure, and stroke lesion with cortical enhancement [27]. However, the patient's brain lesion did not disappear after 33 days of hospitalization, and there was no family history of mitochondrial disease or diabetes.

The limitations of this case report include the unavailability of the laboratory data upon the ED presentation, and the T1DM auto-antibody test is not widely available in Indonesia; thus, the underlying autoimmunity cannot be identified. The diagnosis of T1DM in this case is solely based on HbA1C and C-peptide.

In conclusion, cerebral edema accounts for the majority of intracerebral complications in DKA. In our case, the presentation of no improvement in consciousness marked the stroke appearance; delayed recognition of hemiparesis was our pitfall; and seizures were signs of a stroke that occurred later. The diagnosis of stroke-associated DKA is challenging, with difficulty in overlapping symptoms with cerebral edema. Diagnosing T1DM before the onset of DKA is the best way to prevent intracerebral complications and their sequels. Thus, it is essential to consider stroke in DKA patients with neurologic deficits and raise suspicion for an intracerebral complication in patients with no improvement in consciousness.

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