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Silent Stroke in a 4-Year-Old Boy During Recovery from Diabetic Ketoacidosis: A Rare Complication with Diagnostic Pitfalls

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Case Report

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Abstract

Background: Stroke is an exceedingly rare complication of diabetic ketoacidosis (DKA) in children and may be overlooked due to its atypical presentation.

Case: We report a case of previously healthy 4-year-old boy was admitted with severe DKA (pH 6.9, blood glucose 620 mg/dL). On the 3rd day of ICU admission, post-resolution of acidosis, he developed acute right-sided hemiplegia. Brain MRI demonstrated an acute ischemic stroke in the left middle cerebral artery (MCA) territory, with no evidence of underlying cardiac or thrombophilic abnormalities.

Follow-up: The patient was managed with aspirin, adequate hydration, and glycemic control were initiated immediately. At a 3-month follow-up, residual mild hemiparesis persisted despite rehabilitation.

Conclusion: This case highlights the importance of considering stroke in children with neurologic deficits during or after DKA, even in absence of conventional risk factors.

Key Clinical Message

Ischemic stroke is a rare but serious complication of diabetic ketoacidosis (DKA) in children. Clinicians should consider stroke in pediatric patients presenting with neurologic deficits during DKA recovery, even without traditional risk factors, to enable prompt diagnosis and management.

Introduction

Diabetic ketoacidosis (DKA) is a life-threatening metabolic disturbance characterized by hyperglycemia, metabolic acidosis, and ketonemia, primarily due to insulin deficiency. It is commonly triggered by infections, stress, or new-onset diabetes, and presents with symptoms such as abdominal pain, vomiting, polyuria, and deep (Kussmaul) breathing (3). Despite advances in treatment, DKA still poses significant risks, particularly in vulnerable populations such as young children and pregnant women (1, 3). While cerebral edema is the most common and well-recognized neurological complication of DKA in children, ischemic stroke is a rare but potentially devastating event, occurring in fewer than 0.1% of pediatric DKA cases (1). The exact pathophysiology of stroke in the setting of DKA remains unclear, but several mechanisms have been proposed. These include dehydration-induced hemoconcentration, hyperviscosity due to hyperglycemia, cerebral vasospasm resulting from severe metabolic acidosis, and a prothrombotic state caused by systemic inflammation and endothelial dysfunction (5, 9, 10, 11).

Case History

Healthy 4-year-old boy with no prior medical history or family history of DM type I or hypercoagulability, was admitted with classic features of DKA including polyuria, lethargy, and Kussmaul breathing. Admission vital signs are described in Table 1. Laboratory evaluation confirmed severe DKA, as is depicted in Table 2. Following correction of acidosis and normalizing the serum bicarbonate level,

clinical improvement in the child's general condition was anticipated. However, on the third day of hospitalization, his general condition deteriorated, and his physical examination revealed abnormal finding, prompting investigate further.

Table 1
Vital Signs of the patient at the time of admission

Vital Sign	Result
Heart Rate	115 beats/min
Respiratory Rate	35 breaths/min
Temperature	36.5°C
Blood Pressure	95/60 mmHg
Oxygen Saturation	95%
Signs of Dehydration	Present

Table 2
The results of the patient's blood test in the first hour of hospitalization

Test	Result	Units	Status
W.B.C	17.8	10^3/uL	High
R.B.C	5.48	10^6/uL	Normal
Hemoglobin	17.6	gr/dl	Normal
Hematocrit	43.2	%	Normal
Platelets	416	10^3/uL	High
RDW	11.4	%	Normal
M.C.H	25.72	pg	Low
M.C.V	78.83	fl	Low
M.C.H.C	32.63	gr/dl	Normal
ph	7,14		Low
Bicarbonate	5.2	mmol/L	Low
Anion gap	28	mEq/L	High
Blood glucose	695	mg/dL	High

Table 1. Vital Signs of the patient at the time of admission

Table 2. The results of the patient's blood test in the first hour of hospitalization

Differential Diagnosis, Investigations and Treatment

The patient was treated according to the ISPAD protocol, including intravenous fluids and insulin drip. Acidosis resolved within 36 hours. Upon development of new neurological symptoms, treatment adherence was reviewed. There were no clinical signs of cerebral edema or herniation. The fluid and insulin administration were appropriate in both volume and rate.

On day 3, new-onset right hemiplegia was observed (NIHSS score: 8). The patient was lethargic but responsive to painful stimuli. Vital signs included dehydration, tachycardia (115 bpm), tachypnea (35 breaths/min), normal blood pressure (95/60 mmHg), and oxygen saturation of 95% on room air.

After a neurology consultation, the patient first underwent a Doppler ultrasound of the neck vessels and echocardiography. The next step was a CT scan due to suspicion of cerebral hemorrhage, and finally, a stroke was diagnosed by performing an MRI and MRV of the brain.

Non-contrast CT Brain, on day 3, revealed hypodensity in the left middle cerebral artery (MCA) territory, suggestive of acute ischemia. (Fig. 1)

Figure 1. Axial section of plain cerebral CT scan shows hypodense lesions (28 × 13 mm) in the right hemisphere.

Brain MRI showed acute infarction in the left MCA territory and MRA reported no vasculitis or arterial dissection. (Fig. 2)

Figure 2. Diffusion-weighted MRI (DWI) showing left MCA infarct. Restricted diffusion in right middle cerebral artery territory. MR Angiography, No vascular occlusion or dissection.

Laboratory findings included HbA1c: 12.7% (new-onset type 1 diabetes), hypercoagulability panel normal (including protein C/S, antiphospholipid antibodies, and anti-factor X antibodies) and laboratory Findings were leukocytosis (WBC 17.8 × 10^3/μL), hemoconcentration (Hb 17.6 g/dL), thrombocytosis (416 × 10^3/μL) and microcytic anemia (MCV 78.83 fL). Echocardiography reported no thrombosis or patent foramen ovale (PFO).

The patient was started on low molecular weight heparin (enoxaparin) and low-dose aspirin (3 mg/kg/day) for stroke prophylaxis, insulin for intensive glycemic control (target glucose 100–180 mg/dL), and mannitol (0.5 g/kg) for suspected elevated intracranial pressure. Early physiotherapy and rehabilitation were also initiated.

Outcome and Follow-up

Outcome and Follow-up

The patient was managed with aspirin, adequate hydration, and glycemic control initiated immediately. At a 3-month follow-up, residual mild hemiparesis persisted despite rehabilitation. This case highlights the importance of considering stroke in children with neurologic deficits during or after DKA, even in the absence of conventional risk factors.

Discussion

DKA is a life-threatening metabolic disturbance characterized by hyperglycemia, metabolic acidosis, and ketonemia, primarily due to insulin deficiency. It is commonly triggered by infections, stress, or new-onset diabetes, and presents with symptoms such as abdominal pain, vomiting, polyuria, and deep (Kussmaul) breathing (3). Despite advances in treatment, DKA still poses significant risks, particularly in vulnerable populations such as young children and pregnant women (1, 3).

While cerebral edema is the most common and well-recognized neurological complication of DKA in children, ischemic stroke is a rare but potentially devastating event, occurring in fewer than 0.1% of pediatric DKA cases (1). The exact pathophysiology of stroke in the setting of DKA remains unclear, but several mechanisms have been proposed. These include dehydration-induced hemoconcentration, hyperviscosity due to hyperglycemia, cerebral vasospasm resulting from severe metabolic acidosis, and a prothrombotic state caused by systemic inflammation and endothelial dysfunction (5, 9, 10, 11).

DKA triggers a robust inflammatory response marked by elevated levels of C-reactive protein (CRP) and pro-inflammatory cytokines such as IL-6, IL-1β, and TNF-α. These inflammatory mediators can cause vascular endothelial injury and activate the coagulation cascade, leading to a hypercoagulable state (11). This state is further aggravated by abnormalities in platelet function, coagulation factors, and alterations in blood viscosity and volume. Impaired cerebral autoregulation and systemic hypoperfusion may further increase the risk of both ischemic and hemorrhagic events (11).

Previous research indicates that the keto acid β-hydroxybutyrate (β-OHB) is transferred across the placenta into fetal circulation, leading to its accumulation in the developing brain and subsequent severe metabolic derangements. Similarly, during episodes of DKA in older children, β-OHB rapidly concentrates in the basal ganglia, a process associated with neuronal injury. (12)

Our patient, a previously healthy 4-year-old boy, was diagnosed with type 1 diabetes during this admission. He developed an ischemic stroke in the left middle cerebral artery territory on the third day of hospitalization, after resolution of acidosis. The absence of conventional stroke risk factors, such as congenital heart disease, coagulopathy, or arteriopathy, highlights the role of DKA itself as a trigger for cerebrovascular injury. Notably, initial neurological examination such as muscle strength and tone, and deep tendon reflexes did not reveal focal deficits, and the patient's mental status improved as acidosis resolved—making stroke a challenging diagnosis at first.

Management of CNS complications of DKA should prioritize treatment of cerebral edema, which is more likely in severe acidosis, serum bicarbonate level < 5 meq/L, rapid insulin administration, high volume of

fluid intake, and children < 5 years of age, and should be suspected in the presence of Cushing's triad. (13) Because the initial manifestations of stroke in children can be subtle and may be confused with DKA cerebral edema, initial imaging is essential for any young person with neurological deterioration associated with an episode of DKA after emergency treatment for cerebral edema and stabilization of vital signs. Post-DKA strokes often occur after resolution of acidosis, delaying diagnosis. (14)

This case underscores the diagnostic superiority of MRI over CT for the detection of acute cerebral ischemia, as CT findings can be notoriously unremarkable in the initial phases. Management of confirmed stroke includes antiplatelet therapy (aspirin) and a comprehensive rehabilitation strategy to manage subsequent neurological deficits, including spastic paralysis.

Neurological deficits may mimic cerebral edema (a more common complication of DKA), and every endocrinologist must manage their patients with a holistic view and not ignore very rare complications.

Abbreviations

DKA – Diabetic ketoacidosis, **ICU** – Intensive Care Unit, **MCA** – Middle cerebral artery, **MRI** – Magnetic Resonance Imaging, **CT** – Computed Tomography, **NIHSS** – National Institutes of Health Stroke Scale, **MRV** – Magnetic Resonance Venography, **MRA** – Magnetic Resonance Angiography, **DWI** – Diffusion-Weighted Imaging, **HbA1c** – Hemoglobin A1c, **WBC** – White Blood Cell (count), **Hb** – Hemoglobin, **MCV** – Mean Corpuscular Volume, **PFO** – Patent foramen ovale, **ISPAD** – International Society for Pediatric and Adolescent Diabetes, **CRP** – C-reactive protein, **IL-6** – Interleukin-6, **IL-1β** – Interleukin-1 beta, **TNF-α** – Tumor Necrosis Factor-alpha, **β-OHB** – β-hydroxybutyrate, **CNS** – Central Nervous System

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from the patient’s parents for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Consent for publication

Written informed consent for publication of clinical details and clinical images was obtained from the parents of the patient.

Availability of data and materials

All data are available from the corresponding author upon reasonable request.

Competing Interests

None

Funding

None

Authors' contributions

M.D. drafted the manuscript, E.A. and F.J. collected the data, D.D. and A.H. searched and conducted the manuscript revision, A.F. supervised and structured the study

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Figures

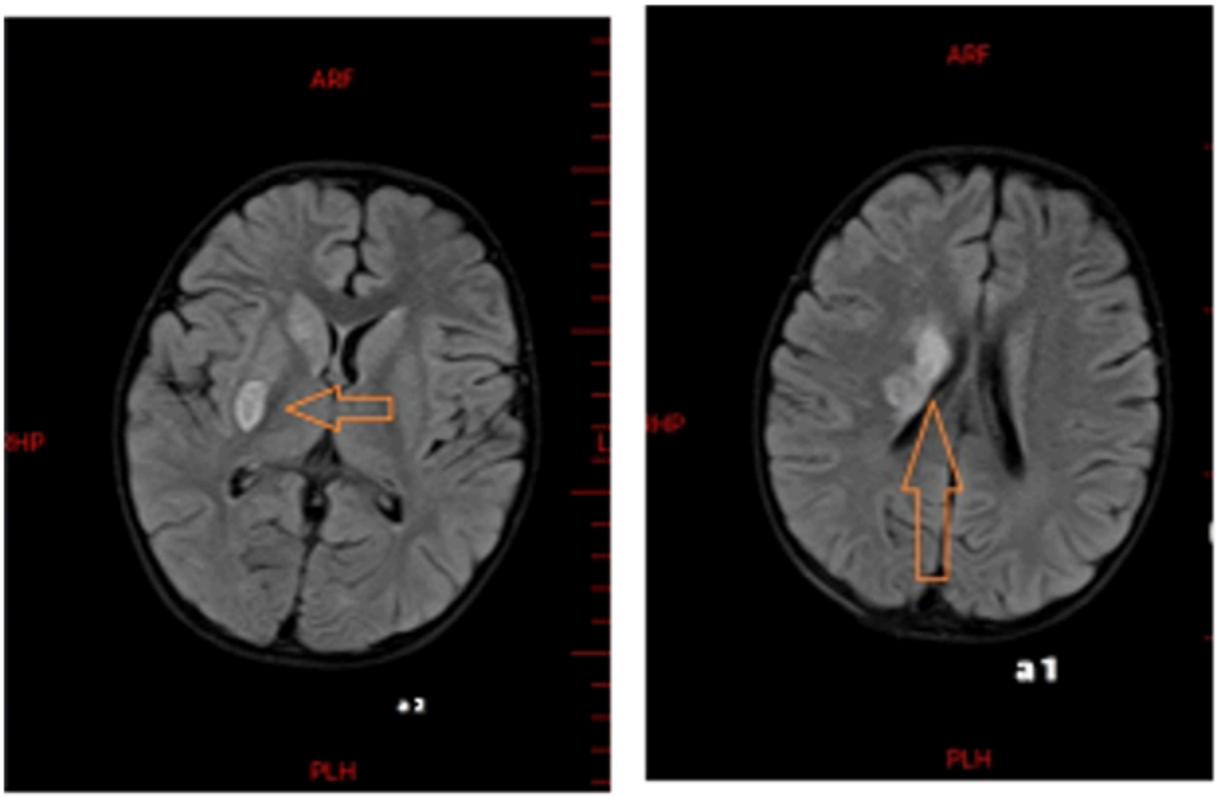


Figure 1

Axial section of plain cerebral CT scan shows hypodense lesions (28 × 13 mm) in the right hemisphere.

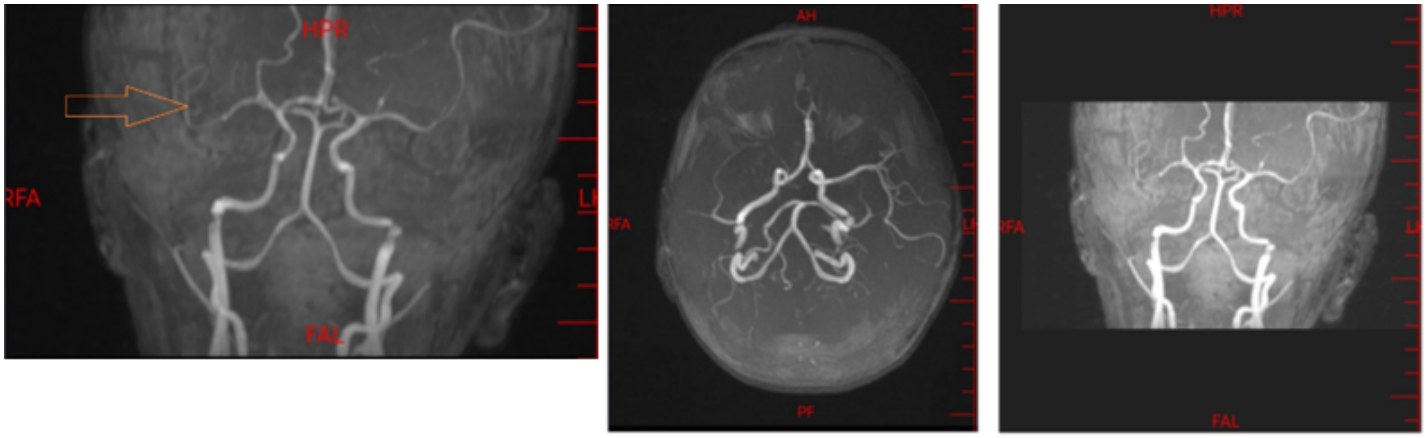


Figure 2

Diffusion-weighted MRI (DWI) showing left MCA infarct. Restricted diffusion in right middle cerebral artery territory. MR Angiography, No vascular occlusion or dissection.

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