Pediatric stroke in a new-onset type 1 diabetes mellitus with and without DKA: Case series

Amal Y. Kentab[1,2*]

[1] Department of Pediatrics, College of Medicine, King Saud University, Riyadh, Saudi Arabia
[2] Division of Pediatric Neurology, Department of Pediatrics, King Saud University Medical City, King Saud University, Riyadh, Saudi Arabia

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BACKGROUND

Neurological deterioration in children with a new-onset diabetes mellitus is caused by cerebral edema. It is associated with diabetic ketoacidosis (DKA) or cerebrovascular accident secondary to a hypercoagulable state and thrombosis produced by prolonged exposure to uncontrolled hyperglycemia. A focal neurological deficit in a newly diagnosed type 1 diabetes mellitus (T1DM) should raise the suspicion of sino-venous thrombosis or ischemic stroke. Aim: To determine the causes, and clinical characteristics of strokes in children presenting with T1DM

METHODS

A retrospective chart review for all newly diagnosed children with T1DM who presented acutely with strokes, and were admitted to King Khalid University Hospital, Riyadh, Saudi Arabia from 2015 to 2020. Patients had been screened for coagulation and lipid profiles, homocysteine level, specific immunologic studies, vasculitis workup, an extended prothrombotic and metabolic screen. ECG/ ECHO, brain MRI, MRV, and MRA were also done.

RESULTS

Three cases of stroke associated with new-onset T1DM were identified. A 24-month-old girl presented with Sino-venous thrombosis related to infection; partially treated meningitis with subdural empyema, while the second one was a 9-year-old girl who presented with cortical vein thrombosis and epilepsy. The last one, a 7-year-old girl, was treated for DKA and had a late diagnosis of ischemic stroke and epilepsy. (Table-1)

CORRESPONDENCE
AMAL Y. KENTAB. MD
Associate Professor of Pediatrics &
Consultant Pediatric Neurologist.

Amkentab@hotmail.com, akentab@ksu.edu.sa

T1DM, DKA, SINO-VENOUS THROMBOSIS, ISCHEMIC STROKE, MRI, CHILDREN.

Table 1: Demographic, and clinical features in 3 pediatric cases of a new – onset diabetes mellitus, and stroke.

	Patient I	Patient II	Patient III
Sex	Female	Female	Female
Age at onset	2 year	7 year	9 year
Ethnicity	Saudi	Saudi	Saudi
Preceding events	Tonsillitis; Fever, ear ache; Rx penicillin for 7 days	Abdominal pain, polyuria, and polydipsia for 2 weeks. Received antibiotic for vulval infection	Polyuria and polydipsia for 2 weeks Epigastric abdominal X 24 hrs,
Clinical features	Noted 2 weeks later Fever, vomiting, headache/ ear ache, abdominal pain, and clouding of consciousness (apathy, sleepiness alternating with periods of restlessness, agitation). Feeding difficulty (drooling, and choking). Motor regression ie; lost ability to walk and sit. Slurred speech left-sided central facial palsy Left-sided hemiparesis	Noted 2 weeks later Headache (fronto-temporal), Focal type (Right-sided) unprovoked seizures Carbamazepine was started Fluctuating hyperglycemia resulted in increased frequency (atonic, focal, and 2nd generalized type), shifted to Valproic acid Mild slurred speech Right-sided hemiparesis (3 wks post event)	Noted 24 hrs later persistent right frontal headache X 6 hrs, numbness, tingling, and mild left-sided weakness X 3 hrs followed by prolonged left-sided focal seizures Transient left–sided Todd`s paresis
First presentation of diabetes Hyperglycemia Episode	+	+	+
Hyperglycemia Episode	+ Glucose 16.8mmol/L	+ Glucose 14mmol/L	+ Glucose 17.5mmol/L

Table 2: Laboratory profiles, management, follow-up period, outcome, and the proposed etiology in 3 pediatric cases of a new – onset diabetes mellitus, and stroke.

	Patient I	Patient II	Patient III
Laboratory profiles Hemogram	Leukocytosis (19x 10^3); microcytic hypochromic anemia; thrombocytosis	Normal WBC (5.2x 10^3); normochromic normocytic anemia HB 10.3 g/dl; normal platelet	Normal WBC (7x 10^3); HB 12.5 g/dl; normal platelet
ESR / CRP	62 / 200	11/not detected	26/ not detected
Serum electrolytes	Na 134, CL 118, K 4.8	Na 139, CL101, K4.3	Na 137, CL104, K 4
Metabolic acidosis	PH:7.38, HCO3 14.5	PH: 7.34, HCO3: 19	PH 7.24, HCO3 11
Urinary Ketones	2+	-	4 +
Urinary glucose	PH:7, Glucose 4+	PH:7, Glucose 4+	PH:7, Glucose 4+
HBA1C	7.8% (NR3.9-6.7)	12.6% (NR3.9-6.7)	13.1% (NR3.9-6.7)
EEG	Slow background more on the right hemisphere, no epileptiform activities	N (awake, and sleep)	N (awake, and sleep)
Management	Hydration, Surgical drainage, IV antibiotics (3 weeks) Insulin, Heparin (UFH X 2 weeks), Aspirin	Hydration, Insulin, Carbamazepine changed to Valproic acid, Aspirin	Hydration, Insulin, IV antibiotics (10 days), Heparin (UFH X 2 weeks), Lamotrigine, Aspirin
Neurologic outcome	Motor improvement / able to walk Incoherent speech Mild left-sided facial palsy, and hemiparesis.	Regained epilepsy control. Good academic achievement.	Epilepsy controlled Medication weaned off Excellent academic achievement.
Follow-up period	2 years	3 years	4 years
Proposed etiology	Hyperglycemia, DKA provoked by Infection (partially treated meningitis with subdural empyema) Cerebral sinus thrombosis	Fluctuating hyperglycemia	Hyperglycemia, DKA Para nasal sinus infection / focal meningitis Cortical vein thrombosis

Patient I

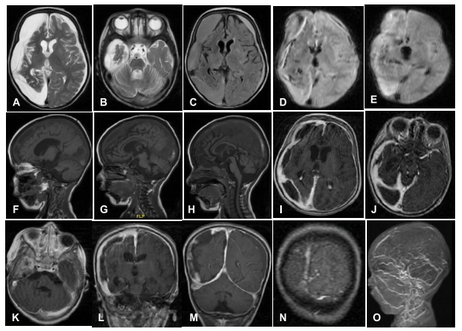

Figure 2: Patient I. Subdural empyema, secondary ischemic stroke, and multiple sinuses thrombosis in new diabetic child: **Brain MRI;** Axial T2 WI (A,B), axial FLAIR (C), and axial diffusion sequence (D& E) demonstrating extra-axial high signal intensity in A & B & C representing subdural empyema, and localized cortical right fronto- parieto-occipital representing ischemic changes. Right cerebral white matter abnormal high signal intensity with diffusion restriction (in D & E) suggesting periventricular ischemic changes. Sagittal T1 WI (F, G & H), and Post – Contrast Axial T1WI (I, J & K) and Coronal T1WI (L & M) demonstrated contrast enhanced extra-axial fluid collection with intense enhanced meninges, and large expanding sagittal sinus, sigmoid, and lateral sinuses with high signal intensity on T1WI (F, G & H), and filling defect on post-contrast sequence (I, J & K), and lack of flow on **MRV** (N&O) indicating multiple sinus thrombosis.

Patient II

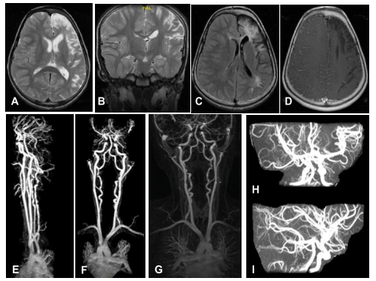

Figure 3: Patient II. Ischemic stroke in a new diabetic child. **Brain MRI;** Axial T2 (A), Coronal (B) and Axial FLAIR (C) demonstrated high signal intensity involving the left pericentral gyrus region , cortics of fronto-parital , and occipital lobes with dilation of left ventricle and atrophy of the left hemisphere suggesting ischemic infarction. Post – Contrast Axial T1WI (D) demonstrated previous findings noted on previous images with no contrast enhancement. **MRA PJN** view Lateral image (E),Anterior (F) and Coronal (G) with contrast showing normal vessels with clear kinking. Anterior (H) and lateral (I) close proximity image demonstrated normal circle of willis vessels with kinking.

Patient - III

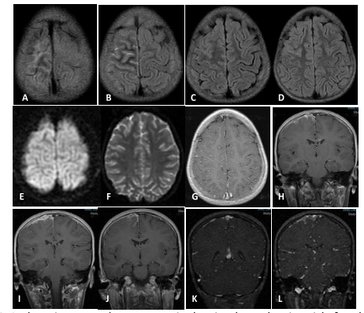

Figure 4: Patient III. Ischemia secondary to cortical vein thrombosis with focal meningitis in a new diabetic child. **Brain MRI;** Axial FLAIR (A,B,C & D) sequence showed partial effacement of the cortical sulci overlying the right frontal lobe at high convexity level with high signal intensity, and Axial diffusion sequence showed m diffusion restriction on the same region. Post – Contrast Axial (E) , and Coronal (F,G &H) T1WI sequence demonstrated focal thickening and abnormal meningeal enhancement at the right superior frontal cortical region with blurred, and partially compromised underlying cortical vein suggestive of localized ischemia, and focal meningitis with superficial cortical vein thrombosis. **MRV** demonstrated normal patent dural sinuses with tiny enhancing nodule at the body of corpus callosum of uncertain nature.

CONCLUSION

Pediatric stroke in a new-onset T1DM is rare. DKA, especially in the infantile type DM, poor glycaemic control, intracranial infection, and dehydration can increase the risk of stroke. Promote diagnosis and medical or mechanical interventions for stroke are associated with reduction of morbidity and mortality.