Case Report

New-Onset Idiopathic Generalised Seizure in a Nigerian Teenager with Type 1 Diabetes Mellitus

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Abstract

We described a case of a 19-year-old female with a new onset Idiopathic Generalised seizure and Type 1 Diabetes mellitus(TIDM). Diagnosis of epilepsy is based on at least two unprovoked seizures. Anti-glutamic acid decarboxylase (Anti-GAD) antibodies positivity is a potential link between TIDM and epilepsy as seen in this case similar to other report. There is a need for careful glycaemic control in children with epilepsy and TIDM as hypoglycemia-induced seizure must be avoided using appropriate insulin dosage.

Keywords: Diabetes mellitus, epilepsy, hypoglycemia, insulin

Introduction

Hypoglycemia-induced seizure is common among individuals with Type 1 diabetes mellitus (TIDM) who are insulin-dependent and type 2 DM patients on certain oral hypoglycemic agents. Seizure may also occur in TIDM with diabetes ketoacidosis (DKA) receiving insufficient insulin administration. Furthermore, there is a recognised association between TIDM and idiopathic generalised epilepsy.[1] However, more needs to be known about this association among African TIDM patients hence this case report.

Case Report

A 19-year-old female diagnosed at aged 11 years to have DKA presented with severe abdominal pain, vomiting, tachypnea and lethargy, and acanthosis nigrican with elevated random blood glucose (RBG) (520 mg%), glucosuria and ketonuria. She was managed as TIDM (C-Peptide 33.0 pmol/L (Range 364–1655pmol/L). Her hemoglobin genotype was "AC" with Rhesus positive blood Group B.

Over the years, she was doing well on her insulin regimen (ultrashort aspart and long-acting glargine) and other nonpharmacological modalities until she was brought to the emergency room in the early hours of the morning and was said to have had generalised tonic–clonic seizure, foamy mouth, eyes staring lasting <10 min with postictal generalised body weakness while at home. No tongue biting, no urinary, or fecal incontinence was noted. RBG immediately postictus done by her parents was 135 mg%.

No blurring of vision or history of galactorrhea. She had mild headache which was relieved with the use of acetaminophen and self-induced insomnia a day before the onset of seizure. No recent febrile illness, head injury, use of alcohol, or psychoactive substances. She had good developmental milestone with satisfactory APGAR score at birth. There was no maternal febrile illness (negative for TORCH screening) and family history of seizure disorder.

Her general physical and neurological examinations were essentially normal. An assessment of new-onset idiopathic generalised seizure disorder in a Type 1 DM was made. Her electroencephalogram (EEG) was normal while brain magnet resonance imaging revealed no structural abnormality. Serum calcium, liver function test, lipids, electrolyte urea and creatinine, urinalysis, and glycosylated

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hemoglobin (HbA1c) were all normal while malaria parasite screening and psychoactive drugs screening were negative. Anti-glutamic acid decarboxylase (anti-GAD) antibodies were absent.

Discussion

In general, a diagnosis of epilepsy is based on at least two unprovoked seizures – Not resulting from an external cause, such as injury or consumption of prescribed medications or other drugs – in a person with normal blood glucose levels (above 3.9 mmol/L) and with an interval >24 h between the seizures.[1] Seizure without known aetiology or precipitant has been identified in TIDM patients. In a UK study, a group of adult with epilepsy were found to have a four-fold higher prevalence of TIDM compared to the general population.[2]

Onset of seizure in this case was eight years postdiagnosis of TIDM which is comparable to an Italian centre report of diagnosis of diabetes on average of 2.8 years before epilepsy.[3] A large data involving 45,847 TIDM patients aged between 0.1 and 20 years from Germany and Austria as part of the Diabetics Prospective Follow-up (DPV) initiative showed a significantly higher frequency of epileptic seizures in children and adolescents with diabetes than expected: Twice as high as in children without diabetes, with no gender difference in the prevalence.[4] Of 705 patients with epilepsy, 375 patients were treated with antiepileptic medication and 330 patients were without anticonvulsive therapy. No similar data in the African setting and this index case did not receive any anticonvulsive therapy and has been seizure-free on follow-up.

Children with diabetes and epilepsy are usually younger at the onset of diabetes than children with diabetes only. This is similar to this case report. The reason for the increased frequency of epilepsy in TIDM is unknown. Schober and Holl[1] observed a significantly increased double-fold risk for DKA in children and adolescents with TIDM and epilepsy than those with TIDM alone. It could be that people with repeated episodes of DKA are more prone to epilepsy. The index case only had one occurrence of DKA at diagnosis, and no record of such again even with the onset of seizure.

As shown in Table 1, Anti-GAD antibodies positivity is a potential link between TIDM and epilepsy as seen in this case similar to other report. Both severe hypoglycaemia and DKA can lead to abnormalities in an EEG. Despite the need to avert hypoglycemia among children with both diseases, HbA1c levels and insulin dosage-to-body weight ratios were similar to those in the children without epilepsy and no difference was found in the type of treatment – pump or injections.[1] However, this is not the case in our patient. There was no need to adjust the insulin regimen in the index case postonset of seizure. However, further health education was given to the patient and her parents to avoid hypo-and-hyperglycaemia.

Table 1: Laboratory findings in the patient with type 1 diabetes mellitus and seizure

	Results	Normal values
Sodium	132	130–150 mmol/L
Potassium	4.9	3.0–5.0 mmol/L
Chloride	102	95–110 mmol/L
Bicarbonate	26	20–30 mmo/L
Urea	2.3	2.5–8.0 mmol/L
Creatinine	58	53–106 µmol/L
Lipids panel	Normal	
Albumin	44.7	35–55 g/L
Globulin	34.6	g/L
Serum calcium	2.38 mmo/L	2.00–2.65 mmol/L
T4F	11.6	10.15–21.1 pmol/L
T3F	4.0	3.1–6.8 pmol/L
Thyroid stimulating hormone	2.61	0.27–4.20 mIU/mL
Haemoglobin A1c	5%	4%–6.5%
Anti-GAD antibody	<5.0 IU/mL	<10.0
Anti-IA2 antibody	<10.0 IU/mL	<10.0
Malaria parasite film	None seen	
Beta-HCG, qualitative	Negative	
Drug screening	Negative	
Urinalysis	Normal	
Brain studies		
EEG	Normal	
Magnetic resonant imaging	Normal	

EEG: Electroencephalogram, Anti-GAD: Anti-glutamic acid decarboxylase, Beta-HCG: Beta-human chorionic gonadotropin, Anti-IA2: Anti - islet antigen 2, T4F: Thyroxine free, T3F: Triiodothyronine free

Conclusion

Metabolic disturbances such as hypoglycemia may not be the precipitant of seizure in TIDM patients. There is a need for careful glycaemic control in children with epilepsy and TIDM as hypoglycemia-induced seizure must be avoided using appropriate insulin dosage. The need to identify the symptoms of metabolic disturbances, allowing earlier diagnosis of DKA cannot be overemphasised. Hence, health-care provider must work with the full cooperation of the patient and patient's caregivers at home for optimum health outcomes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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