OCCIPITAL LOBE SEIZURES – A RARE HYPERGLYCEMIC SEQUELAE OF TYPE 1 DIABETES MELLITUS

Muhammed Jasim Abdul Jalal1*, Ramesh Gomez2, Murali Krishna Menon3, Arunkumar K4

1MBBS, DNB (Family Medicine), Junior Resident, Lakeshore Hospital and Research Centre, Ernakulam, Kerala, India.
2MD, DM (Endocrinology), Endocrinologist, Lakeshore Hospital and Research Centre, Ernakulam, Kerala, India.
3MD, DM (Neurology), Senior Consultant Neurologist, Lakeshore Hospital and Research Centre, Ernakulam, Kerala, India.
4MD (Psychiatry), DNB (Psychiatry), Psychiatrist, Lakeshore Hospital and Research Centre, Ernakulam, Kerala, India.

ABSTRACT
A 15 year old boy presented with osmotic symptoms and photopsia. He had short term memory impairment, visual hallucinations and headache. His RBS was 474mg/dl, HbA1c - 9.4% and GAD-65 > 2000IU/ml. MRI-Brain and CSF study were normal. Digital EEG was suggestive of bilateral hemispheric occipital lobe seizures. He responded well to insulin and anti-epileptic medications.

Keywords: Type 1 Diabetes mellitus, Occipital lobe Seizures, Osmotic symptoms.

Introduction
Hyperglycemia has a wide variety of neurological manifestations [1] including visual hallucinations [1], seizures [1], choreoathetosis [2], hemiballismus [3], dysphagia [3], somatosensory symptoms or headache [4] associated with nausea and vomiting, and severe cases as coma [2,3].

While seizures associated with hypoglycemia are mainly generalized, seizures associated with hyperglycemia are usually focal in nature[3,4]. Previous reports have described occipital seizures associated with hyperglycemia, although the descriptions are rare and few of them have showed electroencephalographic (EEG) findings.

We report the clinical presentation of a 15 year old child with hyperglycemia who presented with focal occipital seizures which was confirmed by EEG.

Case report
A 15 year old boy with two weeks duration of osmotic symptoms (polyuria, polydipsia and weight loss) was diagnosed to have Diabetic ketoacidosis (DKA) and was put on insulin infusion. His random blood sugar was 474 mg/dl and urine ketones were positive. Arterial blood gas analysis showed metabolic acidosis. He experienced flashes of lights (photopsia), well-formed visual hallucinations, fearfulness and headache. There were no tonic clonic movements.

On examination, he appeared confused and anxious. He was hyper alert with ideas of persecution. He had recent and immediate memory impairment without any focal neurological deficits.

His HbA1c was 9.4% and Glutamic acid decarboxylase – 65 (GAD-65) IgG Serum (EIA) was > 2000IU/ml (normal value being <10 IU/ml). MRI Brain and CSF study was normal. His EEG (Figure: 1) showed spike and waves arising from the right occipital region suggestive of occipital lobe seizures.

He improved with hydration and insulin. His occipital lobe seizures recovered very well with clobazam therapy.

Corresponding Author: - Muhammed Jasim Abdul Jalal Email: jasimabduljalal@yahoo.com
DISCUSSION

Seizure types described in patients with hyperglycemia are diverse [1]. This includes epilepsy partialis continua, which is the most frequent type and simple or complex partial seizures. The less common recognized types are apnea, somatosensory symptoms, aphasia, and visual disturbance [2]. Of the two distinct patterns of hyperglycemia (ketotic and non-ketotic), the non-ketotic hyperglycemia is more commonly associated with seizures [3]. Potential explanation is that ketoacidosis decreases neuronal excitability by increasing levels of GABA via activation of glutamic acid decarboxylase, increased cellular concentration of glutamic acid and decreased GABA shunt [4].

Other than changes in GABA, K_ATP channels have been recently shown to be important in hyperglycemia – induced seizures. K_ATP channels are well known for their action in pancreatic cells, where an increase in intracellular ATP/ADP ratio leads to closure of the channels preventing potassium efflux leading to cell membrane depolarization and insulin secretion [5]. Recent evidence indicates that neurons (e.g. hippocampal and neocortical) also have K_ATP channels and these channels are responsible for increasing neuronal excitability in hyperglycemic environment [5].

Our patient had a variety of transient neurological symptoms including occipital seizures in the form of headache, well-formed visual hallucinations and memory impairment. All these symptoms are consistent with a global brain dysfunction related with hyperglycemia [4]. Well-formed visual hallucinations in this patient could be an equivalent of Todd’s phenomena following occipital seizures [4]. Previous reports are consistent with the semiology of our patient. Wang et al., [6] discussed a patient with flickering objects in the right visual field associated with complex visual hallucinations. Raghavendra et al., [7] and Lavin [8] reported the presence of visual hallucinations in non ketotic hyperglycemic hyperosmolar state.

CONCLUSION

Occipital seizures are a unique presentation of hyperglycemia with EEG showing sequential spikes, slowing and attenuation [9]. The focal character of the seizures described during hyperglycemia has been reported consistently over the years [9]. Focal seizures are common in non-ketotic hyperglycemia. They have an excellent prognosis when glucose levels get controlled [9].

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CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Figure 1. EEG suggestive of occipital lobe seizures

REFERENCES
1. Maccario M. Neurological dysfunction associated with non ketotic hyperglycemia. Arch Neurol, 19, 1968, 525–34.
2. Chung S J, Lee J H, Lee S A, No Y J, Im J H, Lee M C. Co-occurrence of seizure and chorea in a patient with non
ketotic hyperglycemia. *Eur Neurol*, 54, 2005, 230–2.
3. Stahlman G C, Auerbach P S, Strickland W G. Neurologic manifestations of non ketotic hyperglycemia. *J Tenn Med Assoc*, 81, 1988, 77–80.
4. Guisado R, Arief A I. Neurologic manifestations of diabetic comas, correlation with biochemical alterations in the brain. *Metabolism*, 24, 1975, 665–79.
5. Miki T, Seino S. Roles of KATP channels as metabolic sensors in acute metabolic changes. *J Mol Cell Cardiol*, 38, 2005, 917–25.
6. Wang C P, Hsieh P F, Chen C C, Lin W Y, Hu W H, Yang D Y, et al. Hyperglycemia with occipital seizures, images and visual evoked potentials. *Epilepsia*, 46, 2005, 1140–4.
7. Raghavendra S, Ashalatha R, Thomas S V, Kesavadas C. Focal neuronal loss, reversible subcortical focal T2 hypo intensity in seizures with a non ketotic hyperglycemic hyperosmolar state. *Neuroradiology*, 49, 2007, 299–305.
8. Lavin P J. Hyperglycemic hemianopia, a reversible complication of non-ketotic hyperglycemia. *Neurology*, 65, 2005, 616–9.
9. Harden C L, Rosenbaum D H, Daras M. Hyperglycemia presenting with occipital seizures. *Epilepsia*, 32, 1991, 215–20.