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## Case report on hypoglycemia induced seizures

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Article History	Abstract
Received on: 10-10-2020 Revised On : 18-01-2021 Accepted on : 26-01-2021	This case report is mainly about neurological symptoms in relationship to hypoglycemia. Some of the symptoms of neuroglycopenic are cognitive impairment, behavioral changes, psychomotor abnormalities, weakness, seizures, and coma. Normally coma is the dominating neurological symptom in hypoglycemia and seizures are rare manifestations. So it may be overlooked when the patient admitted due to seizures by the hypoglycemia. Seizures may be associated due to hyperglycemia or hypoglycemia because abnormal blood glucose is seen in daily clinical practice. <i>Keywords:</i> Hypoglycemia, seizures, coma
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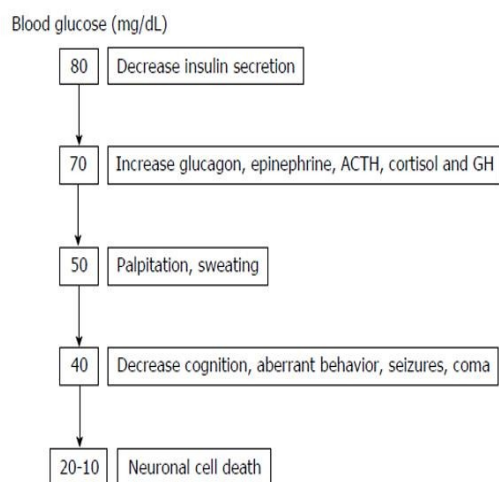
## Introduction

Epileptic seizures is also associated with the electrolyte abnormalities and other metabolic derangements [1]. In the daily activity we can frequently encounter the low blood sugar [2]. Some non-metabolic risk factors such as stroke, traumatic brain injury, anoxic encephalopathy, lesions in the brain, CNS infection may also cause seizure. Sometimes the electrolyte imbalance like sodium, potassium may also cause seizure. The most common neurological symptom is coma which is related to hypoglycemia. Epileptic seizures in relationship with hypoglycemia is a rare condition. The most common symptom of hypoglycemia are cold sweating, headache and confusion.

In addition with some neurological signs and symptoms are delirium, hypothermia, brain stem dysfunction, stroke like illness and focal or generalized seizures may occur [3]. Hypoglycemia can be very dangerous if untreated it may lead to seizures, loss of consciousness, coma and death because brain can't make glucose or store very much glycogen it requires continuous supply of glucose from

blood circulation. Hypoxia and hypoglycemia decrease cerebral oxygenation which might disrupt the cell membrane bound Na-K+ ATPase with net movement of Na+ into brain [4]. Hypoglycemia doesn't decrease brain ATP or phosphocreatine [5,6]. Duff-Nelson and Lowry [4] explained that hypoglycemic coma leads to lack of specific energy such as glucose and its metabolites (lactate and glutamate) used as energy sources of brain under low blood glucose levels [7,8,9].

In some conditions like hyperglycemia, insulin which is absent may directly increase the Na-K+ transport into brain [10]. Such effect of insulin may present during hypoglycemia which may lead to accumulation of ions in brain which further cause cerebral edema. This cerebral edema may be responsible for the hypoglycemia and hypoxic coma which lead to brain edema by two different pathological entities. Changes in the brain water and electrolyte metabolism may occur during hypoglycemia and hypoxia to define the mechanism of coma.



**Figure: 01 Flow chart representation :Mechanism of seizures**

### Case Report

A male patient of 35 years old admitted to KGH with a chief complaint of seizures, generalized tonic-clonic seizures of 5 episodes and vomiting. He was diagnosed as k/c/o of epilepsy presented as status epilepticus due to hypoglycemia. The patient has past history of seizures or epilepsy since 5 yrs and on medication with tab.phenytoin 100mg po BD .patient came with chief complaint of involuntary movements of all 4 limbs for 5 min each ,6 episodes since yesterday evening i.e 21/11/2020 last episode on 2-3am i.e 22/11/2020 along with loss of consciousness ,no gain of consciousness in between seizure episodes. Patient has altered sensorium of 2 episodes and has severe involuntary movements along with drooling of saliva, moaning along with blood in saliva scanty in amount. He doesn't have any tongue bite, involuntary micturition, defecation and no prolling of eye balls and no postictal confusion and also no headache and trauma. Patient was reported history of vomiting since 3 times contains food particles .history of weakness of right lower limb since 2hrs. Patient has similar complaint in past roughly 6-7 months back even with drug intake. Bowel and bladder habits altered sleep and appetite is decreased. He is known smoker and non-alcoholic. He was given inj lorazepam 2cc in 3cc NS slow IV stat bolus and inj 25% dextrose 100ml IV stat bolus followed by 1.DS @150ml/hr.followed to monitor 2<sup>nd</sup> hrly, inj zofer 8 mg iv BD.patient GRBS of 42mg/dl after giving dextrose it is 247mg/dl. Patient condition became normal after 2 days and had been discharged on third day.

### Discussion

Hypoglycemia may cause neurological symptoms, only limited epidemiological data are available on the relationship between the incidence of seizures and hypoglycemia. Neither the neurology nor the endocrinology may unequivocally stated that hypoglycemia cause seizure because it may be due to

alcohol abuse,metabolic abnormalities due to renal problems or stroke. A large Australian regional diabetes center found that the prevalence of epilepsy in children with type 1 diabetes was similar to that in the general population and noted that health care providers should not assume that a seizure in a type 1 diabetic patient is the result of hypoglycemia [11]. Banting concluded that,'subcutaneous injections of dextrose solutions is the antidote for convulsions and other symptoms, so that the animal (rabbit) in a few minutes becomes restored to a tolerably normal condition.

### Conclusion

Seizure not only associated by the non-metabolic disturbance but also metabolic disturbance in which hypoglycemia induced seizures is the most predominate one. Hypoglycemia induced seizures is the one which can't be known by the patient condition which can be misunderstood as normal seizures or due to drug incompliance. Low blood glucose levels may be one of the cause of CNS symptoms which may be seizures, if it is not treated on time it may lead to coma. Immediate treatment must be there for the patient of these conditions. Mainly I conclude that hypoglycemic induced seizures should not be overlooked when the patient has a history of diabetes or if the patient id diabetic.

### Ethical Approval

This study received support from king George hospital, Visakhapatnam.

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### Author Contribution

All authors contributed equally to this work.

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