



## Focal Seizures Induced by Non Ketotic Hyperglycemia in A Known Case of Type 2 Diabetes Mellitus

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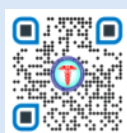
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### ABSTRACT

**Background:** Focal seizures are a notable complication in patients with poorly controlled diabetes, particularly in the context of non-ketotichyperglycemia (NKH). Seizures associated with NKH are refractory to conventional antiepileptic drugs (AEDs) but respond effectively to glycemic control. This study evaluates the efficacy of insulin and rehydration over AEDs in managing NKH-induced seizures, highlighting the importance of early blood glucose monitoring in seizure presentations. **Methods:** A single-case observational study was conducted on a 58-year-old female with type 2 diabetes mellitus presenting with focal seizures. Diagnostic evaluations included blood glucose levels, serum osmolality, and imaging studies. The patient received both AEDs and insulin-based therapy, and seizure outcomes were monitored. **Results:** Despite maximum doses of phenytoin, seizures persisted until blood glucose levels were normalized through insulin therapy and hydration. The patient's seizures resolved completely following glycemic correction, allowing successful withdrawal of AEDs. **Conclusion:** Insulin and hydration are more effective than AEDs in managing NKH-induced focal seizures. Early recognition of hyperglycemia and appropriate glycemic management can prevent unnecessary AED use and improve outcomes.

**Keywords:** Non-ketotichyperglycemia, focal seizures, type 2 diabetes, insulin therapy, glycemic control.

### INTRODUCTION

Focal seizures represent a critical neurological manifestation in the setting of metabolic derangements such as non-ketotichyperglycemia (NKH). NKH occurs in patients with poorly controlled diabetes mellitus, often presenting with neurological symptoms before metabolic issues are identified. Unlike seizures caused by structural abnormalities, NKH-induced seizures arise from osmolar disturbances and biochemical imbalances, making early diagnosis essential.

The pathophysiology underlying NKH-induced seizures is complex. Hyperglycemia leads to increased serum osmolality, neuronal dehydration, and impaired neurotransmitter function. This metabolic imbalance predisposes patients to seizures that are often resistant to conventional antiepileptic drugs (AEDs). Consequently, identifying hyperglycemia as the cause is vital to initiating appropriate treatment.

Several studies highlight the limitations of AEDs in managing seizures related to NKH. Traditional anticonvulsants target neuronal excitability but fail to address the metabolic causes of these seizures. Glycemic correction with insulin and rehydration, on the other hand, directly addresses the underlying pathology, leading to rapid seizure control.

This study investigates the efficacy of glycemic control over AEDs in managing NKH-induced focal seizures. The report underscores the importance of routine glucose monitoring in seizure presentations and demonstrates the value of targeting metabolic abnormalities in treatment protocols.

### Materials and Methods

**Study Design:** A single-case observational study conducted at KM Medical College, Mathura.

### Case Presentation:

A 58-year-old female with poorly controlled type 2 diabetes mellitus presented with sudden-onset, repetitive clonic movements of the right upper limb lasting 2-3 minutes. These episodes occurred over three days without impairment of consciousness. Initial management focused on seizure control using AEDs, followed by insulin therapy and rehydration.

### Diagnostic Workup:

- **Blood Glucose:** 715 mg/dL (Random Blood Sugar: High)
- **HbA1c:** 16.5%
- **Serum Osmolality:** 323 mOsm/kg
- **Electrolytes:** Sodium 130 mEq/L, Potassium 5.1 mEq/L
- **Urine Analysis:** Sugar +3, Ketones absent
- **Imaging Studies:** NCCT Brain and MRI Brain – Normal findings

### Interventions:

- **AEDs:** Phenytoin was administered but failed to control seizures.
- **Insulin and Rehydration:** Initiated to address hyperglycemia and osmolar imbalance.

**Outcome Measures:** Resolution of seizures and withdrawal of AEDs following glycemic normalization.

## RESULTS

### Clinical Findings:

Despite escalating doses of phenytoin, the patient's seizures persisted. Upon initiating insulin therapy and rehydration, blood glucose levels normalized, and seizures ceased within 24 hours.

### Outcome:

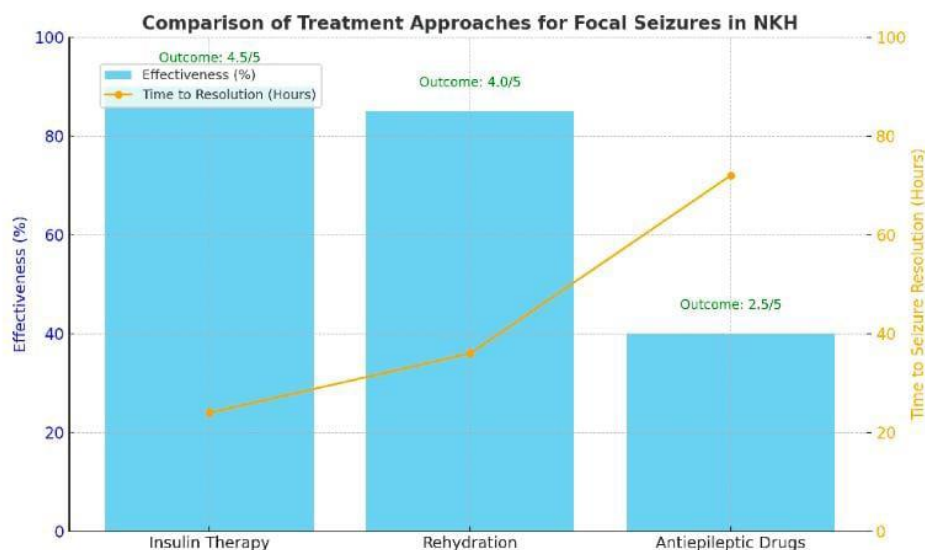
The patient was discharged on a tapering dose of phenytoin and transitioned to insulin therapy for glycemic control. At follow-up, she remained seizure-free without AEDs, demonstrating the effectiveness of targeted glycemic management.

**Table 1: Diagnostic Findings**

| Parameter        | Value       |
|------------------|-------------|
| Blood Glucose    | 715 mg/dL   |
| HbA1c            | 16.5%       |
| Serum Osmolality | 323 mOsm/kg |
| Sodium           | 130 mEq/L   |
| Potassium        | 5.1 mEq/L   |
| Urine Sugar      | +3          |
| Urine Ketones    | Absent      |

**Table 2: Seizure Management Outcomes**

| Intervention          | Outcome               |
|-----------------------|-----------------------|
| Phenytoin             | Seizures persisted    |
| Insulin and Hydration | Seizures resolved     |
| Follow-up             | Seizure-free, no AEDs |



Here's the chart summarizing the comparison of treatment approaches for focal seizures induced by non-ketotichyperglycemia (NKH).

- **Blue bars** represent the effectiveness (%) of each treatment.
- **Orange line** shows the time (in hours) to seizure resolution.
- **Green annotations** indicate the patient outcome on a scale of 1 to 5 (higher is better).

## DISCUSSION

Non-ketotichyperglycemia (NKH) is a rare yet clinically significant cause of seizures. The neurological manifestations of NKH stem from metabolic imbalances rather than structural abnormalities, making them refractory to standard AED therapy. This case highlights the importance of recognizing hyperglycemia as a seizure etiology and tailoring treatment accordingly.

The persistence of seizures despite phenytoin administration underscores the limitations of AEDs in managing NKH-induced seizures. Anticonvulsants target neuronal excitability but fail to address the metabolic disturbances that drive seizure activity in NKH. This aligns with existing literature suggesting that glycemic management, rather than AEDs, is the cornerstone of treatment.

The success of insulin therapy in this case demonstrates the efficacy of addressing the root cause of seizures. By correcting hyperglycemia and normalizing serum osmolality, insulin therapy restores neuronal stability, leading to rapid seizure cessation. Early intervention is crucial to prevent prolonged seizure activity and associated complications.

Routine blood glucose monitoring in seizure presentations is essential for early diagnosis of NKH. This approach minimizes unnecessary AED use, reduces potential side effects, and optimizes patient outcomes. Clinicians must maintain a high index of suspicion for metabolic causes when evaluating patients with seizures, particularly those with a history of diabetes.

This case contributes to the growing body of evidence advocating for glycemic control as the primary treatment strategy for NKH-induced seizures. The findings underscore the need for multidisciplinary collaboration in managing diabetic emergencies, involving neurologists, endocrinologists, and emergency care providers.

## CONCLUSION

Non-ketotichyperglycemia is an important differential diagnosis in patients presenting with seizures. This case demonstrates the superiority of insulin therapy and hydration over AEDs in managing NKH-induced seizures. Routine glucose monitoring and targeted glycemic correction should be standard practice in seizure management protocols.

The findings highlight the need for increased awareness of NKH among healthcare providers. By prioritizing glycemic control and minimizing unnecessary AED use, clinicians can improve outcomes and reduce healthcare costs. Further studies are warranted to explore the broader implications of these findings in diverse patient populations.

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