International Journal of Advances in Medicine
Swati et al. Int J Adv Med. 2026 Sep;13(5):335-337
https://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20262833

Case Report

Recurrent seizures in non-diabetic patient presenting with hypoglycemia: an eye opener for clinicians

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Received: 28 May 2026
Revised: 08 June 2026
Accepted: 01 August 2026

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ABSTRACT

Seizures represent transient episodes of abnormal, excessive, and synchronized electrical discharges of neurons in the brain that may be associated with various structural, metabolic, infectious, toxic, autoimmune, or genetic conditions. Metabolic disorders also include hypoglycemia which is recognized as one of the possible seizure triggers, since reduced glucose levels hinder energy metabolism in the brain resulting in neuronal dysfunction and hyperexcitability. Therefore, estimation of blood glucose should be included in the initial approach when dealing with patients experiencing acute seizures. The opposite condition-hypoglycemia caused by seizures-is very uncommon but still exists. Despite the fact that the decrease in blood glucose may appear after prolonged generalized tonic-clonic seizures as a result of increased glucose uptake by the brain and muscles, recurrent hypoglycemia after seizure termination is quite rare, especially in the absence of diabetes mellitus. Potential mechanisms of hypoglycemia development in such cases are related to increased metabolic needs during the seizure, decreased glycogen storage in the liver, decreased gluconeogenesis and in rare cases, exaggerated insulin production.

Keywords: Seizures, Hypoglycemia, Arachnoid cyst

INTRODUCTION

A seizure is characterized by sudden and abnormal electrical discharges within the brain, resulting in transient alterations in behavior, consciousness, sensation, or motor activity. Recurrent seizures may occur secondary to a wide range of etiologies. Acute symptomatic causes include central nervous system (CNS) infections such as meningitis, encephalitis, and intracranial abscesses. Metabolic abnormalities including hypoglycemia, hyponatremia, hypocalcemia, hepatic encephalopathy, and inherited metabolic disorders in children are also important contributors.

Other recognized causes include cerebrovascular disease, traumatic brain injury, intracranial space-occupying lesions, drug intoxication or withdrawal, hypoxic injury, hypertensive emergencies, and autoimmune disorders.[1]

Arachnoid cysts are cerebrospinal fluid (CSF)-filled lesions located within the arachnoid membrane. Although most arachnoid cysts remain asymptomatic and are detected incidentally, some patients may develop neurological manifestations, including seizures and epilepsy.[2] The exact mechanism underlying epileptogenesis associated with arachnoid cysts remains uncertain; however, compression of adjacent cerebral structures has been proposed as a possible explanation. Clinical manifestations, when present, are largely dependent on the size, location, and influence of the cyst on cerebrospinal fluid dynamics. Common neurological presentations include headache, seizures, hydrocephalus, developmental delay, focal neurological deficits due to compression of neural tissue, raised intracranial pressure, and compensatory enlargement of the skull.[3]

In rare situations, arachnoid cysts may also interfere with glucose regulation by affecting regions involved in

metabolic homeostasis, particularly the hypothalamus and pituitary gland. These structures play a crucial role in maintaining insulin sensitivity and glucose balance. Nevertheless, the precise mechanism through which arachnoid cysts contribute to impaired glucose regulation remains poorly understood and is likely influenced by the cyst's anatomical location, size, and surrounding structural involvement. The present case highlights an unusual presentation of seizure-induced hypoglycemia associated with an arachnoid cyst.

CASE REPORT

A 45-year-old male farmer presented with a 4-5-month history of recurrent episodes of abnormal body movements, frothing at the mouth, eye-rolling, lightheadedness, profuse sweating, and loss of consciousness lasting several hours. These episodes were consistently linked to hypoglycemia, with a recorded blood glucose level of 54 mg/dl, and symptoms typically resolved with glucose or sugar administration. Upon presentation, his random blood glucose was 43 mg/dl, and his Glasgow coma score (GCS) was E3V4M5. He was treated with intravenous fluids and dextrose, resulting in an improved GCS (E4V5M6) and normalized blood glucose (240 mg/dl). Neurological and systemic examinations were unremarkable, with no focal deficits, normal reflexes, and intact sensory and cerebellar functions. Laboratory Investigations revealed a urea level of 14 mg/dl, creatinine of 0.86 mg/dl, sodium of 139 mmol/l, potassium of 4.6 mmol/l, and calcium of 8.5 mg/dl and HBA1C of 4.1%. Insulin and C-peptide levels (0.65 ng/ml, normal range:0.78-1.89 ng/mls) were within normal ranges.[8] am serum cortisol was slightly elevated [28.39 mcg/dl (normal range: 4.3-22.4 mcg/dl)]. Non contrast Compute Tomography (NCCT) head done in an outside facility revealed a large CSF dense area (70×40 mm) posterior to cerebellar hemisphere, extending close to 4th ventricle inferiorly, suggestive of arachnoid cyst (Figure 1).

The patient was started on Antiepileptic (Levetiracetam) introduced on 24/10/24 in view of seizure management. Post-treatment with Levetiracetam, the patient remaine/d free from seizures /and hypoglycemia, suggesting potential effects of the cyst on glucose-regulating brain centers.

A 72-hour diagnostic fast under supervision did not reveal any recurrence of hypoglycemic or neuroglycopenic episodes. Patient was followed up on outpatient basis and was found to have no further episodes of seizures and hypoglycemia.

This case report highlights the possible effect of arachnoid cyst on glucose regulating centres in brain which presented as hypoglycemia induced seizures.

This case suggests a possible interaction between the arachnoid cyst and glucose-regulating brain centres, contributing to hypoglycemia and its neurological manifestations. Further research is needed to clarify this association.

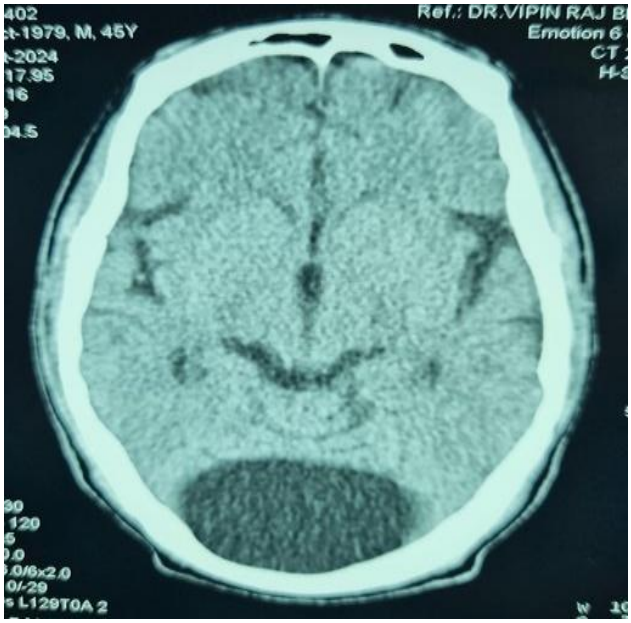

Figure 1: Non-contrast CT scan of the brain (axial section) showing a large CSF dense area (70×40 mm) posterior to cerebellar hemisphere, extending close to 4th ventricle inferiorly, suggestive of arachnoid cyst.

DISCUSSION

The human brain depends almost entirely on glucose as its principal source of energy. Neurons, particularly in adults, possess high metabolic demands and therefore require a constant supply of glucose from the circulation. Although the brain accounts for only approximately 2% of total body weight, it utilizes nearly 20% of the body's glucose-derived energy, emphasizing its vital metabolic significance.[4] Glucose metabolism is essential for maintaining normal cerebral function, as it provides adenosine triphosphate (ATP) necessary for neuronal activity, cellular maintenance, and neurotransmitter synthesis. Consequently, tight regulation of glucose homeostasis is critical for normal brain physiology, while disturbances in glucose metabolism may contribute to a wide spectrum of neurological and systemic disorders.[5]

During epileptic activity, there is a marked increase in cerebral metabolic demand, oxygen consumption, cerebral blood flow, and neuronal glucose uptake. Seizures are associated with a metabolic shift toward accelerated glycolysis and increased lactate production, a phenomenon referred to as ictal hypermetabolism. This phase is often followed by postictal hypometabolism, during which cerebral metabolic activity falls below baseline levels. The excessive glucose utilization occurring during and after seizures may predispose susceptible individuals to hypoglycemia in the postictal period.[6]

The hypothalamus and brainstem are key regulators of glucose metabolism through their extensive interactions with peripheral metabolic organs.[7] Within the hypothalamus, the arcuate nucleus (ARH), situated near the base of the third ventricle, contains neuronal populations that play a major role in energy balance and glucose regulation. Similarly, the dorsal vagal complex (DVC) of the brainstem is critically involved in detecting fluctuations in blood glucose levels and maintaining glycemic homeostasis.[8,9] The DVC comprises the nucleus tractus solitarius (NTS), dorsal motor nucleus of the vagus (DMV), and area postrema (AP). Sensory signals from the gastrointestinal tract are conveyed to the NTS and AP through vagal afferent pathways.[10] In response to hypoglycemia, GABAergic neurons within the NTS activate pathways projecting to the DMV, thereby stimulating glucagon secretion via parasympathetic mechanisms and promoting restoration of blood glucose levels.[11]

An arachnoid cyst may potentially interfere with glucose regulation by exerting pressure on neural structures involved in metabolic control. This intricate neuro-metabolic network highlights the important role of the brain in maintaining glucose balance and explains how disturbances in these systems may present as both metabolic and neurological manifestations, including seizures associated with hypoglycemia.

CONCLUSION

Glucose metabolism is fundamental for normal cerebral function, and abnormalities in glucose homeostasis can result in neurological complications such as hypoglycemia-induced seizures. Brain regions involved in metabolic regulation, particularly the hypothalamus and brainstem, are essential for maintaining glycemic stability. Structural lesions or metabolic disturbances affecting these centers may contribute to impaired glucose regulation and associated neurological manifestations. Further studies are needed to better elucidate the underlying mechanisms linking neurological disorders with altered glucose metabolism and to develop effective therapeutic strategies for improving clinical outcomes in such patients.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Langenbruch L, Krämer J, Güler S, Möddel G, Geßner S, Melzer N, et al. Seizures and epilepsy in multiple sclerosis: epidemiology and prognosis in a large tertiary referral center. J Neurol. 2019;266:1789-95.
2. Cryer PE, Axelrod L, Grossman AB, Heller SR, Montori VM, Seaquist ER, et al. Evaluation and management of adult hypoglycemic disorders: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metabol. 2009;94(3):709-28.
3. Pourmotabbed G, Kitabchi AE. Hypoglycemia. Obstet Gynecol Clin N Am. 2001;28(2):383-400.
4. Erbsloh F, Bernsmeier A, Hillesheim H. The glucose consumption of the brain and its dependence on the liver. Archiv fur Psychiatrie und Nervenkrankheiten, vereinigt mit Zeitschrift fur die gesamte Neurologie und Psychiatrie. 1958;196(6):611-26.
5. Mergenthaler P, Lindauer U, Dienel GA, Meisel A. Sugar for the brain: the role of glucose in physiological and pathological brain function. Trends Neurosci. 2013;36(10):587-97.
6. Meldrum B, Chapman A. Metabolic consequences of seizures. In: Brady S, Siegel G, Albers RW, Price DL, editors. Basic Neurochemistry: Molecular, Cellular and Medical Aspects. 6th ed. Philadelphia (PA): Lippincott-Raven. 1999.
7. Roh E, Song DK, Kim MS. Emerging role of the brain in the homeostatic regulation of energy and glucose metabolism. Exp Mol Med. 2016;48(3):e216.
8. Grill HJ, Hayes MR. Hindbrain neurons as an essential hub in the neuroanatomically distributed control of energy balance. Cell Metab. 2012;16:296-309.
9. Ritter S, Li AJ, Wang Q, Dinh TT. Minireview: The value of looking backward: the essential role of the hindbrain in counterregulatory responses to glucose deficit. Endocrinology. 2011;152:4019-32.
10. Schwartz GJ. The role of gastrointestinal vagal afferents in the control of food intake: current prospects. Nutrition. 2000;16:866-73.
11. Xu H, Boychuk JA, Boychuk CR, Uteshev VV, Smith BN. Nicotine enhances inhibition of mouse vagal motor neurons by modulating excitability of premotor GABAergic neurons in the nucleus tractus solitarii. J Neurophysiol. 2015;113:1165-74.

Cite this article as: Swati, Garg A, Deol A, Sheetal, Kant R. Recurrent seizures in non-diabetic patient presenting with hypoglycemia: an eye opener for clinicians. Int J Adv Med 2026;13:335-7.