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# Hypoparathyroidism as a cause of recurrent seizures

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**ABSTRACT**

Metabolic imbalances are among the most common causes of recurrent seizures. Herein, we aimed to report a 16 year-old man with idiopathic hypoparathyroidism presented to our outpatient clinic with recurrent seizures having been introduced antiepileptic drug five months ago. Upon focal to bilateral tonic– clonic seizure with the duration of 2-3 minutes three times in a month, he had applied to a pediatrician. Carbamazepine treatment had been introduced with suspicion of epilepsy, but his seizures had continued with same intervals and durations. The onset of the first seizure in an unexpected age and focal seizure characteristics of the patient referred to our outpatient clinic were evocative features of a probable underlying disorder. Hypocalcemia and hyperphosphatemia were detected on laboratory examinations. Serum parathyroid hormone level was measured as 0.2 ng / L (8-24 ng / L) with normal vitamin D level. Computed tomography of brain revealed seizures symmetrical cerebral calcifications. We considered his episodes as symptomatic seizures. After calcium and vitamin D replacement, he got completely seizure free. The history of this case highlights the importance of searching the secondary causes of acute symptomatic seizures other than epilepsy, in particular for the patients having the first seizure in an unexpected age with focal characteristics. A complete cessation of seizures could be achieved with the control of serum calcium levels in patients with hypoparathyroidism.

**Keywords:** hypoparathyroidism, hypocalcemia, seizure, epilepsy

**ÖZ**

**Rekürren Nöbetlerin Nedeni Olarak Hipoparatiroidizm**

Metabolik bozukluklar, tekrarlayan nöbetlerin sık nedenleri arasındadır. Bu yazıda, beş ay önce başlayan tekrarlayan nöbetler nedeniyle antiepileptik tedavi başlanan idiyopatik hipoparatiroidizmli 16 yaşındaki bir erkek hastayı bildirmeyi amaçladık. Fokal başlayıp bilateral tonik-klonik hale dönüşen ve 2-3 dakika süren nöbetlerin bir ay içinde üç kez tekrarlaması üzerine bir pediatri hekimine götürülmüştü. Epilepsi şüphesiyle karbamazepin tedavisi başlanmıştı, ancak nöbetleri aynı aralıklarla ve sürelerle devam etmişti. Polikliniğimize yönlendirilen hastadaki ilk nöbetin beklenmedik bir yaşta oluşu ve fokal özellikler taşıması, altta yatan olası bir hastalığın varlığını düşündürdü. Laboratuvar testlerinde hipokalsemisi ve hiperfosfatemisi mevcuttu. Serum paratiroid hormon düzeyi 0,2 ng / L (8-24 ng / L) olarak ölçülürken, D vitamini düzeyi normaldi. Beyin bilgisayarlı tomografisi simetrik serebral kalsifikasyonları gösterdi. Kalsiyum ve vitamin D tedavisi sonrası hastanın nöbetleri durdu. Bu olgunun öyküsü, özellikle beklenmedik yaşlarda fokal nöbetlerle başvuran hastalarda olası metabolik nedenlerin araştırılmasının öneminin altını çizmektedir. Hipoparatroidili hastalarda serum kalsiyum seviyelerinin kontrolüyle nöbetler kesilebilmektedir.

**Anahtar Sözcükler:** hipoparatroidi, hipokalsemi, nöbet, epilepsi

## INTRODUCTION

Seizures are transient signs due to abnormal excessive or synchronous neuronal activity in the cortex. The term 'acute symptomatic seizures' refers to the seizures developing in association with an underlying systemic or neurological disorder. In opposite to the condition in epileptic patients, the symptomatic seizures usually resolve in a short term after the alleviation of triggering disturbances. Therefore, the epilepsy defined as a clinical condition characterized with 'unprovoked' seizures or with the diagnosis of an peculiar epileptic syndrome unlike the acute symptomatic seizures.[1] The recurrent symptomatic seizures could be prevented if the permanent cerebral damage is avoided with appropriate treatments. Therefore a long-term antiepileptic medication is not recommended for them.[2] Electrolyte imbalances are among the common treatable causes of recurrent symptomatic seizures. Hypoparathyroidism which is characterized by hypocalcemia and hyperphosphatemia is one of the rare causes of symptomatic seizures. Herein, we aim to report a patient with idiopathic hypoparathyroidism presented with recurrent symptomatic seizures.

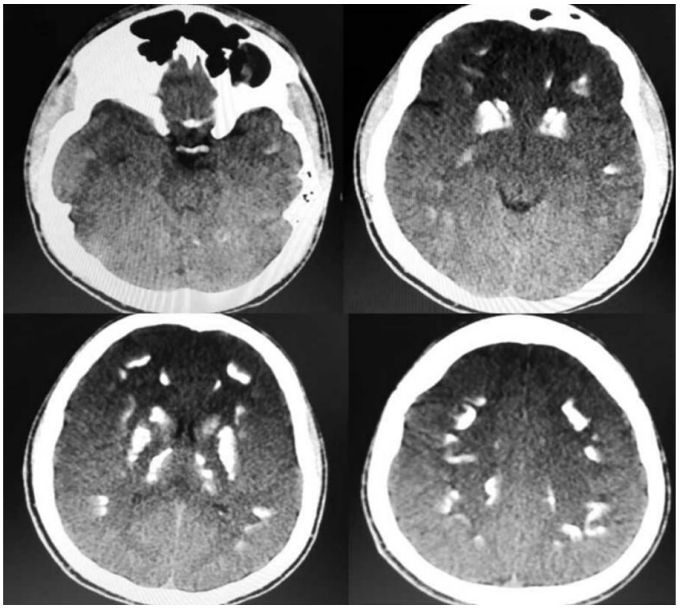
## CASE PRESENTATION

A 16 year-old man admitted to our outpatient clinic with recurrent seizures. Upon focal to bilateral tonic–clonic seizure with the duration of 2-3 minutes three times in a month, he had applied to a pediatrician. Carbamazepine treatment (400 mg/day) had been introduced with suspicion of epilepsy, five months ago. The family could not make the recommended laboratory analyses. His seizures had continued with same intervals and duration. He admitted to our hospital for getting a health report from our medical board, and was referred to our outpatient clinic. The neurological examination was normal. The focal characteristics of his seizures and onset of the first seizure in an unexpected age were evocative features of a secondary cause for us. We determined hypocalcemia (7.4 mg / dL) and hyperphosphatemia (9.3 mg / dL ). Then, serum parathyroid hormone level was measured as 0.2 ng / L (8-24 ng / L) with normal vitamin D level (34.4 ng / L). Brain computed tomography showed bilateral symmetrical calcifications in the basal ganglia and subcortical area (see the Figure). His episodes were defined as symptomatic seizures due to hypocalcemia secondary to hypoparathyroidism. We consulted him to our pediatric health and diseases department, but he dropped from the follow up. Three months later, his family brought him back to our outpatient clinic to continue the procedures to receive the medical board report. We measured the serum parathyroid hormone level as 0 ng / L, calcium as 5.5 mg / dL, and phosphorus as 11.3 mg / dL. Interictal electroencephalography was normal with hyperventilation and intermittent photic stimulation. We consulted him to our pediatric endocrinology department. Calcium acetate (4000 mg / day), calcium carbonate (7500 mg / day), vitamin D3 (3520 IU / day) and calcitriol (0.75 µg / day) were administered. Twenty days later, his serum calcium level was 8.4 mg / dL and phosphorus level was 8.6 mg / dL, and he got completely seizure-free. There was not any pathological finding on thyroid and neck ultrasound. We stopped antiepileptic treatment. He reported that the seizures did not occur again during the outpatient clinic control at the end of the third month.

## DISCUSSION

The patients admitted with a first seizure should be searched for any other underlying causes before considering them as having the first seizure of an epileptic syndrome. Seizures that occur during the course of systemic diseases (e.g. infections, ketoacidocis, hypoglycemia, hypocalcemia) or events affecting the central nervous system (e.g. trauma, meningoencephalitis, vascular events) are called symptomatic seizures. It is important to recognize the symptomatic seizures as they are not expected to recur if the underlying disorder is treated appropriately, and do not require long-term anti-epileptic drug treatments unlike the epileptic patients. Hypoparathyroidism is also a rare cause of symptomatic seizures. Hypocalcemia may cause a decrease in excitatory threshold and an increase in neuronal transmission and neuromuscular excitability in these patients.[3] It may also lead to increased susceptibility of hippocampal neurons to seizures, and impaired cerebral functions due to the cerebral edema, increased intracranial pressure, and metabolic disturbances.[4] Differential diagnosis of seizures has a particular importance in the patients with hypoparathyroidism as the tetanus observed frequently during the hypocalcemic states can be misinterpreted as an epileptic seizure. In addition, intracerebral calcifications, neuropsychiatric symptoms, cognitive disorders and extra-pyramidal system disturbances could presented with the symptoms mimicking epileptic seizures. The peculiar features on the history of the present case, such as a relatively unexpected age of onset for the first seizure and the focal characteristics of his episodes, are suggestive for a secondary cause. Besides, his seizures persisted despite the carbamazepine treatment, and this resistance also led us to get a more detailed anamnesis and investigate the probable etiologies. Then, the symmetrical cerebral calcifications on computed tomography and the hypocalcemia provided us to suspect hypoparathyroidism. The diagnosis was confirmed when the low serum parathyroid hormone level was detected. There are a few previous reports regarding the patients presented with the recurrent seizures despite the use of antepileptic drugs and diagnosed as having hypoparathyroidism after a long time as in present case.[5-11] In a recent one year follow-up study of 42 patients, Liu et al. revealed that antiepileptics did not affect the frequency and duration of seizures in these patients, including the seizures suspected to be resulted from structural insults like subcortical calcifications.[12] The present case also used an carbamazepine approximately for eight months in total, but the frequency or duration of his seizures did not change. However, his seizures ceased completely after proper control of calcium levels. In conclusion, clinicians should made the necessary examinations for the probable underlying etiologies in patients experiencing the first seizure in unexpected ages. Investigations

**Figure.** Bilateral symmetrical calcifications in the basal ganglia and subcortical structures on computed tomography of brain.



for metabolic, structural and infective causes have a particular importance for the patients having focal symptoms during their seizures and/or antiepileptic drug resistance. The history of present case underlines that getting a detailed history and reviewing the performed tests are essential for the proper evaluation of recurrent seizures.

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