Insulinoma May Present as Epilepsy: A Case Report

İnsulinoma, Epilepsi Kliniği ile Presente Olabilir: Bir Vaka Raporu

İnsulinoma ve Epilepsi / Insulinoma and Epilepsy

Halil Murat Şen, Çağdaş Balcı, Adile Özkan, Handan Işın Özışık Karaman
Nöroloji Ana Bilim Dalı, Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Çanakkale, Türkiye

Özet

İnsülinomaya bağlı gelişen hipoglisemiler epileptik nöbet benzeri klinik bulgularla prezente olabilir ve elektroensefalografi bulguları da epilepsi ile uyumlu olabilir. Bu nedenle insülinoma hastaları senelerce tedaviye dirençli epilepsi nedeni ile takip edilebilmektedir. Bizim vakamız da 20 yaşında kadın hastaydı ve epilepsi nedeni ile takip edilmekteydi. Hastanın takiplerinde kan testlerinde hipoglisemi saptandı ve yapılan incelemelerde hastaya insülinoma tanısı konuldu. Biz de bu vakayı özellikle tedaviye dirençli epilepsilerde insülinomanın unutulmaması gereken bir etyolojik neden olduğunu vurgulamak amacıyla sunduk.

Anahtar Kelimeler

İnsülinoma; Hipoglisemi; Epilepsi

Abstract

Hypoglycemia developing linked to insulinoma may present with clinical findings similar to epileptic seizures and electroencephalography findings may be in accordance with epilepsy. As a result some insulinoma patients are followed as epilepsy resistant to treatment for years. Our case was a 20-year old female patient and was followed for epilepsy. The blood tests indicated hypoglycemia and the result of examination, the patient diagnosis was insulinoma. We present this case with the aim of emphasizing the importance of not forgetting insulinoma as an etiology in epilepsy resistant to treatment.

Keywords

Insulinoma; Hypoglycemia; Epilepsy

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Corresponding Author: Halil Murat Şen, Nöroloji Ana Bilim Dalı, Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Terzioğlu Kampüsü, 17000, Çanakkale, Türkiye.
T.: +90 2862183738 GSM: +905326766455 F.: +90 2862180018 E-Mail: hmuratsen@gmail.com

Introduction

Insulinoma is generally benign and originates in the pancreatic beta-islet cells. The annual incidence of this frequently-seen pancreatic endocrine tumor is calculated at 0.12-0.4/100,000 [1]. The symptoms of the disease may be multivariate: neuropsychiatric, cardiovascular, and gastrointestinal. Due to the variety of these symptoms and because some initial findings may be mild, diagnosis may be difficult [2]. Central nervous system findings are common in insulinoma and may occur as confusion, agitation, loss of consciousness, transient ischemic attack, psychosis, and epilepsy [1]. Patients with insulinoma may have epileptic seizures during hypoglycemic periods. These patients do not have partial epilepsy in fact but may be misdiagnosed due to clinical characteristics and typical electroencephalography (EEG) abnormalities [2]. As these patients do not have epilepsy in fact, they may be monitored for years for medication-resistant epilepsy [1].

We present this case becasue of having insulinoma who had been following as epilepsy resistant to antiepileptic treatment for years.

Case Report

A 20-year-old female patient applied to our clinic with a complaint of unresolved epileptic seizures in spite of anti-epileptic treatment. The patient's complaints began 2 years before with loss of consciousness. After this initial loss of consciousness, her complaints continued in the form of seizure attacks. During these attacks, the patient had disordered consciousness, nonsense gaze, slipping of the eyes, and contractions of the extremities. These episodes generally lasted in 10 minutes and occurred 1 or 2 days per month, and could be repeated several times in the same day. One year after the onset of complaints she received a diagnosis of epilepsy. For 1 year, she used 600 mg/day carbamazepine; however, her complaints continued without change and the patient applied to our clinic due to continued seizures. Brain magnetic resonance imaging (MRI) was normal. EEG showed sharp waves, more obvious especially in the left temporal and parietal regions (Fig. 1). This sharp wave activity frequently spread to all areas of both hemispheres (Fig. 2). Routine blood tests were normal. Due to these frequent seizures, the carbamazepine dose was raised to 800 mg/day. On follow-up fifteen days later, the seizures had continued with the same frequency. Blood testing found a fasting blood glucose level of 28 mg/dl. Testing repeated one day later found a fasting blood glucose level of 36 mg/dl. The patient was examined for a hypoglycemic etiology. A fasting test found blood glucose levels of 32 mg/dl at 18 hours. Abdominal computerized tomography (CT) found a mass lesion in the pancreas. The patient was operated on with a diagnosis of insulinoma.

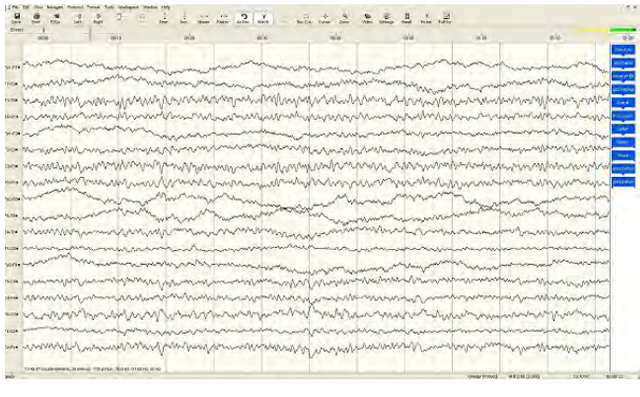


Figure 1. Sharp waves, more obvious especially in the left temporal and parietal regions

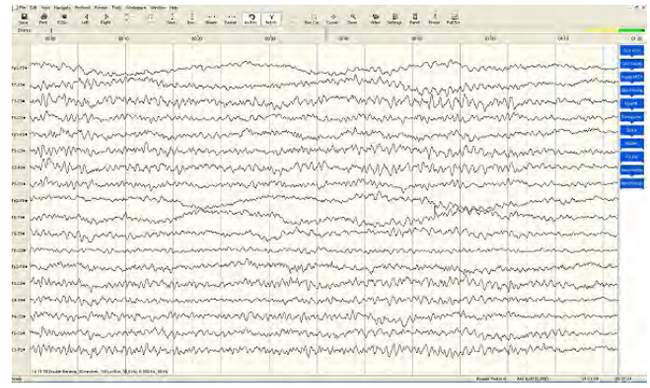


Figure 2. Sharp waves, spread to all areas of both hemispheres

Discussion

We report an insulinoma case with continued seizures although she had been using anti-epileptic treatment for years. The patient was diagnosed as insulinoma 2 years after onset of clinical symptoms of epileptic seizures. Correct diagnosis of insulinoma may be delayed by 1 to 30 years [1], and there are several reasons for this delay. The main reason is the low incidence of insulinoma and as a result the possibility of it may not be thought of during the differential diagnosis. Second, some symptoms of insulinoma, such as confusion, personality changes, seizures, and movement disorders are nonspecific findings in many neurological and psychiatric diseases. Lastly, as insulin release is pulsatile, fasting blood glucose levels may be within normal limits [3].

Our insulinoma patient with clinical symptoms mimicking epilepsy had abnormal findings on EEG. Widespread or focal slowing may be observed on EEG; at the same time, interictal epileptic discharge and electrical seizures may be observed [4]. Animal experiments have triggered hypoglycemic seizures and observed that seizures originated in the mesial temporal structures, such as the amygdala and hippocampus [5]. These clinical and EEG findings together may cause the clinician to think the diagnosis is epilepsy. As mentioned above, since insulin release is pulsatile, fasting blood glucose levels may be within normal limits. Our patient was followed for epilepsy due to clinical findings and EEG findings, and previous blood tests had not identified hypoglycemia.

Hypoglycemic attacks at intervals should bring insulinoma to mind. One of the most appropriate diagnostic tests is the fasting test. In this test, the patient fasts for 72 hours and glucose levels are checked every 1–2 hours until they fall below 50 mg/dl. [1].

Insulinoma may be observed with typical epileptic seizures while EEG findings are in accordance with epilepsy, and measured fasting blood glucose levels may be normal. Our aim in presenting this case is to point out the importance of metabolic causes in the differential diagnosis of epilepsy.

Competing interests

The authors declare that they have no competing interests.

References

1. Findley A, Arenas RB, Kaplan EL. Insulinoma. In: Percopo V, Kaplan EL, editors. GEP and multiple neuroendocrine tumours. Padua: Piccin,1996.p.299–318.
2. Striano S, Striano P, Manganelli F, Boccella P, Bruno R, Santoro L, et al. Distal hypoglycemic neuropathy. An insulinoma-associated case, misdiagnosed as temporal lobe epilepsy. Neurophysiol Clin 2003;33(5):223-7.
3. Mitrakou A, Fanelli C, Veneman T, Perriello G, Calderone S, Platanisiotis D, et al. Reversibility of unawareness of hypoglycemia in patients with insulinomas. N Engl J Med 1993;329(12):834-9.
4. Wang S, Hu HT, Wen SQ, Wang ZJ, Zhang BR, Ding MP. An insulinoma with clinical and electroencephalographic features resembling complex partial seizures. J Zhejiang Univ Sci B 2008;9(6):496-9.
5. Tokizane T. Sites of origin of hypoglycemic seizures in the rabbit. AMA Arch Neurol Psychiatry 1957;77(3):259-66.

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