Case report / *Olgu sunumu*

Multiple migration defects of cerebral cortex in a patient with epileptic seizures mimicking panic attacks

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ABSTRACT

Disorders of neuronal migration can lead to malformations of the cerebral neocortex and presentation and clinical course of patients these patients are variable. In this paper, we report a patient with episodic attacks initially thought to be panic attacks which on further investigation were identified to be seizure episodes to be associated with cerebral cortical migration abnormalities. ***(Anatolian Journal of Psychiatry 2011; 12:158-159)***

Key words: *migration defects, cortex, epileptic seizures, anxiety*

Panik ataklarını taklit eden epileptik nöbetleri olan bir hastada serebral kortikal migrasyon defektleri

ÖZET

Nöronal migrasyon anomalileri serebral neokorteks malformasyonlarına neden olabilir ve bu hastaların ilk başvuruları ve klinik gidişi değişkenlik gösterebilir. Bu yazıda, başlangıçta epizodik panik atakları olduğu düşünülen, ancak ileri incelemelerden sonra bunların serebral kortikal migrasyon anomalileri ile ilişkili epileptik nöbetler olduğu anlaşılan bir olgu sunulacaktır. ***(Anadolu Psikiyatri Derg 2011; 12:158-159)***

Anahtar sözcükler: *Migrasyon defektleri, korteks, epileptik nöbetler, anksiyete*

INTRODUCTION

Disorders of neuronal migration can lead to malformations of the cerebral neocortex, which greatly increase the risk of seizures.[1] Initial presentation and clinical course of patients with malformations of cortical development are variable and they seem to be correlated with the extent of cortical involvement.[2] In this case report, we report on a patient whose initial symptoms were seizures resembling anxiety attacks.

CASE REPORT

A 19 year-old woman, referred to our hospital with the complaint of paroxysmal events characterized by anxiety, fear of loss of consciousness, gastric distress and palpitation. These attacks had started two weeks ago, but clouding of consciousness and urinary incontinence were observed during her last four attacks. We also learned that she had been referred to emergency services many times and diagnosed as having panic attacks and at her last attack she was referred to a psychiatrist and a neurologist. In her past medical history taken by her family there was a doubtful infectious disease when she was a six-month baby. Mild right hemiparesis was observed by her family after this infection disease, but her mental and motor development were normal. In neurological examination, her orientation and cooperation was normal. Mild right hemiparesis was found in her muscle strength examination. Hyperreflexia and extensor plantar response was seen on right side. Magnetic resonance imaging (MRI) of the brain revealed thickening and smoothing of the gyrus (lissencephaly, pachygyria) at the superior part of the anterior temporal lobes that was more prominent at right side, and a linear lesion

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similar to cerebrospinal fluid density at the level of centrum semiovale extending to atrium of the lateral ventricle of the left parietal lobe. There was also a 2x2 centimeters phorencephalic appearance near the linear lesion (schizencephaly) Her electroencephalography revealed epileptic activity at the right temporal area of the brain hemispheres. The patient was put on antiepileptic treatment (lamotrigine 150 mg/day) and after this medication she became seizure free.

DISCUSSION

The genetic malformations of the cerebral cortex are usually characterized by malposition and abnormal differentiation of grey matter.[3] Causes of these malformations are abnormal neuronal migration and abnormal cortical organization.[4,5] Patients with malformations of cortical development present with a wide spectrum of clinical manifestations ranging from asymptomatic cases to those with epilepsy and neurodevelopmental problems.[2] Our patient's initial complaint was paroxysmal phenomenon of anxiety, fear of loss of consciousness, gastric distress and palpitation mimicking panic attacks and her diagnosis became definite after seizure type had been changed. After neurological and electrophysiological examinations, an emergent MRI performed to investigate the etiology of acute onset seizures with localizing EEG findings. Suspicion of an epileptic phenomenon in this patient has been very helpful for the final diagnosis. As 80% of patients with cortical dysplasia present with epilepsy and 60% of them are drug-resistant, a definite diagnosis is crucial for the patients while planning treatment.[6] Our patient's initial diagnosis was panic attacks and it is not surprising because some symptoms of panic attacks such as anxiety, fear, derealization or depersonalization, altered autonomic functions and cardiovascular fluctuations may be found in ictal and interictal periods of epileptic patients. Partial seizures initially misdiagnosed as panic attacks may be a clue for the diagnosis of symptomatic right temporal lobe epilepsy.[7] In this case, the EEG showed epileptic activity at the right temporal area convenient to the literature. Psychiatrists commonly consider a diagnosis of temporal lobe epilepsy when a patient presents with atypical panic attacks, atypical response to medications or an abnormal EEG.[8] In our case lack of anxiolytic response was another cause for suspicion of the psychiatrist.

Mental retardation was reported in 74% and focal neurological deficits were reported in %30 of patients with cortical dysplasia.[4] In this case mild right hemiparesis was found in her neurological examination but it was thought to be secondary to a central nerve system infection in her childhood. So, cranial imaging which had not been performed to her before showed us the structural lesions. Additionally, it is interesting that there were only mild clinical symptoms and neurological findings in this patient inconvenient to these multiple cortical lesions. However, it is also reported that there are no correlations between the type of cortical dysplasia and the severity of the clinical picture, especially the level of mental retardation and presence of drug-resistant epilepsy.[4] There was not enough time to decide about the drug-resistance, but our patient is seizure free after the treatment of 150 mg/day lamotrigine.

In conclusion, in clinical practice it is still not easy to make the proper diagnostic steps in patient suffering from epileptic seizures with psychiatric symptoms. Some patients, as our case, who have structural brain lesions can have only minor neurological deficits that are thought to be secondary to events in childhood period and suspicion of the clinician can be the most important step for the diagnosis in the absence of major clinical findings.

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