



Seizures Presented with Severe Apneic-Bradycardic Attacks: A Case Report

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Convulsive apnea is rarely seen after neonatal period, and it is infrequently accompanied to severe bradycardia. We present a 4-month-old boy with convulsive apnea associated with bradycardia. Careful clinical assessment of the infant revealed partial seizures characterized by staring, tonic deviation of the eyes and head just before apneic-bradycardic attacks. This case indicates that comprehensive procedures is not always necessary for the diagnosis of epilepsy.

Key Words: Convulsion, Apnea, Bradycardia, Epilepsy

Apneik-Bradikardik Ataklarla Karakterize Epilepsi Olgusu

Yenidoğan döneminden sonra konvülfif apne ve onu eşlik eden bradikardi nadirdir. Biz bradikardinin eşlik ettiği konvülfif apneli 4 aylık bir bebek sunuyoruz. Dikkatli klinik değerlendirme, bu bebekte apneik-bradikardik ataklardan hemen önce olan boş bakma, baş ve gözlerde tonik deviasyon ile karakterize parsiyel epilepsi tanısını koydu. Bu olgu epilepsi tanısında her zaman ileri tetkiklerin gerekli olmadığını gösterir.

Anahtar Kelimeler: Konvülfizyon, Apne, Bradikardi, Epilepsi

Seizure is a common neurologic disorder in the pediatric age group and occurs in 3-5% of children. Epilepsy occurs in 0.5-1.0% of the population and starts in childhood in 60% of cases.¹ Seizures in infants can be difficult to diagnose, because of nonspecific initial symptoms such as apnea, bradycardia, and cyanosis.^{2,3} We present an infant suffered from recurrent, life threatening apneic-bradycardic episodes that was finally diagnosed as a convulsive disorder.

CASE REPORT

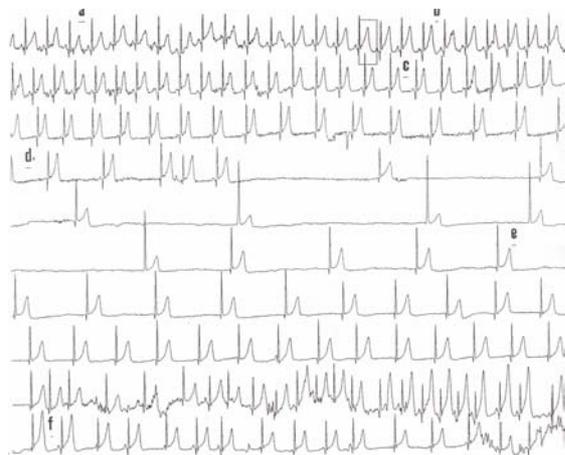
This 4-month-old boy was born at term by spontaneous vaginal route after an uncomplicated pregnancy with a birth-weight of 3600 grams. There was no problem in his birth history. There was first degree consanguinity between his parents. He was healthy 5 days prior to admission. The patient was admitted with repeated attacks of cyanosis, apnea, bradycardia, state of decreased consciousness and flexion posture in both upper and lower limbs lasting for about two minutes. His physical examination including detailed neurological examination showed no abnormality.

The following laboratory studies were normal: urine analysis, complete blood count, liver and renal function studies, serum electrolyte levels including sodium, potassium, calcium, and magnesium, cerebrospinal fluid analysis and urine-blood aminoacide profiles. Repeated interictal routine and long electroencephalographic (EEG) recordings did not reveal any epileptic activity. Examination for gastro-esophageal reflux was unremarkable. Magnetic resonance imaging (MRI) scan of the brain was normal. Electrocardiogram and echocardiogram yielded normal results.

He experienced four severe attacks which were requiring resuscitation during his follow up in clinic. Cardiac and respiratory monitoring revealed a gradual decrease of both heart and breathe rate during the attacks. The reason-

result relationship could not be differentiated between apnea and bradycardia. Due to the lack of simultaneous monitorization of cardiac rhythm, respiration and EEG, 24 hour Holter monitoring was performed. Electrocardiographic (ECG) recordings were obtained during two separate attacks. The times of the characteristic hallmarks (staring, tonic deviation of the eyes and head, beginning of the apnea, beginning of the resuscitation, duration of the event and initiation of the spontaneous breathing) of the event during the attacks were recorded. Sequence of the signs were as following; an uncommunication state characterized by staring, tonic deviation of the eyes and head, apnea, flexion posture in both upper and lower limbs, gradual decrease in heart rate, cyanosis, and a full recovery after resuscitation (Figure 1). This clinical picture was interpreted as a complex partial seizure. On the 3rd day of admission, intravenous phenobarbital, a loading dose of 20 mg/kg followed by a maintenance dose of 5 mg/kg/d, was administered. During the first two days of phenobarbital, he developed additional three similar attacks. The serum level of phenobarbital was 13.2 µg/ml (normal: 15-35 µg/ml). He had no new attacks after additional dose of phenobarbital.

Figure 1: Rhythm strip, recorded during an episode while the 24-hour Holter monitoring, shows the sequence of the signs. a: staring, b: tonic deviation of the head and eyes, c: beginning of apnea, d: beginning of bradycardia, e: beginning of resuscitation, f: full recovery



DISCUSSION

Paroxysmal conditions are usually interpreted as seizure at initial stage and they are routinely consulted with pediatric neurology units. Epilepsy is one of the most important paroxysmal neurological disorders in childhood,⁴ however, nonepileptic

paroxysmal events (NEPE) such as breath holding spells, migraine and its variants, syncope, cardiac conduction disorders and arrhythmias, masturbation, sleep disorders, and Sandifer's syndrome are often seen in the same population. Several movement disorders including jitteriness, shuddering attacks, tic disorders and familial paroxysmal choreoathetosis mimic epilepsy.⁵ For this reason, the correct diagnosis of epilepsy may be too difficult. The true diagnosis of paroxysmal events may be impossible in some cases during the first year of life than in older age groups.⁵ The history and physical examination of our patient and the observed clinical characteristics of the attacks did not suggest the NEPE, and also routine investigations excluded both the cardiac disorders and gastro-oesophageal reflux.

It had been first reported by Jackson that apnea may be an ictal manifestation. It was confirmed by using EEG that this phenomenon originates in the limbic system.⁶ Apneic attacks suggest a convulsive disorder if they are associated with tonic deviation of the eyes and head or characteristic extremity movements.⁷ The characteristic evident of our case appeared as apneic-bradycardic episodes requiring resuscitation, but the most important clue in the diagnosis of seizure was very brief periodical tonic deviation of the head and eyes on the right direction occurring just before these episodes.

Presence of tachycardia in association with prolonged apnea suggests the possibility of a convulsive disorder.^{8,9} On the contrary, apnea with bradycardia usually is not a sign of convulsive disorders.⁷ However, transient bradycardia/sinus arrest can occur during or soon after convulsive-apneic attacks.⁹ The probable mechanism of bradycardia associated with convulsive-apnea is the ictal parasympathetic overactivity due to cardiorespiratory reflexes as well as an additional contributory factor secondary to hypoxia.⁹ The diagnosis of convulsive disorders must depend on the clinical basis and the presence of epileptic discharges on the EEG which is carried out during the attacks along with appropriate techniques. Also, an ambulatory EEG recording may help to clinicians in this process.⁵ Therefore, it is clear that the true diagnosis of a convulsive disorder can be established in an epilepsy monitoring unit. But these equipments are very expensive and usually unavailable. Also, the transportation of some patients to a developed center possessing an epilepsy monitoring unit may be life-threatening. Therefore, we tried to make a diagnosis on clinical basis. Although four interictal

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EEG recordings were in normal limits, our case showed characteristic head and eye movements just before the attacks suggesting typical complex partial seizure.⁷ Also, the Holter recordings cleared the reason-result relationship between the apnea and bradycardia. Depending on these clinical evidences, an antiepileptic drug was started. Disappearance of the seizures soon after the AED strongly supported the diagnosis of convulsive disorder.

Consequently, the clinical success obtained in the case reported herein indicates that it always does not justify the necessity of the utilization of the sophisticated facilities for the diagnosis of epilepsy. However, since there were serious negative effects of an incorrect label of epilepsy on both child's and parents' lives, misdiagnoses must be avoided.¹⁰ Therefore, we point out that a perfect description, careful clinical assessment and repeated examinations are very important in distinguishing true seizures from NEPE and that clinicians should always keep in mind the utilization of the sophisticated facilities in the differential diagnosis whenever they are available.

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