Epileptic "Paroxysmal Arousal" in the Differential Diagnosis of NREM Parasomnia "Confusional Arousal"

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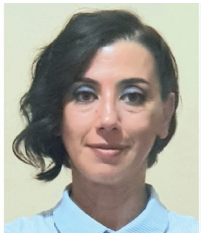

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Presented in: The case (Patient 1) was previously presented as an oral presentation at the 21st National Sleep Medicine Congress, held online from November 5-7, 2021. The authors of the presentation were Yılmaz Öz B, Ser MH, Benbir Şenel G, and Karadeniz D, and the topic was the differential diagnosis between epileptic "paroxysmal arousal" and NREM parasomnia "confusional arousal" in the context of a case.

The case (Patient 2) was previously presented as a poster at the Clinical Neurophysiology EEG-EMG Congress held from 26 to 30 October 2022 in Bodrum-Muğla. The poster, titled "Presentation of Two Patients Diagnosed with Epileptic Paroxysmal Arousal and Its Differential Diagnosis with Confusional Arousal" (Poster Presentation - P026), was authored by Çalışkan B, Karadeniz D, and Benbir Şenel G.

Abstract

Sleep-related hypermotor epilepsy (SHE) should be differentiated from NREM parasomnias in terms of a similar clinical presentation. The lack of ictal and/or interictal epileptic encephalographic (EEG) features in SHE complicates the differential diagnosis. Moreover, epileptic paroxysmal arousal (PA) does not present with associated hyperkinetic motor events typical of SHE, and this should be carefully evaluated from confusional arousal (CA), a type of NREM parasomnia. In this term, this paper aims to present three patients referred to the Sleep and Disorders Unit with the prediagnosis of CA but diagnosed as epileptic PA following video-polysomnography (PSG) with multichannel EEG recordings; and aims to discuss the clinical, EEG, and PSG characteristics of these patients on the basis of literature data.

Keywords: Sleep-related hypermotor epilepsy, epileptic paroxysmal arousal, NREM parasomnias, confusional arousal

INTRODUCTION

Patients with walking and searching behaviors that occur episodically during sleep at night were first described by Pedley and Guilleminault in 1977.[1] Despite the absence of any accompanying epileptic electroencephalographic (EEG) findings, these attacks were found to be of epileptic origin due to a positive response to antiseizure medication (ASM). This condition was later termed "epileptic nocturnal wandering" (ENW). Afterward, similar cases were reported to occur during the non-rapid eye movement (NREM) sleep phase at night, characterized by dystonic-ballistic complex motor movements, resembling somnambulism attacks, and defined as hypnagogic or nocturnal paroxysmal dystonia (NPD).[2] The idea that NPD is epileptic in origin, even in the absence of epileptic EEG abnormalities, has gained widespread acceptance owing to the brief duration and stereotypic nature of the attacks, as well as the response to ASM. In 1986, Peled and Lavie[3] reported on a series of patients who exhibited frequent paroxysmal awakenings during the NREM sleep stage and suggested that these episodes were of epileptic origin. In the follow-up, Montagna et al. published cases similar to Peled and Lavie's patients, which were characterized by stereotypical recurrent nighttime awakenings, some of these cases also showed EEG anomalies, they used the term "paroxysmal awakening" [paroxysmal arousal (PA)].[3,4] Episodic occurrences of ENW, NPD, and PA during nocturnal sleep have been identified as components of nocturnal frontal lobe epilepsy.[4,5] In subsequent years, the designation "Sleep-related hypermotor epilepsy" (SHE) has been increasingly recognized as an appropriate term, given the identification of epileptic foci beyond the frontal lobe through invasive recordings in affected patients.[6-8]

Although the diagnosis of SHE is more likely in the presence of major attacks characterized by excessive, unusual, and high-amplitude motor movements, it can be challenging to differentiate between NREM parasomnias in the presence of minor attacks with calmer

and less pronounced features such as paroxysmal awakening. According to the International Classification of Sleep Disorders, confusional arousal (CA) are defined by confusion, disorientation, meaningless speech, and impaired responsiveness, particularly in the first half of the night and during deep NREM sleep.[9] Although simple and purposeful motor movements are commonly observed in CA, sometimes more complex and violent movements can accompany these NREM parasomnia attacks. Both CA and epileptic paroxysmal awakenings can have a familial tendency, and parasomnias can also be observed in family members of patients with epilepsy. Furthermore, epilepsy and NREM parasomnias can coexist in the same individual, leading to diagnostic challenges.[10,11] This article aims to discuss the differential diagnosis of two types of nocturnal arousal disorders, confusional awakening and paroxysmal awakening, by presenting three cases. The patients were initially diagnosed with confusional awakening but were later found to have epileptic PA.

CASE PRESENTATIONS

Patient 1

A 10-year-old female patient with unknown developmental delays was referred to our sleep laboratory due to complaints of nocturnal snoring, respiratory pauses, sweating, and abnormal movements during sleep. Additionally, she reported experiencing episodes of sudden awakening, standing up, and staring blankly, which occurred exclusively during sleep, and recurred 2-3 times per night, almost every night since she was 7 years old. According to the patient's mother, these episodes included convulsive movements that started in both arms and spread to the entire body. The duration of episodes was short, lasted minutes, occurring at any time during the night, but were more frequent during the first half of the night. Rarely, the patient reported feeling nauseous or experiencing chest heaviness before the episodes. While some episodes were associated with the urge to urinate, the patient did not actually void during the episodes. The patient reported receiving treatment with valproic acid and levetiracetam, but there was no improvement in her symptoms. The patient scored 2 points on the Frontal Lobe Epilepsy and Parasomnias (FLEP) scale, suggesting epilepsy.

In her medical history, the patient was the fourth child out of five siblings. It was revealed that the mother had an uncomplicated pregnancy and delivery and had no history of febrile convulsions or head trauma. Developmental milestones were reached properly. There was a first degree consanguinity between the patient's parents. Febrile convulsions were reported in her uncle's children.

The patient's previous medical history was unremarkable, with a normal EEG both at wakefulness and during the short sleep period during the daytime. Cranial magnetic resonance imaging (MRI) revealed no obvious pathology. Polysomnography (PSG) and EEG recordings conducted in the sleep laboratory did not reveal any sleep-related respiratory or movement disorders. Pre- and morning awakening EEG recordings showed synchronous and symmetrical tonic and phasic elements of sleep in both hemispheres. During the same night, six episodes occurring in NREM sleep stages were observed. All episodes began with eye opening, and one episode was accompanied by standing up in bed. During the episodes, rapid activity on the left, especially localized in the P3 electrode, was noted, but a typical ictal pattern was not observed. The final episode, which occurred in the morning, started with a very stereotypical pattern characterized by eye opening and rising in bed (Figure 1) and progressed to a bilateral tonic-clonic seizure (Video 1). Similar to the other episodes, rhythmic evolution was observed in rapid activities that can be localized especially on the P3 electrode. No interictal epileptic activity was detected during the entire examination. Brain positron emission tomography revealed the hypometabolism in the left prominent bilateral lateral temporal cortex and the left inferior parietal cortex. With these findings, the patient was diagnosed with epileptic PA and started on carbamazepine treatment. The patient was referred to the epilepsy unit for further evaluation.

Patient 2

A 28-year-old female patient with a prior diagnosis of Joubert syndromepresented to our sleep laboratory with complaints of snoring and respiratory arrest during sleep; and feeling tired upon waking up in the morning. The patient had been experiencing episodes of falling asleep after headaches, accompanied by sensations of shapes and spinning bananas since the age of twelve, leading to a diagnosis of epilepsy and subsequent treatment with carbamazepine. The patient had also previously experienced bilateral tonic-clonic seizures before undergoing tooth extraction. After being treated with ASM, the patient reported that the shape fluctuations largely disappeared. The patient's mother reported incidents of the patient waking up at night and looking around, staring blankly, and then returning to sleep, as well as other sleep-related episodes, such as talking, kicking, moving her legs back and forth, and picking up objects with her hand, for approximately one year. These episodes could occur 1-2 times per night at any time of the night and lasted for a brief duration of less than 1 min. Based on the FLEP scale assessment, the patient scored 0 points, which suggests parasomnia as the likely diagnosis.

According to the patient's medical history, she was the second-born of two siblings. The mother's pregnancy was uneventful, and the patient was delivered at term via cesarean section due to cord entanglement. There was no history of febrile convulsions or head trauma. The patient was noted to be hypotonic at birth and achieved sitting, walking, and sentence-level speech at the ages of one, 3.5, and six years, respectively. The parents were second-degree relatives, and there was a history of epilepsy in the patient's cousin's children.

The patient's previous EEG at wakefulness and short sleep periods during daytime yielded normal results. Cranial MRI findings were consistent with Joubert syndrome. PSG examination, conducted in conjunction with a 16-channel EEG at our sleep and disorders laboratory, revealed no sleep-related breathing or movement

MAIN POINTS

- In the differential diagnosis of sleep-related hypermotor epilepsy, it is important to consider non-rapid eye movement (NREM) parasomnias and more rarely REM parasomnias.
- In the differentiation of these disorders, detailed clinical evaluation, video examinations to examine whether the episodes show stereotypic features , and evaluation with polysomnographic examination including 16-channel electroencephalographic are recommended.
- However, it should be remembered that these two conditions can be seen together.

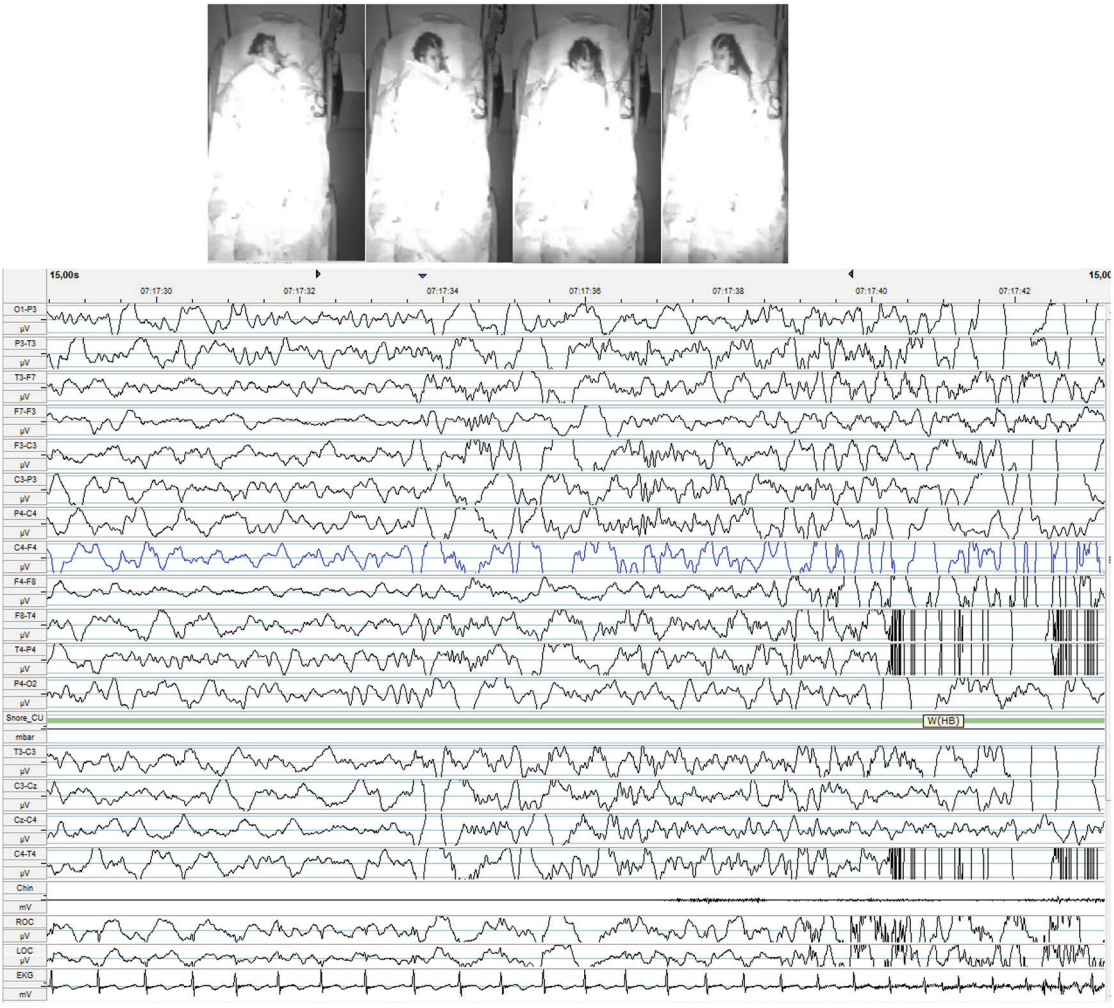

Figure 1. EEG image of Patient 1 at the onset of the epileptic PA episode that evolved into bilateral tonic-clonic seizures
EEG: Electroencephalography, PA: Paroxysmal arousal

disorders. Pre- and morning wakefulness EEG recordings demonstrated a basic bioelectrical activity based on 7-8 Hz alpha rhythm. The tonic and phasic components of sleep were observed simultaneously and symmetrically in both hemispheres, and sleep phases could be easily distinguished. During the study, eight episodes occurring in the N3 sleep stage were observed on the same night. In these fairly stereotypical episodes, following a generalized delta paroxysm on EEG, movements in the patient's right hand and slight deviation of the head to the right were observed. The prominent sharp wave activities in the left frontocentral regions during superficial NREM sleep stages showed an evolution during attacks (Figure 2). The patient was diagnosed with epileptic PA. As the episodes during sleep were electrophysiologically consistent with epileptic seizures, the dosage of the patient's ASM was increased, and the patient was monitored in the epilepsy unit.

Patient 3

A 20-year-old male patient presented with complaints of opening eyes, looking frightened, and making movements as if shaking something while sleeping at night, which had been present since the age of 7 years. The patient experienced episodes of shouting and, in rare cases, getting up and running. The attacks were observed almost every night, particularly in the first half of the night and early morning and recurred 1-4 times per night. The patient reported that the attacks never resolved spontaneously despite receiving various treatments, including different antiseizure and tricyclic antidepressant medications. Although initial treatment provided relief, the attacks eventually resumed. The patient's FLEP scale was calculated as 0 to -1 point (in favor of parasomnia).

There was no significant feature in her past and family history, and no family members with similar complaints were present. The patient's previous waking EEG and cranial MRI were normal.

PSG examination performed using 16-channel EEG in our sleep and disorders unit showed normal baseline bioelectrical activity in the pre- and morning wakefulness EEG recordings. During the N3 sleep stage of the first half of the night, an attack characterized by eye-opening, fear expression, vocalization, and rapid transition to a semi-sitting position in bed was observed. In the second episode, also monitored in the first half of the night and during the N3 sleep stage, a hyperkinetic motor seizure was recorded, characterized as eye-opening, fear expression, and vocalization, followed by rapid, aimless, and high-amplitude limb movements (Video 2). During both attacks, on the background of non-lateralized or non-localized baseline fast activity, an asynchronous activity with high amplitude and sharp elements lasting 2-3 s followed by a suppression pattern

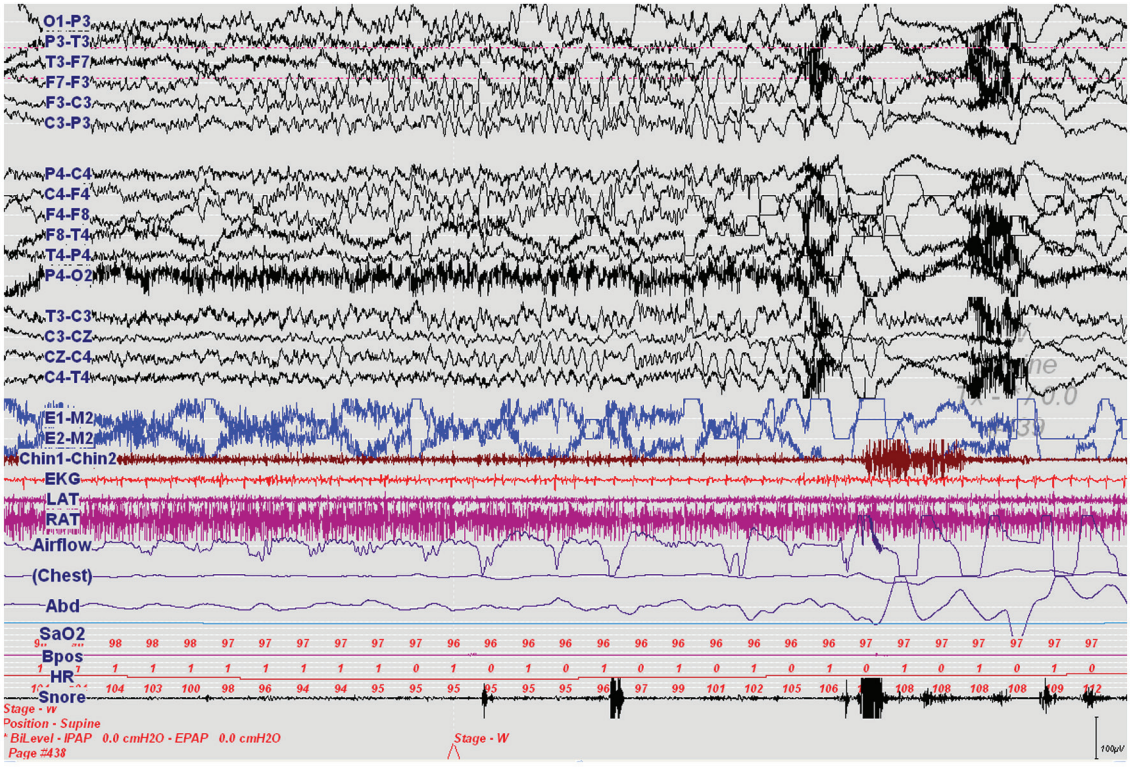

Figure 2. Temporal and spatial propagation of the sharp wave activity that started in the left fronto-central area during the attack in Patient 2

Table 1. Differential diagnosis of SHE, NREM parasomnias and REM sleep behaviour disorder

Features	SHE	NREM parasomnias	REM sleep behavior disorder
Symptom onset (age)	<30 years	<10 years	>50 years
Sleep stage	N1, N2, sleep stage transitions	N3	R
Time of attacks	Any time	First half of the night	Second half of the night
Duration of attacks	5-60 seconds	2-30 minutes	seconds-2 minutes
Frequency of attacks	Clusters in the night	Sporadic, rarely clusters in the night	Sporadic, rarely clusters in the night
Onset-end	Sudden, with arousal reaction	Slow, with N2 sleep stage	Sudden
Semiology	Highly stereotypic, hypermotor, asymmetric, tonic/dystonic	Complex, variable, similar but not stereotypical, eyes are open	Partly stereotypical, eyes are closed, dream recall
Consciousness	Variable	Confusion	Normal after attack
Risk of causing damage	Low	High	Middle
PSG	>50% normal	Rhythmic, hypersynchronous delta activity	REM sleep without atonia

SHE: Sleep-related hypermotor epilepsy, NREM: Non-REM, REM: Rapid eye movement, PSG: Polysomnography

lasting 1 s was observed; there was no ictal epileptic activity except for muscle artifact subsequently. No sleep-related respiratory or movement disorder was detected. After carbamazepine was started, the patient was referred to the epilepsy unit for further investigations.

DISCUSSION

Differential diagnosis is challenging in nocturnal motor events without any epileptiform activity on ictal and interictal EEGs. Although clinical clues can be helpful in some cases (Table 1), this distinction is not always clear in our clinical practice. NREM parasomnias typically manifest at a younger age and commonly follow a benign course, resolving spontaneously before the age of 18 years.[12-14] Compared to epilepsies, the frequency of attacks in NREM parasomnias is lower, with incidence ranging from 1 to 4 per month. In contrast, epilepsy exhibit recurrent attacks and may even relapse during the night. The presence of stereotypic, extrapyramidal system-related findings such as dystonic posture, ballistic movements, and chorea-athetosis as well as violent and agitated motor behaviors indicate epilepsy. In PA, the absence of hyperkinetic motor behavior is a challenging factor in the diagnosis.

Although all three patients were referred to our sleep and disorders unit with suspicion of CA, the occurrence of recurrent stereotypic attacks throughout the night led us to evaluate them further in detail and reach the diagnosis of PA. The diagnosis of PA was also supported by the fact that the attacks starting in a similar manner

evolved into bilateral tonic-clonic seizures. FLEP scale used in the differential diagnosis of NREM parasomnias and SHE in our patients showed that FLEP score of PA attacks that were not accompanied by hyperkinetic motor movements or did not become bilateral tonic-clonic were mostly in accordance with NREM parasomnias, with one supporting epilepsy, but none in accordance with epilepsy.[15] It is therefore important to note that this could result in patients being misdiagnosed with CA as NREM parasomnia. In this term, it is essential for the diagnosis and differential diagnosis that all episodes, rather than a single episode, are considered in clinical evaluation and PSG recording with multiple EEG channels throughout the night.

Ictal EEG findings that accompany epileptic PA are infrequently detected in scalp EEG recordings. However, the presence of ictal EEG findings during PA episodes recorded during stereo-EEG has been demonstrated in patients in whom epilepsy surgery is planned.[16] The stereo-EEG method, which has increased usage in recent years, enables the "*in vivo*" neurophysiological evaluation of epilepsy. This method allows for assessing the physiological and pathological activity in cortical and subcortical areas during wakefulness and sleep, and intracerebral activity during epileptic seizures. In another study, the presence of a functional interaction between different superficial EEG electrodes during nocturnal seizures was recorded and defined as synchronization probability and suggested to be used in the differential diagnosis of CA and PA.[17,18] During NREM parasomnias and CA episodes, the typical EEG pattern is usually characterized by the presence of stage N1-like theta and/or intermittent alpha rhythm following delta activity.[10] In a detailed video-PSG evaluation of patients with definite NREM parasomnia and SHE, major and minor events were analyzed according to sleep stages, and the total number of motor events was found to be significantly higher in SHE patients.[19] In both groups, it was observed that the episodes occurred mostly in the N2 and N3 sleep stages, but it was reported that major episodes occurring out of the N3 sleep stage favored SHE and minor episodes occurring in the N3 sleep stage favored NREM parasomnia.

Video-PSG findings, including 16-channel EEG, of the three patients showed that epileptic EEG features were seen in only one patient. The remaining two patients did not exhibit any ictal or interictal epileptic abnormalities. In the first patient presented (Patient 1), some episodes were preceded by delta-alpha paroxysms supporting NREM parasomnias. In fact, video recordings of the last presented case (Patient 3) showed that (Video 2), the fast theta-alpha frequency rhythms mixed with hypersynchronous delta activity at the onset of the attack are similar to those seen in NREM parasomnias. Although there is a possibility of comorbidity between NREM parasomnia and epilepsy, or less commonly, an epileptic attack triggered by an arousal reaction associated with NREM parasomnia, it was concluded following multidisciplinary evaluations that all three patients exhibited stereotypical and similar attacks of the same nature. Therefore, a diagnosis of epileptic PA was made for all three patients.

Abnormal thalamocortical loops are believed to be the underlying pathophysiological mechanism between NREM parasomnias and frontotemporal seizures.[20,21] A hypothesis suggests that NREM parasomnias are caused by increased cyclic alternating patterns resulting from increased sleep instability and arousal oscillations. This pattern creates a milder stimulus in specific brain regions.[12,22,23] Conversely, epileptic seizures are believed to be triggered by the activation of a more intense stimulus within larger brain regions.[22-24] Although this article focuses on the differential diagnosis between SHE and NREM parasomnias in the context of CA and PA, REM sleep behavior disorder is also included in the differential diagnosis of more agitated and violent attacks (Table 1).[24] In addition to the conditions discussed in this article, the differential diagnosis of epileptic seizures should also include nocturnal panic attacks, nightmare disorders, and paroxysmal hyperkinetic movement disorders.[25,26] Rarely, SHE attacks may occur during the REM sleep stage, and it has been suggested that the transition from REM sleep, which has an antiseizure effect, to the waking phase may trigger the seizure. This is because spontaneous wakefulness reactions are more frequent and the wake threshold is lower during the REM sleep stage.[27]

CONCLUSION

The differential diagnosis of CA, NREM parasomnia, and epileptic PA can still be challenging, as shared by the patients presented in the video examples, despite the use of video-EEG and PSG examinations. In addition to detailed clinical evaluation, demonstration of stereotypic features of attacks with multiple attack recordings in video-PSG recordings including multichannel EEG, careful examination of EEG patterns, and multidisciplinary approaches of neurologists specialized in sleep disorders and epilepsy will ensure the correct diagnosis and an appropriate treatment plan.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: G.B.Ş., S.N.Y., D.K., Design: G.B.Ş., Ö.U., S.N.Y., D.K., Data Collection or Processing: G.B.Ş., R.U., S.N.Y., D.K., Analysis or Interpretation: G.B.Ş., S.N.Y., D.K., Literature Search: G.B.Ş., R.U., Ö.U., S.N.Y., D.K., Writing: G.B.Ş., R.U., Ö.U., S.N.Y., D.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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Video 1. Two episodes of Patient 1 in the same night: the first of the five stereotypically recurrent epileptic PA characterized by eye opening and sitting up in bed; and the last episode starting similar to epileptic PA but evolving into bilateral tonic-clonic seizure
PA: Paroxysmal arousal
http://dx.doi.org/10.4274/ArchEpilepsy.2023.2023.231085.video1

Video 2. Epileptic PA in Patient 3 characterized with eye opening, fear expression, vocalization and rapid transition to semi-sitting position on the bed, followed by SHE episode with similar onset but accompanied by hyperkinetic motor movements
PA: Paroxysmal arousal, SHE: Sleep-related hypermotor epilepsy
http://dx.doi.org/10.4274/ArchEpilepsy.2023.2023.231085.video2