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Treatment of patients with coexisting epileptic and nonepileptic seizures

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Abstract

A majority of studies have reported a rate of concurrence of epileptic seizures (ESs) in patients with nonepileptic seizures (NESs) of about 10–18%. We explored the relationship between the two paroxysmal disorders (ESs and NESs) in a series of patients with both, and report a treatment for these patients that proved remarkably effective: reduction of the dose of antiepileptic drug to the minimum required to achieve optimal freedom from seizures. NESs are hypothesized to have a psychobiological basis, and it has been proposed that they be recognized as posttraumatic startle seizures. Excessive suppression of epileptic paroxysmal activity appears to favor the expression of posttraumatic paroxysmal activity in patients with both paroxysmal disorders, and the manifestation of ESs and NESs tends to alternate. Of etiological significance is the finding that the patients commonly have both a personal history of trauma and a family history of epilepsy.

Keywords: Epileptic seizures; Nonepileptic seizures; Concurrence rate; Excess of antiepileptic drugs; Startle seizures; Alternating pattern of epileptic and nonepileptic seizures

1. Introduction

Nonepileptic seizures (NESs) are detected in 10–40% of patients evaluated at epilepsy centers. In contrast to epilepsy, the etiology and pathogenesis of this other major paroxysmal disorder have remained poorly understood, and no effective treatment has become well recognized and established [1].

Prior to the advent of modern epileptology, nonepileptic seizures (termed *hysterical seizures*) were recognized mainly among female patients, and the role of a premorbid history of painful traumatic events, including physical or sexual abuse, was noted. The term *hysterical* became to be considered pejorative and was deleted. The current modern label of *pseudoseizures* for NESs has a more pejorative sound to patients, suggesting they are faking a real seizure. Use of the more neutral terms *nonepileptic* and *psychogenic*, although more acceptable, awaits recognition of the biological nature of this particular seizure event, which is different from the epileptic seizure (ES), yet nevertheless very real.

Highly significant among patients with NESs is an early history of painful traumatic events, such as physical and/or sexual abuse. Kretschmer [2] had a view of hysterical events similar to Freud's, but further postulated that the hysterical reaction made use of biologically preformed mechanisms, particularly in the form either of a motility storm consisting of regression in a state of terror with hyperkinesis, trembling, and convulsing, or of sham death with stupor, immobilization, or a hypnoid state. The same primitive paroxysmal reactions may be observed as healthy individuals experience acute mortal fear when faced with a sudden life-threatening catastrophe. The startle or surprise response, the neural basis of which has been well established in experiments in animals, may represent the biologically preformed neural mechanism of the NES paroxysm [3,4]. The present study represents an effort toward better understanding the biology of the NES phenomenon by reviewing the treatment of patients with coexisting epileptic and nonepileptic seizures.

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A majority of studies have reported a rate of concurrence of epilepsy in patients with NESs of about 10–18%; most frequently, ESs and NESs occur sequentially rather than simultaneously, often beginning with the former [5]. This group of patients has received insufficient attention. We have explored the alternating relationship of the two types of paroxysmal disorders and report a novel treatment for patients with a history of both ESs and NESs.

2. Case reports

The five cases reported here represent the majority of patients with coexisting ESs and NESs we were able to treat over the past 3 years.

2.1. Patient 1

The first patient, who drew our attention to the pathology and treatment of patients with both ESs and NESs, was a woman, seen first in 2003, at the age of 57.

The patient had some college education, but had been able to work only as a waitress or clerk until age 54. Her father had always been verbally abusive toward her mother and their four children, drank excessively, and committed suicide when our patient was 25 years old. She was married and divorced three times; two of her husbands had been verbally abusive, and several nonmarital relationships were similarly stormy. She had four children from husbands and boyfriends. She reported no family history of epilepsy.

From ages 12 to 42, she experienced nocturnal, generalized tonic–clonic seizures (often with tongue bites) two or three times a month, within 1 h of falling asleep. After a brief seizure-free period of about 2 years, she began to have seizures of a different type: the seizures occurred mostly in daytime, and often, she felt mildly shocked immediately before the seizure. She often experienced weakness in her right or left shoulder joint, her tongue seemed paralyzed, and there was some memory loss. The seizures lasted $\frac{1}{2}$ to 2 min and were hardly ever the same. From ages 44 to 57, after increasing neurological efforts to control her seizures, they gradually increased to an average frequency of 10 daily. She was taking three anticonvulsant drugs, and vagal nerve stimulation (VNS) had been initiated 18 months before she was referred to us. She felt increasingly miserable, with depressive moods, fatigue, and headaches that had become constant; these symptoms further worsened on VNS treatment. Her neurologist referred her to us when home EEG monitoring showed no epileptiform changes during multiple episodes of seizure activity.

On referral, we began to decrease her antiepileptic medication gradually, terminated the VNS, and prescribed 100 mg of nortriptyline at bedtime. Her seizures decreased, and she experienced a remarkable recovery from her headaches and depressive symptoms within a few weeks. Gabapentin was phased out first, then levetiracetam. Sertraline 50 mg in the morning was added to the nortriptyline; Inderal LA 160 mg twice daily was prescribed, as was topiramate 100 mg twice daily for weight loss. A decrease in phenytoin to 100 mg twice daily seemed to increase ESs, and she was maintained on 300 mg daily (her initial dose had been 200 mg twice daily). After 18 months, she became seizure-free for a full month, then had two seizures that appeared to be epileptic.

She was monitored over 5 days at the Comprehensive Seizure Monitoring Unit. On admission, her phenytoin was tapered rapidly. The patient's interictal EEG revealed intermittent left temporal sharp waves, sometimes occurring in runs. On the fifth day of monitoring, she had 20 nonepileptic events clustered in a 1-h period. They were characterized by irregular leg and arm movements that waxed and waned and finally stopped when the patient had one complex partial seizure (event 21) characterized by dystonic posturing of the right arm and tonic extension of the left arm. The ictal EEG correlate of this event was rhythmic sharp activity emanating from the left temporal region and spreading to the left frontocentral region with postictal slowing. She was informed about the findings and the need to maintain merely a small amount of anticonvulsant medication.

One month after the monitoring, she reported four episodes of prolonged minor seizures of the nonepileptic type. Her only antiepileptic drug was phenytoin 100–200 mg. She felt vigorous and was free of headaches and dysphoric symptoms. One month later she reported only one very minor nonepileptic event (stiffening of both hands and holding them together for 15 s). Phenytoin was replaced by lamotrigine 100 mg twice daily. For the second-half of her 3-year treatment period, her recovery persisted, with only minor difficulties. She would be free of seizures for 4–6 weeks, then might experience at times two or more brief partial seizures in a day. A slight recurrence of headaches responded to an increase in sertraline to 100 mg in the morning.

2.1.1. Comment

An increasing frequency of seizures and decline in general well-being (with headaches and depression) after an increase in antiepileptic treatment efforts are characteristic of NESs in our experience. Her illness was reversed by reduction of the antiepileptic drugs (AEDs) to a minimum and maintenance on antidepressant medication. Her NESs began only after her ESs were fully controlled. This response is reminiscent of the important phenomenon of "forced normalization" [6], but suggests here, specifically, a polarity between ESs and NESs. Of particular interest is the finding, on the fifth day of video/EEG monitoring, how her series of NESs was terminated by the first ES.

2.2. Patient 2

The alternating relationship of NESs and ESs is further demonstrated by the course of the illness, over 11 years, of our second patient.