Sleep Medicine 14 (2013) 1217–1220

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Brief Communication

Parasomnia overlap disorder: a distinct pathophysiologic entity or a variant of rapid eye movement sleep behavior disorder? A case series

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ARTICLE INFO

Article history:
Received 26 February 2013
Received in revised form 13 May 2013
Accepted 4 June 2013
Available online 28 August 2013

Keywords:
Parasomnia overlap disorder
Neurodegeneration
REM sleep behavior disorder
Disorder of arousal
Sexsomnia
Status dissociatus
Sleep-related dissociative disorder

ABSTRACT

Background: Parasomnia overlap disorder (POD) currently is classified by the International Classification of Sleep Disorders, Second Edition (ICSD-2) as a variant of rapid eye movement (REM) sleep behavior disorder (RBD), and therefore its diagnosis also implies counseling the patients on the increased risk for developing neurodegenerative disorders. POD pathophysiology is not clear to date.
Methods: The authors report 5 cases of POD, review the literature, and analyze previously published cases of POD.
Results: In all 5 reported cases sleep-related activity was clearly demonstrated, though the RBD component was mild or incidentally discovered. None of the patients had Parkinsonian clinical features. Based on ICSD-2 criteria, there are 139 more POD cases reported in the literature and 69. 2% are idiopathic. The POD patients had an earlier age of onset than the patients with RBD. The RBD component was milder than the disorder of arousal (DOA) in most cases. Recently an updated classification was published, which included new categories of POD. The features mentioned above and the revised classification suggests that POD is not just a subtype of RBD.
Conclusions: We propose that POD is a distinct pathophysiologic parasomnia. Further research to identify the underlying mechanism is needed. Proper counseling is necessary for patients presenting with POD at a young age of onset.

1. Introduction

Parasomnia overlap disorder (POD) is a condition that has clinical features of both a disorder of arousal (DOA) (i.e., sleepwalking [SW], confusional arousals [CA], sleep terrors [STs]) and rapid eye movement (REM) sleep behavior disorder (RBD) [1]. Although an updated and revised classification of POD is in press [2], the International Classification of Sleep Disorders, 2nd edition (ICSD-2) currently identifies POD as a variant of RBD rather than a separate disorder. This classification has potentially serious implications, as RBD constitutes a major risk factor for neurodegenerative illnesses [3], and it is considered to be caused by central cholinergic denervation in the setting of Parkinson disease [4,5,6].

We report five individuals with POD, in whom no abnormalities were present on neurologic examinations; the patients did not have sleep-disordered breathing and the DOA was a more salient feature, whereas the RBD component was less frequent or milder. All patients underwent a video-polysomnography (vPSG) with 10 electroencephalogram, 2 electrooculogram, chin electromyogram, bilateral leg electromyogram, pressure transducer, thermistor, chest and abdomen effort, electrocardiogram, and pulse oximetry channels. By analyzing our patients and the cases previously reported in the literature, we propose that POD most likely is a distinct entity and is not a subtype of RBD.

2. Case 1

A 30-year-old man presented with STs and dream-related movements that started at the age of 19 years. The STs occurred approximately 2–3 times a month. The dream-related movements

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http://dx.doi.org/10.1016/j.sleep.2013.06.012

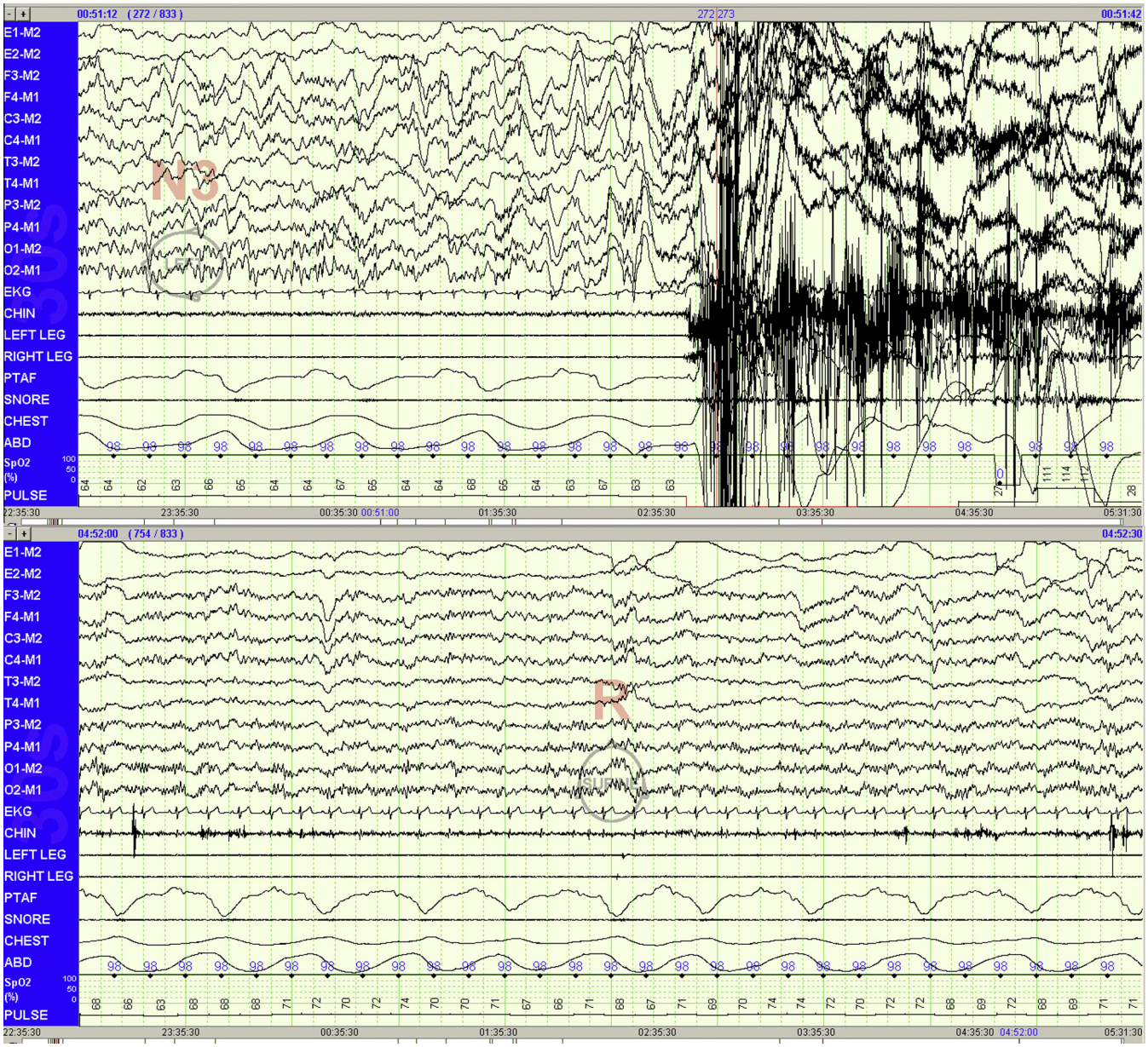

Fig. 1. The top portion of the Fig. is a 30-s epoch from the video-polysomnography (vPSG) of case 5. It demonstrates a confusional arousal arising out of stage 3 sleep. Note the slow waves dominating the first two-thirds of the epoch and the movement artifact, which partially obscures the slow-wave activity underneath as the confusional arousal occurs. The bottom portion of the Fig. is another 30-s epoch from the vPSG of Case 5. It demonstrates the elevated chin electromyogram during stage R sleep.

were rare and not injurious. In the 6 months prior to his presentation, STs had improved, which coincided with him starting venlafaxine for generalized anxiety disorder; however, the dream enactments had become more frequent. This change had resulted in him kicking his wife during sports-themed dreams. He had no other medical concerns, no history of tobacco or drug use, and only occasional alcohol consumption.

The vPSG captured two STs from stage 3 (N3) sleep associated with increased heart and respiratory rates. There also was REM without atonia (RWA) (72% of 3-s mini-epochs), with associated complex running-like leg movements throughout stage R sleep. Venlafaxine was tapered off with his psychiatrist's guidance. Clonazepam at bedtime substantially improved both types of nocturnal events (Fig. 1 and Table 1).

3. Case 2

A 72-year-old woman presented after an ill-defined sleep-related episode during which she fell and injured herself. She had STs since the age of 20 years, but their intensity and frequency increased by age 50 years. Six months prior to presentation she leapt out of bed twice because she dreamt of being chased. This enactment resulted in a minor bruise once and a laceration requiring several sutures the second time. She was not on any antidepressant agents. She had hypertension controlled by lisinopril. She quit smoking in her 30s, she did not drink alcohol, and she had no history of substance abuse. The vPSG captured three partial arousals from stage N3 sleep. There was RWA for more than 90% of stage R sleep associated with an overall increase in physical activity.