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Journal search and commentary

The importance of sleep medicine consultation for diagnosis of REM sleep behavior disorder in most patients with Parkinson's disease☆

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Objective

To determine the frequency of REM sleep behavior disorder (RBD) in patients with Parkinson's disease (PD).

Study design

Clinical and polysomnographic (PSG) evaluation of a group of consecutive PD patients with or without sleep complains. Results were compared with an age and sex matched control group of healthy subjects without sleep complains. Patients treated with benzodiazepines and antidepressants, and those with advanced PD (Hoehn and Yahr stage >3) were not included.

Methods

Thirty-three consecutive patients with mild to moderate PD stage, 21 men and 12 women with a mean age of 62.9 years, were evaluated clinically and by PSG. The PSG included audiovisual monitoring allowing detecting both of the major RBD characteristics, i.e. REM sleep movements and REM sleep without atonia (RWA). Twenty-seven (81%) patients were treated with dopaminergic agents.

RWA was defined by the method previously established by Lapierre and Montplaisir based on the submental electromyographic (EMG) tonic activity during the nocturnal PSG [1]. This study did not evaluate the submental EMG phasic activity nor either phasic or tonic EMG activity of any limb muscles.

RBD was diagnosed by PSG as the presence of RWA associated with complex motor and vocal behaviors.

RBD was diagnosed by clinical history if the minimal criteria of the International Classification of Sleep Disorders were fulfilled [2]. Four subjects were not evaluated for a clinical diagnosis.

Results

Eleven (33%) patients, ten men and one woman, met the proposed PSG diagnostic criteria of RBD. Of these, nine (81.8%) were treated with dopaminergic agents for their PD.

In five (17%) patients RBD was diagnosed by both clinical criteria [2] and PSG with audiovisual monitoring. Four (13.7%) asymptomatic patients were diagnosed as having RBD only by PSG and audiovisual monitoring criteria. Thus, the sensitivity of the minimal criteria of the International Classification of Sleep Disorders for the diagnosis of RBD was 56% (5/9).

Nineteen (58%) patients had RWA. Of these, 8 (42.1%) were asymptomatic and audiovisual PSG recording during REM sleep did not show vocalizations or abnormal movements.

Conclusions

Thirty-three percent of the PD patients had RBD as demonstrated by PSG studies with audiovisual monitoring. In some patients with PD, asymptomatic RBD and RWA may occur. In PD patients, RBD is underestimated when the diagnosis is made only by clinical history without a sleep medicine consultation and PSG recordings.

Comment

This study focused on the evaluation of the frequency of RBD in a group of patients with PD based on clinical and PSG findings. Unlike other studies, this paper evaluates the presence of this parasomnia by PSG. In addition, audiovisual monitoring synchronized with the PSG was used to

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detect abnormal behaviors during REM sleep helping to disclose more precisely the prevalence of RBD. PSG findings were compared with the patients' and bed-partners' reports suggestive of RBD, and the controls PSG results.

This study demonstrates that RBD is common (33%) in patients with PD and that, like the idiopathic form of RBD, it is more frequent in males than in females. It also shows that RBD occurs frequently in PD patients treated with dopaminergic agents, although it has been reported that in some patients these drugs may ameliorate the symptoms of this parasomnia [3]. Most of the patients with untreated RBD who several years after starting RBD developed the onset of the diurnal motor symptoms of PD (tremor, rigidity, etc.) did not report improvement of the RBD symptoms when they started levodopa or dopamine agonist treatment. The frequency of RBD in PD reported here is lower than in the patients with other neurodegenerative diseases such as multiple system atrophy and dementia with Lewy bodies.

This study confirms that in PD the diagnosis of RBD is clinically underestimated [4], showing that some subjects with RWA plus abnormal behaviors during REM sleep do not provide clinical reports of their nocturnal behaviors. On the other hand, other patients with RWA were asymptomatic and their PSG studies did not detect abnormal behaviors. These two subgroups of PD patients are assumed to represent the preclinical forms of RBD. Clinical and PSG follow-up of these patients may elucidate whether they will develop more severe symptoms and will then report having enacted dreams during REM sleep. However, the clinical symptoms of RBD depend on the patient's and bed partner's perception of abnormalities during the night. It is known that some patients with RBD do not recall dreaming, nightmares, or vigorous movements while sleeping and falsely report continuous undisturbed sleep. In such cases it is only the bed partner who reports the occurrence of the patient's abnormal behavior during the night, and for unclear reasons some patients appear reluctant to report these aggressive movements. Thus, in patients who sleep alone or whose partner does not or cannot report the behavior, RBD is an underdiagnosed condition. Even among subjects who injure themselves during the night, diagnosis and treatment can occur only after the event, which may not always be reported.

This study also demonstrates that sensitivity of the minimal criteria proposed by the International Classification of Sleep Disorders for the diagnosis of RBD is not high. On one hand, some patients with other clinical conditions such as severe sleep apnea, seizures during the night, nocturnal hallucinations related to dopaminergic drugs or mesopontine structural lesions, nocturnal-confusional states in neurodegenerative diseases, and night terrors in adults may fulfill the minimal criteria mimicking RBD. In these situations, only PSG may exclude RBD and detect these other disorders. On the other hand, RBD may be associated by chance with sleep apnea, and only PSG can detect in a subject these two clinical conditions, which require two very different treatments. Other authors [4–6] have also suggested the need for PSG with audiovisual recording for the accurate diagnosis of RBD.

This is a well designed study in which the sample evaluated consisted of consecutive patients with or without sleep complains, did not include subjects treated with drugs known to affect the sleep architecture such as antidepressants and benzodiazepines, and was compared with a matched group of healthy subjects without sleep complains. The frequency of RBD and RWA might have increased if this study: (1) only selected PD patients who complain of vigorous behavior during sleep [4]; (2) included patients treated with antidepressants [7]; and (3) evaluated the phasic submental and limb muscle activity during REM sleep.

In a prior review and commentary on a related article [7] Dr Trenkwalder emphasized both the need to consider routine PSG to exclude RBD in conditions associated with a high risk of RBD and also the importance of demonstrating improvement in quality of life and physical health if the RBD is detected and treated earlier [8]. Although we do not have the treatment outcome data for this study, it seems unreasonable to have to wait for a patient to inflict injury before initiating treatment. Clinical diagnosis alone, at least for PD patients, is insufficient to avoid injury and distress from RBD; a sleep medicine consultation and laboratory evaluation is required, particularly for patients not reporting nocturnal symptoms. It now seems clear that such an evaluation should be considered for inclusion in the routine care of patients with PD, but at what point? A routine referral of PD patients for a clinical review by sleep medicine specialists should certainly be considered, and this study may provide the needed clinical experience for developing such guidelines for evaluation.

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