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Mild Head Injury Induced Rapid Eye Movement Sleep Behaviour Disorder

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Abstract

Rapid eye movement (REM) sleep behaviour disorder (RBD) is a parasomnia characterised by REM sleep without atonia, causing dream-enacting behaviours (DEBs) or vocalisation. The aetiology of RBD is largely unknown, likely a result from breakdown of the circuits underlying REM sleep atonia. It is also strongly associated with neurodegenerative disorders. Recent studies suggested that head injury could be a risk factor for RBD. We present a case of new-onset RBD, triggered by mild head injury in a patient with well-managed severe obstructive sleep apnoea (OSA). Our case provides a rare opportunity to examine the link between head injury and RBD.

Keywords: Sleep medicine; REM sleep behavioural disorder; Head injury

Abbreviations: REM: Rapid Eye Movement; RBD: Rapid Eye Movement Sleep Behaviour Disorder; DEB: Dream-Enacting Behaviours; OSA: Obstructive Sleep Apnoea; CPAP: Continuous Positive Airway Pressure; PSG: Polysomnography; PD: Parkinson's Disease

Introduction

Sleep behaviour disorder is characterized by loss of the normal voluntary muscle atonia during rapid eye movement. This associated with excessive motor activity while dreaming and this behaviours result frequently in injuries to the patient. This case has been reported to illustrate a direct relationship between mild head injury and new-onset RBD.

Case Report

A 57-year-old Caucasian man presented with symptoms of dream-related kicking, punching, vocalisation and falling out of bed during sleep for 12 months. He was initially seen by a respiratory physician and was diagnosed with severe OSA, successfully managed with continuous positive airway pressure (CPAP). The persistence of his symptoms led to referral for neurological consultation [1].

The symptoms started after a mountain bike injury 12 months prior. He was hospitalised for pneumothorax secondary to multiple rib fractures. He cracked his helmet and sustained an episode of unconsciousness for less than 5 minutes. During hospitalisation, he had witnessed vocalisation during sleep. Post-discharge, his wife noticed his sporadic motor and vocal behaviours during sleep on an average of 3-4 times per month. He was unaware of these symptoms and was alert and oriented upon awakening.

There were no associated focal neurologic or cognitive complaints. The past medical history was unremarkable, and he was taking no regular medications. There was no family history of neurodegenerative or sleep disorders. He was a teacher, non-smoker, occasional drinker, and had no occupational pesticide exposure. Neurological examination was unrevealing. Brain MRI showed no evidence of intracranial lesions or microbleed. Polysomnography (PSG) with overnight electrocochleography revealed REM sleep without atonia.

The differential diagnosis for DEBs includes OSA, rhythmic movement disorder, sleep walking/terrors, seizures, confessional arousals, alcohol/drug use or withdrawal, post-traumatic stress disorder and sleep-related dissociative disorder [2]. OSA-induced DEBs are not associated with loss of atonia during REM sleep, and symptoms should resolve with CPAP [2]. Based on the history and excluding other possible aetiologies, the diagnosis of RBD was made. Sleep environment safety was discussed to prevent injuries and Clonazepam two milligrams at bedtime was commenced, which improved his symptoms by reducing the frequency without exacerbating his OSA.

Discussion and Conclusion

RBD is a parasomnia characterised by REM sleep without atonia, manifested as DEBs [1]. According to the 3rd edition of the International Classification of Sleep Disorders (ICSD-3), a diagnosis of RBD should meet all the following criteria:

1) Repeated episodes of sleep-related vocalisation or complex motor behaviour.

2) Behaviours are either documented by PSG or presumed to occur during REM sleep.

3) Presence of REM sleep without atonia on PSG.

4) Absence of epileptiform activity.

5) The sleep disturbance is not better explained by another medical, neurological, mental disorder, or medication/substance use. Our patient satisfied all criteria.

The pathophysiology of RBD is unclear. The common consensus suggests that it results from failure in the network underlying REM sleep atonia [3]. It is likely to be a prodromal syndrome of synuclein-mediated neurodegenerative disease, including Parkinson's Disease (PD), Lewy body dementia and multiple system atrophy [1,2]. As such, certain risk factors for PD, namely low educational level, previous head injury and occupational pesticide exposure were also predisposing factors for RBD [1]. Other risk factors for RBD include smoking, ischaemic heart disease and use of corticosteroids [1,4]. The mechanism whereby head injury increases the risk of RBD is unknown. Based on studies exploring the link between head injury and PD, head injury can cause oxidative

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stress and inflammation, potentially leading to pathological alpha-syn aggregation in the brainstem, one of the earliest sites affected [5,6]. As the main circuits for generating REM sleep are within the brainstem [3], any pathological process in this region may cause the breakdown of signalling pathways of REM sleep atonia.

To the best of our knowledge, no case has been reported to illustrate a direct relationship between mild head injury and new-onset RBD. In our case, brain MRI excluded the possibility of structural abnormalities, particularly hypothalamic lesions, causing RBD. While RBD is a possible early manifestation of neurodegeneration, no co-existing risk factors were identified. Therefore, his RBD was induced by the mild head injury, particularly considering the timeframe of onset and course of progression. Nevertheless, long term follow-up should be encouraged for the early detection of possible neurodegenerative disease.

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