

## REM sleep behaviour disorder: The stuff of dreams

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### Abstract

We describe the case of a sixty-five year old male presenting with acting out his violent dreams over a period of twelve years, causing injury to himself and his spouse. His case was not conducive to sleep studies, and the diagnosis of idiopathic REM sleep

behaviour disorder was made on positive clinical features and exclusion of possible neurodegenerative aetiologies. He showed marked response to treatment with a daily dose of clonazepam 2mg.

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### Introduction

A parasomnia is an undesirable physical and/or experiential phenomenon, which could occur in any stage of sleep. It involves skeletal muscle activity, autonomic changes, and emotional-perceptual experiences (1). REM sleep behaviour disorder (RBD) is a rare parasomnia characterised by the intermittent loss of the classic REM sleep electromyographic (EMG) atonia and elaborate motor activity associated with dream mentation (2). Sixty-percent of cases reported are idiopathic (3). Acute RBD is commonly associated with withdrawal from alcohol, benzodiazepines and barbiturates, as well as administration of tricyclic antidepressants, selective serotonin reuptake inhibitors, cholinergic agents, and monoamine oxidase inhibitors (4). The pathogenesis of antidepressant induced acute RBD remains unclear. Follow up studies suggest that a large portion of patients with RBD go on to develop Parkinson's disease or dementia with Lewy body disease (3).

Patients with REM sleep behaviour disorder can present to psychiatric practice. We describe a patient who presented with nocturnal behavioural disturbances.

### Case history

A sixty-five year old retired teacher, presented with a 12-year history of distressing violent dreams on most days of the week. The nightmares started during a family crisis, and had persisted beyond its resolution. A few months later he started enacting these violent dreams during sleep. His wife claimed that about three hours after falling asleep, he would start yelling, kicking, grabbing, punching the wall, sitting on and jumping out of bed. He had sustained bruises and lacerations due to this behaviour, on most days. He would wake around 3 a.m. with intense fear, palpitations and sweating. He would recall the content of the dreams and vividly described being pursued and attacked by large beasts, from whom he attempted to escape or fight back. Thereafter he failed

to initiate sleep again and would lie awake ruminating on the dream content. He usually started his day feeling unrefreshed, with a total sleep duration of about 4 hours.

There was no history of snoring, apnoeic attacks during sleep, night terrors, somnambulism or compulsive eating during partial arousals. There was mild daytime hypersomnolence but there was no daytime napping and no instances of falling asleep while driving, conversing or attending to activities at home. He did not experience sleep paralysis, hypnogogic/ hypnopompic hallucinations or sudden loss of muscle tone following an emotional precipitant.

His wife had started sleeping in a separate room seven years ago, subsequent to sustaining injuries after being pushed from the bed and grabbed by the neck, by the patient. Four years ago during a particularly violent dream the patient had jumped out of bed and fallen face down on the floor, and he had sustained a large laceration on his forehead. Following this incident he started to sleep in a room padded with mattresses.

Prior to the onset of these symptoms, he used to have a satisfactory sleep pattern, where he had restorative sleep for 6-7 hours per day. His sleep latency had been about fifteen minutes with no frequent awakenings at night. He was able to reinitiate sleep in less than a few minutes if he did awake at night. There was no dream enactment on the occasions when he did experience nightmares.

His weekly alcohol consumption did not exceed 2-3 units and there was no history of past alcohol abuse. There was no significant drug history. There were no features suggestive of parkinsonism or memory disturbances. He did not give a history of cerebrovascular accidents. There was no childhood history or family history of dream enactment, somnambulism, night terrors or epilepsy.

Consequent to his symptoms he was unable maintain a normal sleep environment and its associated interactions,

especially with his spouse. He was unable to sleep safely in any other environment apart from his customised bedroom, for fear of injury to himself and to others.

Mental state examination showed that he was alert and conscious. Multiple scars were seen on his forehead and all four limbs. There were no features of parkinsonism. His speech was coherent with normal prosody. The mood was euthymic and reactive. He worried about the dream content and risk of harming himself during sleep. There were no suicidal or homicidal ideas, no delusions or hallucinations. He was oriented with partial insight into his illness.

His extended cognitive functions were normal. The total score of the mini mental state examination total score was 29 out of 30, and he scored 28 out of 30 on the Montreal Cognitive Assessment (MoCA).

His physical examination was normal. Fasting blood glucose, lipid profile, full blood count, serum electrolytes, liver and renal profile were also normal.

The management involved explaining the diagnosis, advice on modifying sleep environment, discussion of sleep hygiene measures and stress management strategies. He did not respond to two weeks of melatonin 5mg at night, which was then increased to 10mg. The response remained unsatisfactory and thereafter he was switched to clonazepam 2mg nocte. Two weeks after commencement of clonazepam there was a remarkable improvement in sleep quality, sleep duration and the violent content of the dreams. The patient is currently maintained on clonazepam 2mg at night.

## Discussion

In a presentation of violent dreams with dream enactment, while considering RBD, a differential of somnambulism, sleep terrors, dissociative disorders, nocturnal seizures and periodic limb movements should also be entertained. Violent or injurious behaviour during sleep, sleep behaviour disrupting sleep continuity and polysomnographic abnormality during REM sleep (elevated submental electromyographic tone and/or excessive phasic submental and/or limb electromyographic twitching and absence of EEG epileptiform activity during REM sleep) supports the diagnosis of RBD (1).

Polysomnographic video recording is the single most important diagnostic test in persons with RBD. Polysomnography was not feasible in this patient due to the potential risk to equipment caused by the patient violently acting out dreams. Neuroimaging is not routinely indicated in persons who have no neurologic cause of RBD. Imaging studies should be considered in patients younger than 40 years of age, where there is no known precipitant cause such as alcohol or medication use (5, 6). Consequentially this patient's diagnosis of REM sleep parasomnia, and the exclusion of other

differentials was based solely on the history, mental state and physical examination. Neuroimaging was not indicated given his clinical picture.

To date, there are no large randomised controlled trials of treatments for RBD. Small case series and case reports describe efficacy of a wide range of medications, most prominently clonazepam but also melatonin, pramipexole, acetylcholinesterase inhibitors, paroxetine, L-DOPA, alprazolam, carbamazepine, clozapine and sodium oxybate (7, 8). Clonazepam is highly effective in almost 90% of patients with RBD and demonstrates a complete benefit in 79% of patients (7). There is little evidence of tolerance or abuse of the drug. Symptoms relapse promptly on discontinuation of medications in almost all patients; therefore, pharmacologic treatment should be continued indefinitely (7). Clonazepam is thought to decrease the occurrence of sleep related injury caused by RBD.

RBD remains on the fringe of routine clinical evaluation and as such is possibly underdiagnosed. It is not unfair therefore to suggest that it be given more prominent consideration when evaluating patients with sleep disorders, as the symptomatology is acutely distressing and disabling while being eminently treatable.

Long term follow up of patients diagnosed with RBD is essential as it heralds many neurodegenerative disorders such as Parkinson's disease, multiple system atrophy and dementia with Lewy bodies. This would facilitate the implementation of neuroprotective therapies in at-risk individuals.

## Consent

Written informed consent was obtained from the patient for publication of this case report.

## Declaration of interest

None declared

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