In Your Dreams – A Case of Presumed Rapid Eye Movement Sleep Behavior Disorder in the Inpatient Psychiatric Unit

Michelle B. Collier¹*, Stephanie D. Nichols²³ and John J. Campbell⁶

1Tufts University School of Medicine, Boston, Massachusetts, USA
2Husson University School of Pharmacy, Bangor, Maine, USA
3Maine Medical Center, Tufts University School of Medicine, Portland, Maine, USA

*Corresponding author: Michelle B. Collier, B.S., Tufts University School of Medicine, 145 Harrison Ave., Boston, MA 02111, Massachusetts, USA, Tel: (617) 636-6534, E-mail: michelle.collier@tufts.edu

Unlike dyssomnias that influence quality and duration of sleep, parasomnias primarily affect behavior [1]. Rapid eye movement (REM) sleep behavior disorder (RBD) is a parasomnia characterized by loss of normal skeletal muscle atonia during REM sleep [1-3]. Usually, atonia occurs through neural inhibition via pontine nuclei to spinal motor neurons [2]. Dysfunction, due to lesions or neurodegeneration, can lead to dream enactment. Therefore, sleepers may act violently, including: hitting, jumping, or kicking [2].

Those who have neurodegenerative diseases may also simultaneously suffer from psychiatric illnesses, such as schizophrenia [3,4]. We report the unusual case of a patient who presented with bizarre delusions, difficulty differentiating dreams from reality, and presumed RBD in the context of schizophreniform disorder. His psychosis was quickly treated to remission. The initial treatment with ramelteon did not improve the patient’s presumed RBD symptoms, thus melatonin was started and proved successful.

Case Report

A 33 year-old Caucasian male with a psychiatric history significant for anxiety and depression, was admitted after jumping from a 3rd story window and sustaining a spine injury. He reported jumping immediately after awakening from a dream in which he feared that a Hitler-like entity would gas him in his apartment. Despite appreciating that these were nightmares, he developed the belief that he was actually being pursued by Nazi-like phantoms for almost 2 years. He denied auditory hallucinations of voices, but reported hearing the “whoosh” of gas and smelling its odor. No risk factors for seizures or head trauma were present when the olfactory hallucinations occurred, prior to jumping from the window. Neuroimaging was within normal limits. Also, he denied tingling, numbing, weakness, tremor/parkinsonism’s, or other abnormalities.

Previous outpatient medication trials of fluoxetine and bupropion were unsuccessful. Fluoxetine was ineffective and bupropion caused visual hallucinations such as the patient seeing a sign that read, “kill Nana” which prompted the patient to attempt to attack his grandmother with a kitchen knife. He was taking sertraline and olanzapine at the time of admission, after jumping from the window.

While hospitalized, the patient continued to have psychotic symptoms and was diagnosed with schizophreniform disorder. He also exhibited dream enactment behavior and somnambulism, and received the diagnosis of suspected RBD. The patient had difficulty differentiating dreams from reality and this contributed to his disorientation in the wake state. It was unclear how long the patient experienced parasomnias. A waking EEG showed no epileptiform activity however, a video-polysomnography could not be obtained.

He was initially treated with risperidone and his delusions resolved completely but the RBD symptoms persisted. Addition of ramelteon 8 mg nightly improved, but did not relieve, the symptoms of RBD. On one occasion, while taking ramelteon, he was facing the window making dodging movements, reporting dreaming that he was a fighter pilot in a “dogfight”. On another occasion, he stood on his bed, and after being awakened and cleared, he reported preparing for a luge run. While fully awake, his behavior was completely normal. Ramelteon was discontinued and melatonin 6 mg was administered nightly at bedtime. Melatonin was well tolerated and resulted in resolution of presumed sleep-related behavioral disturbances. Overall, his psychiatric symptoms and confusion upon awaking have improved.

Discussion

While we do not have video-polysomnography confirmation of RBD in this case, his behaviors are suspicious for the disorder and he responded to evidence-based treatments. First line therapy for RBD includes clonazepam and melatonin [5]. Ramelteon is an FDA-approved melatonin receptor agonist with a longer half-life than melatonin itself. Very limited data may support ramelteon’s use in the treatment of RBD, including a report of 2 successful cases and an open-label pilot study of 10 patients, however robust evidence is lacking [6-8].

As illustrated in our patient, there may be differences between melatonin and ramelteon in RBD treatment. Melatonin receptors can be sub classified as MT1, MT2, or MT3 (Table 1). Both melatonin and
ramelteon agonize MT<sub>1</sub> and MT<sub>2</sub> receptors, although ramelteon has 6- and 3-fold higher affinities for MT<sub>1</sub> and MT<sub>2</sub>, respectively, versus melatonin [9]. Further, only melatonin binds to MT<sub>3</sub> receptors. Differences in the binding affinities and selectivity may explain why our patient only fully responded to melatonin [10].

Presumed RBD in a 33 year old male is an atypical presentation since RBD usually presents in males over 50 years old. Medications may have been contributors in this case. For example, SSRIs, TCAs, and venlafaxine can provoke RBD. Therefore, sertraline may have contributed to the emergence of presumed RBD in this patient [11]. Additionally, risperidone has been shown to significantly reduce REM sleep versus placebo [12].

Our case represents the second published case of (presumed) RBD presenting to a psychiatric unit, but our patient’s case relates to psychosis and the first case did not [13].

By adequately treating presumed RBD with melatonin, in the context of schizophrenia, our patient was discharged to a group home, functioning to both support his independence and increase his quality of life.

References

1. (2014) International Classification of Sleep Disorders (3rd ed), American Academy of Sleep Medicine, Darien, IL.

Table 1: Difference in binding affinities of MT<sub>1</sub>, MT<sub>2</sub> or MT<sub>3</sub> receptors.

<table>
<thead>
<tr>
<th>Function</th>
<th>Ramelteon Affinity</th>
<th>Melatonin Affinity</th>
</tr>
</thead>
<tbody>
<tr>
<td>MT&lt;sub&gt;1&lt;/sub&gt; (humans) Initiation of sleep</td>
<td>Ki = 14</td>
<td>Ki = 80</td>
</tr>
<tr>
<td>MT&lt;sub&gt;2&lt;/sub&gt; (humans) Regulation and maintenance of circadian rhythm</td>
<td>Ki = 112</td>
<td>Ki = 383</td>
</tr>
<tr>
<td>MT&lt;sub&gt;1&lt;/sub&gt;/MT&lt;sub&gt;2&lt;/sub&gt; ratio Lower number identifies greater selectivity for MT&lt;sub&gt;1&lt;/sub&gt; over MT&lt;sub&gt;2&lt;/sub&gt;</td>
<td>0.13</td>
<td>0.21</td>
</tr>
<tr>
<td>MT&lt;sub&gt;3&lt;/sub&gt; (hamsters) Modulates the enzyme: Quinone reductase 2</td>
<td>Ki = 2650</td>
<td>Ki = 24</td>
</tr>
</tbody>
</table>

Table 1: Difference in binding affinities of MT<sub>1</sub>, MT<sub>2</sub> or MT<sub>3</sub> receptors.