

# REM Behavior Disorder (RBD) and Body Rolling in a Chronic Alcoholic Patient: Just a Coincidence or an Inherent Predisposition Precipitated by Alcohol?

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## Case Report

A 65 years old patient was referred to our sleep center due to the recent appearance of nocturnal episodes characterized by shouting, arms and legs movements mimicking the dreams. He also reported difficulty to initiate sleep and excessive diurnal sleepiness (Epworth Sleepiness Scale, ESS score 8, normal value <10). His BMI was 29.6 Kg/m<sup>2</sup>. The patient was adopted and he did not know any information about his familial history and his childhood. He was a chronic alcoholic. He had suffered from anxiety and depression for 15 years and was treated with escitalopram 20 mg/die, trazodone 150 mg/die and alprazolam according to necessity. Neurological examination showed postural tremor of both arms, left hemypoesthesia and right Babinski. The patient was submitted to a neuropsychological evaluation. His scores were 26/30 on Mini Mental State Examination (MMSE), 15 on Pittsburgh Sleep Quality Index (PSQI) (>5 bad sleep quality), 26 on Hamilton Anxiety Rate Scale (HARS: 14-17 mild anxiety; 18-24 moderate anxiety; 25-30 severe anxiety), 27 on Beck Depression Index (BDI, >10 mood disorders); he also underwent the SF-362v questionnaire to evaluate quality of life: Mental Component Score (MCS) 59.23 and Physical Component Score (PCS) 25.00, showing a bad quality of life.

He obtained a brain CT showing chronic ischemic alterations of subcortical white matter and a video Polisomnography (video-PSG) including 8 leads EEG. Video-PSG detected parameters included: monopolar electroencephalogram, electro-oculogram, electromyogram of chin and tibialis anterior muscles, airflow, thoracic and abdominal efforts, snoring, electrocardiogram, body position, and oximoglobin saturation. The PSG recordings were scored according the second edition of International Coding of Sleep Disorders (ICSD-2, 2005) rules [1].

Video-PSG unexpectedly revealed rhythmic movements of the trunk, the head and body (300 episodes) predominantly distributed at sleep onset and upon arousals from different sleep stages including REM sleep. These movements lasted from 5 to 60 seconds. The patient had never reported these symptoms/events.

The EEG montage showed no ictal or interictal abnormal discharge, excluding the presence of paroxysmal events.

Sleep pattern was discontinuous and superficial with an arousal index of 24.5. Apnea/Hypopnea Index (AHI) was 12.9 (normal value <5). No periodic or a periodic leg movements were revealed during the night, neither bruxism.

His REM sleep, albeit short, showed sustained loss of muscle atonia (REM-without atonia), but no dream-enacting episodes were observed during the night.

Therefore Clonazepam 1 mg before bedtime was prescribed

along with reduction and/or suspension of alcohol intake. Results were unfortunately poor. The patient continues to drink, assumes Clonazepam with reduction of nocturnal episodes of abnormal behavior, but persistence of rhythmic movements referred by his wife.

## Discussion

Manni and Terzaghi [2] reported 2 patients diagnosed with RBD, presenting rhythmic head rolling in the context of dream enacting or loss of muscle atonia episodes, respectively. They suggested a potential link between RBD and Sleep Related Rhythm Movement Disorder (SRRMD), involving the Central Pattern Generator (CPG) neuronal networks.

A patient suffering from SRRMD since childhood newly diagnosed with RBD was described by Xu et al. [3] in 2009.

Our patient probably had Rhythm Movement Disorder (RMD) since childhood without knowing it with persistence into adulthood. Dream-enacted episodes subsequently appeared, probably triggered by alcohol addiction [4]. The head and body rolling occurred at sleep onset and during NREM and REM sleep. When in REM sleep, they preceded the loss of muscle atonia. Could the two sleep complaints have a link also in our case?

The presence of two sleep related movement disorders in the same patient could be however coincidental since RBD in this case is most likely due to alcohol abuse whereas SRRMD most likely persisted from childhood. Indeed the improvement of RBD episodes on Clonazepam (CNZ) without amelioration of SRRMD may suggest two different underlying mechanisms.

Our case is more similar to the one described by Xu et al. [3] even if RBD is clearly secondary to binge drinking rather than idiopathic as in the case previously described. Whether SRRMD may represent a liability factor for the subsequent development of RBD, remains to be explored via a more careful anamnestic and V-PSG longitudinal evaluation of patients with movement related parasomnias.

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