

The Effectiveness of Melatonin in Head Banging: A case report

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ABSTRACT

Head banging is one of the three typical subtypes of rhythmic movement disorder. It typically involves anteroposterior movements of the head. Rhythmic movement disorder usually resolves in early childhood but less commonly persists into adolescence and adulthood. Although benzodiazepines commonly used, the universal effectiveness of any pharmacologic agent has not been approved. Herein, we present an 8-year-old girl patient suffered from head banging who responded to melatonin after in failure to imipramine treatment. Although nocomplete remission has been obtained, this is the first melatonin trial in a child patient with headbanging.

Keywords: Sleep, Melatonin, Child.

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INTRODUCTION

Rhythmic, stereotyped, nonepileptic, large-amplitude body movements may occur shortly before sleep onset and persist into sleep in some cases¹. When the movements significantly interfere with normal sleep/daytime function or result in self-harm/injury, they are classified as rhythmic movement disorder (RMD). Head banging is one of the three typical subtypes of RMD which involves anteroposterior movements of the head, most commonly onto the pillow or mattress^{2,3}. RMD is most frequent in light NREM sleep but may sometimes be seen during REM sleep⁴. It usually resolves in early childhood but less commonly persists into adolescence and adulthood^{3,5}. Most of the RMD cases do not need pharmacological treatment. However, in the circumstances of poor sleep or self-inflicted bodily injuries should be treated^{5,6}. In the literature; mostly used treatment strategy was benzodiazepines. The therapeutic effect of imipramine has been shown in head banging^{7,8}. Other treatments include antidepressants, behavioral interventions, hypnosis, and sleep restriction⁶. Herein, we present an 8-year-old girl patient suffered from head banging who responded to melatonin after in failure to imipramine treatment.

CASE REPORT

An 8-year-old girl was referred to our child psychiatry clinic with head banging during nocturnal sleep. Her parents first noticed it when she was 18 months of age. The patient was never aware of these events, but her parents particularly worried about whether brain injury would result from the head banging. Since the nose-bleeding occurs in which the consequence of head banging, parents have begun to sleep with their daughter. The patient's medical history indicated that prenatal history was significant for abortus risk and oligohydramnios. She was born as a full-term infant with the low birth weight. There was not postnatal period complication. She had normal developmental stages. Her past psychiatric history included psychostimulant treatment in the preschool years (of unknown dose and duration). Family history revealed epilepsy and major depressive disorder in the patient's father. According to her parents, there was no family history of nocturnal head banging or other parasomnias.

Her first evaluation at the age of 33 months by the pediatric neurologist showed the diagnosis of nocturnal epilepsy and, oxcarbazepine started. Parents reported that she had continued this treatment until six-year-old with no benefit. At the age of six, she was evaluated by another clinician. This evaluation included brain MRI (magnetic resonance imaging) and EEG (electroencephalography) (while awake and sleep), their results showed structurally and electrically normal brain. The clinician ruled out neurological disorder but did not address head banging to be related to any sleep problem. Eventually, the non-beneficial anti-epileptic treatment ceased, and the patient was referred to our center for a psychiatric evaluation.

At the initial visit, a detailed anamnesis of head banging episodes recorded. These episodes usually emerged in the

early morning (from 05:00 to 08:00 h) or in the midnight (from 23:00 to 02:00 h) and, nearly several times at night. The patient considered her sleep to be normal and, she denied any daytime sleepiness. The subjective reports of the patient and her parents revealed complaints including attention difficulties, nocturnal enuresis, daytime sleepiness and, tiredness. They reported that if she was awakened by parents twice a night, enuresis did not occur.

In the past, she was evaluated for enuresis, and any organic etiology had not been found. There were not any symptoms that suggestive of other sleep pathology. Physical, neurological and laboratory examinations showed no apparent abnormalities. Although her academic achievement is within the normal ranges, WISC-R (Wechsler Intelligence Scale for Children-Revised) results (total IQ score; 80) indicated low average cognitive abilities (total IQ score from 80 to 89). The total PSQ (Pittsburgh Sleep Questionnaire) score was 5, and total sleep problems score was 3 points. The Turkish reliability and validity study has shown that PSQ has the power to distinguish the healthy children and ADHD patients from the children with upper airway resistance and snoring. Researchers have suggested that total PSQ score (6 points as a cut-off) could be discriminative for primary sleep disorder in the healthy group, total sleep problems score (5 points as a cut-off) could be discriminative for main sleep problems in the ADHD group⁹.

According to these findings, our patient's PSQ results did not support the upper airway resistance and snoring. The Epworth Sleepiness Scale was not available; therefore, daytime sleepiness symptoms could not be systematically assessed. Unfortunately, we could not be able to PSG because our hospital had no child sleep laboratory facility. Firstly, 25 mg/d imipramine was started. At the end of the first month, any response to imipramine was not obtained. Then, melatonin treatment (3mg/day) was started, and the dose gradually was increased up to 9 mg (within one month). On the third month of melatonin treatment (9mg/d, 30 min before asleep), the frequency of head banging episodes reduced from nearly every night to two-three nights per week and, the number of events observed on these nights also decreased (from the 4-5 times to the 1-2 times). Both the total PSQ and the total scores of sleep problems were decreased by one point. However, the patient did not achieve complete remission. Atomoxetine treatment initiated with the diagnosis of ADHD-Inattentive subtype at the fourth month of follow-up. The patient's caregiver provides informed consent allowing for the use of her clinical data in the research.

DISCUSSION

This case reported herein portrays a girl with RMD who showed a therapeutic response to melatonin. The clinical diagnosis of RMD was performed from the clinical history. Parental descriptions of the many nocturnal behaviors, ranging from seizure to behavioral problems, make the diagnosis may be hard¹⁰. In the presented patient, head banging occurred in the early morning or one to three hours after asleep. Reports on the

sleep stages in which episodes of RMD occur are not conclusive. These movements occurred during various stages of sleep (REM or NREM) and sleep-wake transitions⁶. Unfortunately, we could not be able to perform PSG (polysomnography) because our hospital had no child sleep laboratory facility. Therefore, we are not able to discuss the relationship between the sleep stages and head banging episodes. However, some authors stated that PSG is applied when the clinical history alone is insufficient to diagnose or atypical or particularly violent movements exist³.

The rare clinical presentation confused clinicians, indicating how head banging may incline the clinicians to misdiagnose as a nocturnal behavior problem or a seizure disorder¹¹. Additionally, the diagnostic challenges could result in unnecessary antiepileptic treatment as seen in our patient. Her nocturnal epilepsy diagnosis had caused unsuccessful oxcarbazepine medication for long years. The absence of epileptic abnormalities and present episodes like tongue biting, urinary incontinence, tonic-clonic activity, complex motor automatisms, or other activity resembling seizure are also helpful for us to exclude epilepsy.

Rhythmic movements are also seen in some childhood psychiatric disorders such as autism spectrum disorders, and mental retardation. In these patients, an additional diagnosis of RMD should be made if only the movements are predominantly sleep related⁶. In the presented case, RMD persisted beyond early childhood and she was also diagnosed with ADHD. The association between RMD and psychopathologies when RMD were persisting in older childhood or beyond are controversial⁶. Stepanova et al. (2005) reported that subjects with RMD have symptoms persisting after five years old showed significant ADHD comorbidity. However, the underlying mechanism of this coexistence is not known¹².

Recent findings indicate that melatonin may modulate dopaminergic pathways involved in movement disorders¹³. Melatonin and dopaminergic system interactions have been supported by findings that showed low endogenous melatonin levels in drug-naive patients with schizophrenia and the beneficial effects of high dose melatonin (10 mg/day) in patients with tardive dyskinesia (TD)^{14,15}. The dopaminergic modulating function of melatonin and its antioxidant properties have been suggested potential mechanisms for clinical efficacy in TD¹³. The interaction of melatonin with the dopaminergic system may play a significant role in the nonphotic and photic entrainment of the biological clock as well as in the fine-tuning of motor coordination in the striatum¹³. We suggest that the treatment response to melatonin should be taken into consideration with ADHD comorbidity, since it might reflect different presentation of dopaminergic dysfunctions in both ADHD and RMD.

In most of the cases, pharmacological treatment is unnecessary. Benzodiazepines usually have been used in the treatment of RMD⁶. The universal effectiveness of any pharmacologic agent has not been approved. Although the imipramine was not effective on our patient, the treatment response to imipramine has been shown in head banging cases^{7,8}. In the present

case, the frequency of head banging episodes was reduced with 9 mg/d melatonin at the third month. Melatonin, as a chronobiotic, has been suggested to increase the integrity of the circadian timing system, re-entraining short-term dissociated or long-term desynchronized circadian rhythms^{16,17}. It also affects the locomotor activity during the sleep^{17,18}. The plasma melatonin profile displays an inter-subject heterogeneity¹⁹. The melatonin secretion occurs at night, with maximum plasma levels around 03:00–04:00 a.m., varying with chronotype¹⁹.

In the present case, melatonin treatment could play a role in the regulation of endogen melatonin level. Her episodes which occur three hours after sleep began might be reflection of possible endogenous melatonin dysregulation. Although the consequences of low melatonin secretion on vulnerability to rhythmic organization problems are not well-known¹⁹, some studies indicated melatonin effects including enhancing the rest-activity rhythm in self-reported sleep-wake disturbances, normalized the sleep quantity and quality in REM sleep behavior disorder, effective in periodic limbic movement disorders^{17,18}. Reducing motor activity during sleep and preserving atony related to REM sleep are suggested mechanisms for melatonin^{17,18}. The melatonin treatment was found ineffective on *jactatio corporis* in an adult with dissociative disorder. However, this patient concurrently used melatonin with two other drugs (clonazepam and paroxetine)²⁰.

To our knowledge, the present case is the first report of head banging responded to short-term melatonin treatment. The exogen melatonin might be effective by the stabilization of the circadian timing system, sleep pattern and also locomotor activity. Although complete remission was not achieved, our finding is encouraging to some degree. However, several limitations of this case report require acknowledgment. First, the lack of the PSG evaluation that the gold standard for documentation of the nature of these nocturnal events was the main limitation. Second, the short-term use of melatonin does not allow us to speculate on whether melatonin can reverse RMD. The question remains unanswered on what if the melatonin would be removed and reintroduced would the therapeutic impact be the same or not.

Clarifying the therapeutic effect, it also could be useful. In the presented case, subsequently having started treatment with atomoxetine was also another handicap. While the present case report has certain limitations, melatonin which has relatively benign side-effect profile might be a useful alternative to other treatment regimens (e.g., benzodiazepines) in the treatment of RMD. Future research that would be done with different dose regimens, and also long-term use, might be helpful to understand the melatonin effect on RMD.

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