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Sleep-related eating disorder associated with zolpidem: cases compiled from a literature review

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ABSTRACT

Objective: Zolpidem is associated with sleep-related eating disorder (SRED). We compiled case reports and performed a descriptive study to identify etiology and aggravating factors.
Methods: A literature search on PubMed's MeSH search feature, CINAHL, and SciFinder was performed using search terms "Zolpidem," "Feeding and Eating Disorders/chemically induced," "Dyssomnias," "sleep eating disorder," and "sleep-related eating disorder." Three reviewers examined all English and Spanish citations and extracted pertinent information. A narrative synthesis of the evidence was prepared.
Results: We identified 40 case reports of which 65% were female, and the mean age was 53 years. SRED onset was most commonly seen with daily zolpidem doses of 10 mg or higher (95% of patients). Prior medical history included obstructive sleep apnea (OSA) (35%), depression (32.5%), and restless leg syndrome (RLS) (25%). Even with controlled RLS and OSA, SRED developed in some patients. All patients had either partial or full amnesia with compulsive eating. Onset of SRED occurred as early as the first dose to after 9 years of use. SRED symptoms occurred nightly in 57.5% of patients. Discontinuation of zolpidem resolved SRED in all patients (n = 36).
Conclusion: SRED associated with zolpidem can occur with any dose, but was most common with higher doses of zolpidem. Therefore, prescribers should initiate lower doses of zolpidem. Interestingly, many patients had underlying disorders known to affect sleep (RLS, OSA, depression). Although it is recommended to control these underlying disorders prior to initiating zolpidem, SRED may still occur. Zolpidem discontinuation resolved all cases of SRED.

1. Background

Sleep-related eating disorder (SRED) is defined as recurrent episodes of involuntary eating and drinking during the main sleep period [1]. SRED is often problematic due to weight gain from involuntary eating and drinking and due to daytime fatigue from disrupted sleep. Currently, the DSM V does not include SRED [2].

While the DSM-5 does not include SRED, in 2014 the International Classification of Sleep Disorders-Third Edition (ICSD-3) included SRED as an arousal disorder during non-rapid eye movement (NREM) sleep [1]. To diagnose SRED, the following four criteria must be met.

ICSD-3 Diagnostic Criteria for Sleep-Related Eating Disorder (SRED)

1) Recurrent episodes of dysfunctional eating that occur after an arousal during the main sleep period.
2) The presence of at least one of the following in association with the recurrent episodes of involuntary eating: (A) consumption of peculiar forms or combinations of food or inedible or toxic substances, (B) sleep-related injurious or potentially injurious behaviors performed while in pursuit of food or while cooking food, or (C) adverse health consequences from recurrent nocturnal eating.
3) There is partial or complete loss of conscious awareness during the eating episode, with subsequent impaired recall.
4) The disturbance is not better explained by another sleep disorder, mental disorder, medical disorder, medication, or substance use.

In a 1991 study by Schenck et al., SRED was observed for the first time in a sleep disorder clinic [3]. Prior to Schenck's work, SRED had

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been described as “night eating syndrome”, defined as frequently eating at night after waking from sleep or excessively consuming food after dinner. However, amnesia does not occur with night eating syndrome as it does with SRED. Furthermore, SRED may resemble somnambulism (sleepwalking); however, eating is not a common component with the latter. Therefore, while SRED has similarities to night eating syndrome and somnambulism, these disorders do not involve involuntary eating during the main sleep period.

There could be a genetic component associated with SRED. Approximately 20% of the patients, in a study done by Winkelman, had a first-degree relative with SRED [4]. In addition, there has been a case of fraternal twins presenting with SRED. However, no specific genetic link has been found [5].

Concurrent use of psychotropic medications and presence of SRED is also documented. Psychotropic medications with sedative properties may contribute to the development of SRED by further impairing the arousal level. Use of hypnotic medications was found to be a significant predisposing factor for the development of SRED-like behavior ($p < 0.01$) in one study [6]. There have been associations of SRED with triazolam, amitriptyline, olanzapine, risperidone, and zolpidem [7–10].

Zolpidem, a nonbenzodiazepine sedative-hypnotic, is a favorable treatment option for insomnia due to its short onset, short half-life, and low abuse potential. Zolpidem has shown agonist selectivity towards alpha-1 subunits in GABA-A receptors, which plays a vital role in sedation [11]. However, binding to the alpha-1 receptor decreases the power of NREM, which does not simulate natural sleep. Furthermore, zolpidem has less REM suppression than other benzodiazepines, decreases time to achieve NREM sleep, and prolongs the duration NREM sleep [12]. Therefore, NREM sleep produced by zolpidem is suboptimal [13].

During optimal sleep, consciousness quickly emerges when transitioning from light NREM to wakefulness. This transition may be impacted by many factors including: depth of NREM sleep, circadian rhythm phases, genetic or environmental factors, effects of sedative medications, and conditions associated with repeated, partial arousals during NREM sleep [14]. While the specific etiology of SRED during zolpidem use is unclear, it is known that SRED is distributed across all stages of NREM sleep [15]. Dysfunction of the transition between NREM to wakefulness is associated with SRED due to deepened NREM sleep (as documented with amnesia) with impaired awakening (impaired walking and eating).

Zolpidem is a commonly used insomnia medication with reports of SRED. Furthermore, SRED is a relatively new psychiatric disorder that has not yet been defined in DSM-V. In this case series review, we compile all published case reports of zolpidem-associated SRED to help identify SRED etiology and aggravating factors.

2. Methods

This case series presents zolpidem use and its association with sleep-related eating disorder. This review of the literature included a search of PubMed, CINAHL, and SciFinder for all English and Spanish language case studies on sleep-related eating disorders associated with zolpidem use published through September 11, 2019. The following terms were searched, as MeSH terms or keywords as appropriate: “Zolpidem,” “Feeding and Eating Disorders/chemically induced,” “Dyssomnias,” “sleep eating disorder,” and “sleep-related eating disorder.” From this search, 18 relevant articles, collectively identifying 41 individual case reports. Out of these identified cases, we selected 40 cases featuring patients with provider diagnosed cases of zolpidem-induced cases of SRED. All patients had recurrent episodes of amnestic eating during the main sleep period attributed to zolpidem (consistent with ISCD-3 diagnostic criteria for SRED). We excluded one case where the patient had compulsive evening eating behavior not associated with sleep disturbance [16].

We extracted several components of the case reports - including age, gender, ethnicity, past medical history, concomitant medications, zolpidem dose, level of amnesia (total, partial, or unknown), treatment modification, time to SRED onset, and time to SRED resolution. Patients reporting both total and partial amnesia were classified as having total amnesia. In cases where amnesia was present, however, the level of amnesia was not reported (ie, total or partial amnesia), patients were classified as having an unknown level of amnesia.

3. Results

Of the 40 subjects, 65% were female (Table 1). Prior medical history included 35% with obstructive sleep apnea (OSA), 32.5% with depression, and 25% with restless leg syndrome (RLS). To note, 5% of patients had prior medical history of sleepwalking and no patients had prior history of SRED. SSRIs were the most commonly used concomitant medication (37.5%), followed by benzodiazepine (20%), and dopamine agonists (12.5%).

Also, we evaluated the zolpidem dosage at SRED onset. There were only two cases of patients experiencing SRED with low doses of zolpidem, 5 mg and 6.25 mg. The most common daily dosage of

Table 1
Baseline characteristics.

	N = 40
Age	
Mean ± sd	53 ± 14.4
Gender	
Female	26 (65%)
Race	
White, n (%)	13 (32.5%)
Asian, n (%)	3 (7.5%)
African American, n (%)	1 (2.5%)
Missing, n (%)	23 (57.5%)
Select Medical History	
OSA	14 (35%)
Depression	13 (32.5%)
RLS	10 (25%)
Concomitant Psychiatric Medication Use	
SSRI	15 (37.5%)
Benzodiazepine	8 (20%)
Other Medications	
Dopamine agonist	5 (12.5%)
Zolpidem Total Daily Dosage [a]	
5 mg	1 (2.5%)
6.25 mg	1 (2.5%)
10 mg	30 (75%)
12.5 mg	4 (10%)
15 mg	1 (2.5%)
20 mg	2 (5%)
30 mg	1 (2.5%)
SRED Frequency	
Nightly	23 (57.5%)
Weekly	5 (12.5%)
Monthly	3 (7.5%)
Yearly	4 (10%)
Unknown	5 (12.5%)
Amnesia of episodes	
Total	22 (55%)
Partial	8 (20%)
Unknown Level of Amnesia	10 (25%)

OSA = obstructive sleep apnea.
RLS = restless leg syndrome.
SSRI = selective serotonin receptor inhibitor.
[a] If the patient was prescribed a range, we report the maximum dosage allowed.

zolpidem at SRED onset was 10 mg (75% of patients) [8–10,17–22]. This dosage is currently the highest recommended dose for immediate-release (IR) zolpidem. The next common dosage was 12.5 mg (9.8% of patients), which is currently the highest recommended dose for extended-release (ER) zolpidem. SRED also occurred with zolpidem doses ranging from 15 to 30 mg; however, these high doses are not currently recommended. Therefore, patients were typically on high doses of zolpidem when they experienced SRED.

As for SRED onset, one patient experienced SRED after their first zolpidem dose and the longest onset occurred after 9 years of zolpidem use [9,10]. SRED frequently occurred nightly (57.5%), but also infrequently appeared weekly, monthly, or even yearly (Table 1). Therefore, zolpidem-associated SRED may occur at any point during zolpidem use with many patients experiencing nightly symptoms.

SRED features included nighttime eating and amnesia with descriptions of episodes varying from patient to patient. Some patients found packages of snacks when they woke up the next day and some patients were seen cooking more complex meals [9,10,22,23]. Twenty-two patients reported total amnesia, and 8 patients reported partial amnesia of SRED episodes. The remaining 10 patients were amnestic during episodes, but the level of amnesia were not well-defined. Overall, patients presented with out-of-control eating with partial/full amnesia.

Treatment of SRED ranged from zolpidem discontinuation to zolpidem dose reduction (Table 2). This led to SRED resolution in 97.5% of patients (39 of 40 patients). Zolpidem discontinuation resulted in a complete resolution of SRED in 36 of 36 (100%) patients. In one case of SRED, adding topiramate to zolpidem did not lead to SRED resolution; however, discontinuing zolpidem led to the resolution of symptoms [19]. Zolpidem dosage was decreased in four cases, and 3 of the 4 patients showed a resolution of SRED. In the last patient, reduction of zolpidem dose resulted in a decrease in monthly SRED episodes [22,24]. The decrease of SRED with dose reduction implies a dose dependent effect of zolpidem.

In many cases, patients were switched from zolpidem to other insomnia medications. In four cases, zolpidem was replaced with clonazepam, in one case, it was replaced with trazodone, and in another case, it was replaced with lormetazepam [10,18,22,25]. It wasn't reported whether medication change treated insomnia, but zolpidem discontinuation resolved SRED.

Finally, in 2 patients with a prior history of sleepwalking, SRED onset occurred soon after zolpidem initiation. Furthermore, discontinuation of zolpidem resolved SRED in both cases [8].

4. Discussion

In previous studies by Winkelman and Schenck, 65% of SRED occurred in young female Caucasians [3,4]. Among these patients, SRED was associated with either complete or partial amnesia. In our case series, common characteristics of SRED, including female gender, race, and amnesia, were consistent with previous studies [3,4,26]. Furthermore, a published abstract of 19 patients found those most vulnerable for developing de novo zolpidem-induced SRED included females with insomnia (84%), zolpidem doses of mostly 10–20 mg, and concurrent use of antidepressants or other psychiatric medications (89.5%) [26]. In our case compilation, we similarly identified predominantly females (65%), zolpidem doses of 10–30 mg (95%), and a high percentage of SSRI or benzodiazepine use (57.5%).

SRED may be more common in females due to increased zolpidem prescribing in this gender and decreased zolpidem metabolism. Compared to men, women have a 40% increased risk of developing insomnia, resulting in more women being initiated on zolpidem [27]. Additionally, studies show women metabolize zolpidem 50% slower than men [28]. In 2013, the FDA reduced the recommended zolpidem dose in females by half for all formulations due to impaired alertness the morning after taking zolpidem [29].

The patient's age at SRED onset in our case series was vastly different from previous studies. Previous studies for idiopathic SRED have indicated that SRED typically occurs in the mid-20s [3,4]. Conversely, of our 40 case reports, the average patient age was much older at 53 years. This may be due to the increased zolpidem prescribing in older patients due to advancing age leading to decreased sleep quantity and quality [30].

Previous studies have linked SRED to history of parasomnias, obesity, family or personal history of eating disorders, genetic links, and environmental stressors [3,4]. Only two of our patients had a prior medical history of sleepwalking [8]. None had a previous eating disorder nor a genetic link. In patients without these risks, it may have been helpful to interview the patient to identify sources of stress. Stress from family and relationships, abstinence from alcohol/opiates/cocaine, and cessation of cigarette smoking have been previously shown to induce SRED [31]. Thus, interviewing patients presenting with SRED for stress may have helped identify additional risk factors for SRED.

Interestingly, 26 of our 40 patients with SRED had a history of obstructive sleep apnea (OSA), restless leg syndrome (RLS), and/or depression. These disorders can cause partial arousal during NREM sleep, which can exacerbate zolpidem-induced poor quality NREM sleep [14]. Though OSA is more common during REM sleep, elderly patients frequently experience OSA during NREM sleep [32]. Apneic episodes due to OSA have been shown to precipitate confusional arousals during sleep [33]. Similarly, RLS precipitates arousal due to periodic leg movements during sleep (PLMS). PLMS is more frequent during NREM sleep than REM sleep [34]. Finally, depression may also cause arousal during NREM sleep. Although depression is mainly characterized by increased REM, Germain, et al., concluded patients with depression had persistently elevated brain activity while transitioning from wakefulness to NREM sleep [35]. Most investigators theorize concomitant sleep disorders initiate partial arousal during NREM sleep, predisposing patients on zolpidem to SRED. While RLS and OSA may increase the risk for SRED, zolpidem use likely precipitated the SRED in our patients as discontinuation of zolpidem resolved SRED.

Treatment of SRED should first focus on identifying and resolving the precipitators of arousal from sleep. This may include discontinuing medications like zolpidem that decrease the quality of NREM sleep. In our study, discontinuation of zolpidem resulted in the resolution of SRED symptoms. Next, clinicians should identify and treat disease states that cause confusional arousals, especially RLS, OSA, and depression. Unfortunately, RLS can be misdiagnosed as insomnia. In 15 patients who were misdiagnosed with insomnia and inadvertently treated with benzodiazepines, 80% had a history of amnestic SRED or other sleepwalking behaviors. Treatment of RLS, a condition of dopamine deficiency, with dopaminergic agents

Table 2
Zolpidem modification and SRED symptoms after zolpidem modification.

Zolpidem modification	N (%)
Discontinued zolpidem	36 (90%)
Resolution of SRED	36 (100%)
Fewer SRED episodes per month	0 (0%)
No resolution of SRED symptoms	0 (0%)
Decreased zolpidem dose	4 (10%)
Resolution of SRED	3 (75%)
Fewer SRED episodes per month	1 (25%)
No resolution of SRED symptoms	0 (0%)

decreased SRED frequency by 75%–100% [36]. Thus, discontinuation of precipitating medications and control of these underlying disorders could be beneficial for the prevention and treatment of SRED [37].

If discontinuing medications and controlling disease states fragmenting sleep does not result in SRED resolution, pharmacological treatment should target the underlying sleep disorder. In our case series and previous studies, clonazepam was commonly used to treat SRED [10,22,25]. Monotherapy benzodiazepines, particularly clonazepam, has been historically successful in treating sleepwalking and had good results in idiopathic SRED patients with a history of sleepwalking [33]. This agent decreases slow–wave activity (SWA), thereby improving sleep disorders, which occur during deep NREM with high SWA [14,38]. However, in some cases, clonazepam has caused SRED [31,38]. A new option may include selective serotonin reuptake inhibitors (SSRI), which have been beneficial in patients presenting with idiopathic SRED and depression [39]. Recently, a randomized, controlled study with topiramate for idiopathic SRED demonstrated a reduction of SRED symptoms; however, this study excluded participants on medications thought to contribute to the presence of SRED [40]. While there are several pharmacological options to treat SRED, treating underlying disorders and discontinuing precipitating drugs, like zolpidem, should be considered first.

Due to the increased awareness of drug-induced parasomnias, the FDA is requiring a boxed warning of complex sleep behaviors on insomnia medications as of April 2019 [41]. With this boxed warning, a history of SRED is a contraindication to zolpidem. While serious adverse effects are rare, SRED may become life-threatening when unconsciously engaging in dangerous activities, resulting in serious injuries and death. Physicians should be advised to avoid zolpidem in patients with a family history of SRED. If zolpidem is the best option, FDA currently recommends low initial doses of zolpidem (5 mg for immediate-release and 6.25 mg for extended-release) [29]. In our case series, 95% of patients with SRED were on zolpidem 10 mg or higher. Higher doses of zolpidem should be taken with caution, especially in females due to the decreased elimination rate of the medication.

There are several strengths in this case series. It is the largest and most recent case series compiling all published zolpidem-induced SRED cases. As such, patients in this case series had a diverse medical history with many concomitant medications. SRED is a rare side effect which is not easily identified in randomized, clinical trials. This case series reports on SRED as it occurs in clinical practice, which may be more relevant and easily applied to routine care. Although this case series does not have internal validity because of the lack of competitor group, it does contain external validity due to a diverse range in patients reporting this side effect and lack of treatment interference.

Our study had potential limitations. First, there was a lack of a comparison group because all results were gathered from case reports. The retrospective observation of patient data limited the degree of inclusion, data collection, and patient follow-up. We were missing data points for prior history of eating or sleeping disorders, current stressors in patient's life, time to SRED onset, type of amnesia, and time to SRED resolution. Additionally, the use of zolpidem extended release or immediate release was not specified in the case reports; however, this was easily identified as doses for the formulations were different.

5. Conclusion

Our case series identified 40 published cases of zolpidem-induced SRED. From our results, zolpidem-induced SRED could occur with any dose, but was most common with high doses of zolpidem. Due to black box warnings, prescribers should ensure the patient does not have complex sleep behaviors before prescribing zolpidem. However, patients in our study were more likely to have underlying disorders known to affect sleep (RLS, OSA, depression) than a previous history of complex sleep behaviors. These underlying disorders should be controlled prior to consideration of zolpidem. Despite the control of these underlying disorders, SRED may still occur. If the patient does not have any contraindications, low dose zolpidem can be considered. Patients receiving zolpidem must be given the medication guide and should always be counseled on severe side effects like SRED. In our study, zolpidem discontinuation resolved all cases of SRED. Therefore, if symptoms of SRED develop while taking zolpidem, zolpidem should be discontinued, consideration given to potential underlying sleep pathologies, and referral of patients to a sleep disorder clinic.

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Tiffany Ho: Conceptualization, Methodology, Investigation, Data curation, Writing - original draft. **Alyssa Jimenez:** Conceptualization, Methodology, Investigation, Data curation, Writing - original draft. **Itzayana Sanchez:** Conceptualization, Methodology, Investigation, Data curation, Writing - original draft. **Christina Seeger:** Investigation, Writing - review & editing. **Merlyn Joseph:** Methodology, Formal analysis, Data curation, Writing - review & editing.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleepx.2020.100019.

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