Treatment of Nocturnal Eating Disorders

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Opinion statement

Identifying abnormal nocturnal eating is critically important for patient care and public health. Obesity is a global pandemic and a leading cause of preventable mortality in the United States, with more than 100,000 deaths annually. Normally, nighttime energy homeostasis is maintained, despite an absence of food intake, through appetite suppression and alterations in glucose metabolism that result in stable energy stores. Two conditions break this nighttime fast and are associated with weight gain as well as medical and neuropsychiatric comorbidities. Sleep-related eating disorder (SRED) is characterized by isolated nocturnal eating, whereas the night-eating syndrome (NES) is a circadian delay in meal timing leading to evening hyperphagia, nocturnal eating, and morning anorexia. Recently, SRED has been associated with the benzodiazepine receptor agonist zolpidem. Both SRED and NES are treatable and represent potentially reversible forms of obesity. In SRED, the antiseizure medication topiramate and dopaminergics have both demonstrated promising results. Nocturnal eating associated with NES has responded well to sertraline.

Introduction

The first descriptions of isolated nocturnal eating were noted in association with sleepwalking. In 1981, nocturnal eating while sleepwalking was reported in a 35-year-old woman with schizoaffective disorder [1, Class IV]. In 1991, a series of 19 patients with nocturnal eating was reported [2, Class IV] and subsequently followed in 1993 with a total of 38 patients [3, Class IV]. This condition was named *sleep-related eating disorder* (SRED) and classified as a parasomnia in the first edition of the International Classification of Sleep Disorders [4, Class IV].

The *night-eating syndrome* (NES) was described in 1955 as night eating with morning anorexia in a group of patients with treatment-resistant obesity [5, Class IV]. However, it was not until 1999 that the definition of NES was expanded to include nocturnal eating [6, Class III]. Recently, consensus criteria were established defining NES as consumption of at least 25% of intake after the evening meal, conscious nocturnal eating at least twice a week for 3 months, or both [7•, Class IV].

SRED is characterized by recurrent episodes of feeding after an arousal from nighttime sleep, with adverse consequences [8, Class IV]. Often patients claim involuntary eating, and cohabitants find them difficult to fully awaken, similar to sleepwalkers. Adverse consequences include weight gain, dangerous food preparation, and ingestion of toxic substances [3,9, Class IV]. The nocturnal food consumption in SRED is often high in calories. Specifically, many patients report bingeing on peanut butter, sugar-rich confectioneries, or chocolate [3,9, Class IV]. These findings are consistent with nocturnal eating among NES patients, who take in a higher proportion of carbohydrates at night (70%) than during the day (47%) [6, Class III]. Injuries sustained by SRED patients have included lacerations and burns from hazardous food preparation. In addition, inedible or toxic substances have been consumed, such as frozen raw meat, pet food, unshelled nuts, cigarettes, and cleaning products [3,9, Class IV]. Hunger has been notably absent during nocturnal eating binges in patients with SRED and NES [9, Class IV; 10••, Class III].

Comorbidities in SRED patients are frequent and are often exacerbated by nocturnal eating. Weight gain in

SRED has aggravated diabetes mellitus, dyslipidemia, hypertension, and obstructive sleep apnea. Further, the nocturnal eating of SRED may interfere with otherwise well-controlled glucose regulation in diabetics. Alarmingly, patients with SRED may ingest substances to which they are allergic. Also, many patients will fall asleep with an oral bolus of food (typically high in carbohydrates), which, combined with the nighttime decline in salivary flow, promotes the development of dental caries. Finally, tooth chipping has been reported from biting into hard substances such as frozen pies [3,9, Class IV].

SRED is a common and relentless condition. A study of eating-disorder patients determined high prevalence rates among inpatients (16.7%), outpatients (8.7%), and an unselected group of college students (4.6%) [11, Class III]. The majority of reported patients are women [3,9, Class IV; 10••, Class III].

The presence of impaired consciousness has historically been a defining characteristic of SRED. In the original series, 32 of 38 patients claimed at least partial impairment in awareness [3, Class IV]. In another case series, 21 of 23 patients had incomplete consciousness and/or amnesia for the behavior [9, Class IV].

Conversely, a recent study has demonstrated full awareness during nocturnal eating episodes. In this clinical and polysomnographic (PSG) investigation, all 26 patients claimed full alertness after nocturnal eating in a sleep laboratory. However the authors did not report whether the subjects typically had full awareness during nocturnal eating at home [10••]. This is an important distinction, as the level of consciousness in a sleep laboratory may be different from the level in a more familiar sleeping environment. Also, in SRED, awareness frequently varies from night to night [12, Class IV]. Reduced awareness with subsequent amnesia is no longer a required diagnostic criterion for SRED in the International Classification of Sleep Disorders, 2nd Edition [8, Class IV].

The spectrum of consciousness noted in SRED reports may be explained by comorbid sleepwalking disorders and the use of sedating medication. The first case reports of nocturnal eating with amnesia were associated with sleepwalking or use of a psychotropic medication [1,13,14, Class IV]. Moreover, most patients in the original series were taking sedating medication or had a previous history of sleepwalking [3, Class IV]. In a survey of 92 nocturnal eaters, there were no sleepwalkers among the 80% of participants who claimed full awareness of their behavior. Conversely, the participants who had a history of sleepwalking were very likely (73%) to be at least partially unaware of the behavior [15, Class III]. None of the 26 patients having full awareness of nocturnal eating in the sleep laboratory study discussed above were taking sedating medications, and only one had a history of sleepwalking [10••, Class III].

ASSOCIATION WITH PSYCHOTROPIC MEDICATIONS

Early reports noted an association between amnestic nocturnal eating and psychotropic medications. In fact, the first case of amnestic nocturnal eating, reported in 1981, was associated with a combination of chlorpromazine, amitriptyline, and methyprylon [1, Class IV]. Subsequently, SRED has also been reported with triazolam, lithium, olanzapine, and risperidone [16, Class IV].

Benzodiazepine receptor agonists (BRAs) have been associated with the initiation and exacerbation of sleepwalking disorders. Often these somnambulistic episodes are more complicated and prolonged than idiopathic sleepwalking behavior [17•, Class IV]. In 2002, the first cases of zolpidem-associated SRED (Z-SRED) were described in five middle-aged patients with a history of restless legs syndrome (RLS). Two of these patients already had intermittent episodes of conscious nocturnal eating. Soon after initiating zolpidem, each patient described amnestic nocturnal eating that stopped with discontinuation of the zolpidem [18, Class IV].

Subsequent case studies have documented the diverse spectrum of Z-SRED. One case report described a 51-year-old woman with RLS who noted empty food packaging in the morning soon after she started taking zolpidem. She later discerned that she had been eating sandwiches on several occasions [19, Class IV]. Recently, a case of Z-SRED was described in a 45-year-old man. After 10 days of treatment, he was missing on two occasions after going to bed. Both times he was found eating "sweets" at his place of business. After driving his car more than 2 kilometers, he had climbed into his office through the shutter instead of using the door [20, Class IV]. This type of complex yet inappropriate behavior is not unusual in sleepwalkers. All of these cases ceased after zolpidem was discontinued.

Recently, a case of uncontrollable nocturnal eating occurred in a 40-year-old woman after she took an increased dose of zolpidem [21, Class IV]. This case illustrates the dose effect of hypnosedative-induced complex behaviors. In our experience, Z-SRED occurs when patients desperate to induce sleep escalate the dose on their own. Based on the risk of inducing Z-SRED and other dangerous behaviors, we strongly caution our patients against this practice.

Extended-release formulations of zolpidem have also been implicated in Z-SRED. A 46-year-old woman presented with new symptoms suggestive of obstructive sleep apnea (OSA) after gaining 50 pounds. She claimed that her weight gain was secondary to amnestic nocturnal eating that began 12 months earlier, soon after she started taking extended-release zolpidem for insomnia. The patient was switched to eszopiclone, also a BRA, and her amnestic eating behavior subsequently stopped [22, Class IV]. Another report describes two women (70 and 75 years old) who developed amnestic

nocturnal eating only after switching from immediate-release to extended-release zolpidem. Both patients also had RLS. Nocturnal eating resolved in both patients after they switched back to the immediate-release formulation [23, Class IV].

Recent publications have demonstrated the prevalence of Z-SRED and speculated upon potential underlying neuronal mechanisms. In a cross-sectional study of a psychiatric population, 14 of 22 patients with SRED had been exposed to zolpidem. Interestingly, 4 of the 22 had been exposed to zopiclone, a less frequently prescribed BRA [24], suggesting that amnestic nocturnal eating is a feature of the BRA class, not merely the agent zolpidem. BRAs induce sleep and amnesia by enhancing GABA activity at α1 $GABA_A$ receptors. It has been suggested that the complex behavior noted with some BRAs is partially related to increasing binding affinity to α1 $GABA_A$ receptors [17•, Class IV].

The incidence of disordered nocturnal eating with the use of these agents is unknown; it may be low. Nevertheless, all patients taking BRAs should be screened for nocturnal eating, considering their widespread use (eg, about 26.5 million US prescriptions for zolpidem annually) [25].

A substantial number of cases of Z-SRED are seen with comorbid RLS [18,19,23, Class IV]. RLS is a condition distinct from insomnia but often clinically confused with it. Difficulty initiating and maintaining sleep is a major presenting complaint in patients with RLS. We speculate that inappropriate treatment of RLS as insomnia is a crucial underlying step in the pathogenesis of many Z-SRED cases.

Weight gain is often correlated with the onset of SRED and is frequently reversed with treatment of SRED [3, Class IV]. Some of the most striking examples of SRED-associated obesity are those associated with zolpidem [19,22, Class IV]. These reports concur with our clinical experience of patients reporting dramatic weight gain associated with BRAs.

ASSOCIATION WITH OTHER SLEEP DISORDERS

SRED has also been associated with other sleep disorders, particularly other parasomnias such as sleepwalking. In the original reported series of 38 patients with SRED, sleepwalking was noted in 23, RLS in 5, OSA in 4, and narcolepsy in 3. In another study of 23 SRED patients, 18 had a history of a parasomnia [9, Class IV]. In our experience, sleepwalking without eating often precedes SRED. Then, once nocturnal eating occurs, it may become the predominant or exclusive sleepwalking behavior.

The treatment of SRED should depend on the presence of any associated sleep disorders. Treating conditions such as RLS, periodic limb movement disorder (PLMD), sleepwalking, and OSA often result in excellent treatment of the nocturnal eating [3,14, Class IV].

Treatment

- A primary goal of obesity research is to identify reversible causes of weight gain. Nocturnal eating (whether NES or SRED) is a common phenomenon that is associated with increasing body mass index [3, Class IV; 11, Class III; 16, Class IV; 24,26, Class III]. Importantly, nocturnal eating is treatable, and its treatment frequently leads to weight loss (Table 1). The goals of therapy should be to eliminate feeding episodes and reverse any weight gain attributed to the nocturnal eating.
- Drug-induced SRED can usually be abated by discontinuing the offending medication [18–21,23, Class IV]. Rarely, episodes will persist in patients who did not have nocturnal eating prior to exposure. It is not certain whether this is a temporary phenomenon or whether the offending agent has lowered a threshold in patients who were predisposed to nocturnal eating. We usually recommend bedtime topiramate for these patients.
- For patients with idiopathic nocturnal eating, three types of pharmacotherapies seem effective: dopaminergic agents such as pramipexole, the antiseizure medication topiramate, and the selective serotonin reuptake inhibitor (SSRI) sertraline. More research (especially randomized controlled trials) is needed.
- Initial investigation into nocturnal eating should attempt to identify comorbid sleep disorders and to eliminate inducing agents. SRED is associated with psychotropic medication, most notably the BRA zolpidem. Polysomnography with a seizure montage at an accredited sleep laboratory is needed to identify and treat comorbid sleep disorders. We also ask patients to use sleep and food diaries to help in monitoring treatment response.

Table 1. Studies of pharmacologic treatment of nocturnal eating

Medication class	Agent	Study	Study design	Primary diagnosis	*N*	Responders, *n**
Dopaminergics	Carbidopa/ levodopa	Schenck et al. [3]	Case series	SRED	12	10; temporary remission in 2
	Bromocriptine	Schenck et al. [3]	Case series	SRED	3	2; temporary remission in 1
	Pramipexole	Provini et al. [28]	Pilot double-blind, placebo-controlled, crossover	SRED	11	Decreased nocturnal activity as measured by actigraphy; no change in number of awakenings or ingestions
Antiseizure drugs	Topiramate	Winkelman [29]	Open-label trial	SRED, NES	2 SRED, 2 NES	4; mean weight loss 11.1 kg over 8.5 mo
	Topiramate	Martinez-Sallio et al. [30]	Case report	SRED	1	1 (2-y follow-up)
	Topiramate	Schenck et al. [31]	Case series	SRED	17	12/17; mean weight loss of 9.2 kg among responders after 1.8 y
	Topiramate	Winkelman [12]	Open-label trial	SRED	25	17; mean weight loss 11.1 kg over mean 11.6 mo; adverse events occurred in 25 participants, and 7 responders discontinued treatment within 1 y
Serotonergics	Fluoxetine	Schenck et al. [3]	Case series	SRED	3	2
	Fenfluramine	Spaggiari et al. [32]	Case series	NES	7	6; mean weight loss 11.1 kg over 8.5 mo
	Paroxetine, fluvoxamine	Miyaoka et al. [33]	Case series	Nocturnal eating/drinking syndrome†	4	4 (3 paroxetine, 1 fluvoxamine)
	Sertraline	O'Reardon et al. [34]	Open-label trial	NES‡	17	Compared with baseline, awakenings and nocturnal ingestions were reduced after starting sertraline ($P < 0.01$)
	Sertraline	O'Reardon et al. [35•]	Double-blind, placebo-controlled	NES‡	34	Nocturnal ingestions were reduced 81% with sertraline vs 14% with placebo; overweight and obese patients lost 2.9 kg with sertraline vs 0.3 kg with placebo

*Defined by elimination of nocturnal eating or diminished nocturnal eating with other clinical improvements such as weight loss.
†Terminology not frequently employed.
‡NES studies were included if nocturnal eating was specifically included in the definitiion and stated as an outcome measure.
NES—night-eating syndrome; SRED—sleep-related eating disorder.

Diet and lifestyle

- There is no evidence that changes in diet or lifestyle are effective in the treatment of nocturnal eating.

Treatment options for sleep-related eating disorder

Pharmacologic treatment

Dopaminergic agents (pramipexole)

- Dopaminergic agents such as pramipexole may be effective in the treatment of SRED even in the absence of RLS or PLMD. The original case series noted that either levodopa or bromocriptine at bedtime was effective in eliminating nocturnal eating, especially in patients with associated RLS [3, Class IV].
- The mechanism by which dopaminergic agents suppress nocturnal eating is unknown. Appetite suppression has been demonstrated in animal models [27, Class II]. Recently, pramipexole was investigated in a small double-blind, placebo-controlled crossover trial. Pramipexole was well tolerated in all patients, including those without RLS or PLMD. Participants taking pramipexole noted improved sleep, and reduced nighttime activity was documented with actigraphy. There was no improvement in the number or duration of awakenings [28, Class II].

Standard dosage	0.125–1.0 mg at bedtime.
Contraindications	Thought disorder, elevated mood disorder, bipolar affective disorder, hypotension, renal impairment.
Main drug interactions	Interactions are minimal; there is no interaction with the cytochrome p450 system. Dopamine-blocking agents such as antipsychotics or antinausea agents may diminish the effectiveness of pramipexole.
Main side effects	Sedation, orthostasis, manic episodes, nausea, hallucinations.
Cost	A 1-month supply will cost $100 to $200.

Topiramate

- Early studies indicate that the antiseizure medication topiramate may be an effective treatment for nocturnal eating in SRED. An open-label trial of topiramate in four patients with nocturnal eating demonstrated positive results. The agent was well tolerated, nocturnal eating diminished, and weight loss (mean of 11.1 kg) was noted in all four individuals over 8.5 months [29, Class IV].
- A case report with similar results was recently published. This 28-year-old obese man had a 10-year history of nocturnal eating episodes that were eliminated with topiramate, which was well tolerated over a 2- year follow-up [30, Class IV].
- In a case series published as an abstract, 12 of 17 SRED patients treated with topiramate responded to treatment. The agent was well tolerated, and those who responded lost a mean of 9.2 kg over 1.8 years [31, Class IV].
- Another chart review of 25 follow-up SRED patients reported that 68% were treatment responders. In 1 year, 28% of patients lost more than 10% of their body weight. The rate of adverse events was high, however, and 41% of patients discontinued the medication [12, Class III].

Standard dosage	25–200 mg at bedtime.
Contraindications	Anorexia, renal impairment, history of renal calculus, cognitive impairment, hypotension.
Main drug interactions	Hyperammonemia with valproic acid. Decreased effectiveness of oral contraceptives. Interactions are very minimal at low doses, and nocturnal eaters take a smaller dose than seizure-disorder patients, who take topiramate twice daily.
Main side effects	Weight loss, paresthesias, renal calculus, cognitive dysfunction, orthostasis.
Cost	$50 to $100 for a 1-month supply of the generic formulation.

Selective serotonin reuptake inhibitors

- SSRIs may be effective in treating nocturnal eating in patients with SRED, but more research is needed. In the original case series, fluoxetine was effective in two of three SRED patients [3, Class IV].

Surgery

- Theoretically, bariatric surgery may improve nocturnal eating among patients with SRED.

Treatment options for night eating syndrome

Pharmacologic treatment

Selective serotonin reuptake inhibitors (sertraline)

- Modulation of serotonin in the central nervous system may lead to effective treatment of nocturnal eating. In 1994, an investigation reported that all six nocturnal-eating patients treated with the serotonin agent fenfluramine had a pronounced reduction in caloric intake and episode frequency [32, Class IV]. Another report described complete eradication of nocturnal eating in four patients as early as 2 weeks after initiation of an SSRI [33, Class IV].
- In an open-label trial investigating the effect of sertraline on NES, the patients who completed the 12-week study reported fewer awakenings and nocturnal ingestions [34, Class III].
- A recent double-blinded, placebo-controlled study assessed the efficacy of sertraline in treating 34 patients with NES. After 8 weeks, 71% of subjects in the sertraline group responded, compared with 18% in the placebo group. The number of nocturnal ingestions fell by 81% in the sertraline group and 14% in the placebo group. Importantly, overweight and obese subjects in the sertraline group lost more weight than similar subjects in the placebo group (2.9 kg vs 0.3 kg) [35•, Class II].

Standard dosage	50–150 mg at bedtime.
Contraindications	Suicidal ideation (risk associated with SSRIs), hypotension.
Main drug interactions	Dangerous interactions with monoamine oxidase inhibitors and triptans; potential for the serotonin syndrome. Serotonin is highly protein-bound and thus may displace other highly bound compounds such as warfarin and digoxin.
Main side effects	Sexual dysfunction, diarrhea/loose stools, sedation, orthostasis.
Cost	$20 to $30 for a 1-month supply of the generic formulation.

Surgery

- Although studies have investigated the relationship between NES and bariatric surgery outcomes, we are unaware of any evidence suggesting that bariatric surgery may affect nocturnal eating in NES.

Pediatric considerations

- Evidence suggests that nocturnal eating may begin during adolescence. Unfortunately, there have been no reports regarding treatment of nocturnal eating in pediatric populations.

Disclosure

No potential conflicts of interest relevant to this article were reported.

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