Sleep-Related Eating Disorder in a 29 Year-Old Man: A Case Report with Diagnostic Polysomnographic Findings

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Abstract- This is a case of a 29-year-old man with a 6 year history of sleep-related eating disorder (SRED) that occurred with partial consciousness on a nightly basis. His family or wife witnessed up to 5 episodes every night, with each eating episode lasting 8-16 minutes. Polysomnography documented 4 episodes of sleep-related eating arising from stage 2 Non-REM sleep, when he consumed cookies that he had brought to the sleep lab that night. While eating, his EEG remained in stage 2 sleep or else was a wakeful EEG, and the eating episodes lasted for a mean 13.3 minutes. There was no epileptiform EEG activity during the polysomnogrphic study with a seizure montage and fast paper speed. Therapy with clonazepam, 0.5 mg bedtime, did not control the nocturnal eating. The patient tried to limit access to food in his home before bedtime, and this had modest benefit. This case of SRED has both typical and atypical features, which are discussed.

Key Words: Sleep-related eating, Parasomnia, Eating disorder, Nocturnal eating, Sleepwalking, Clonazepam, Polysomnography

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INTRODUCTION

Abnormal nocturnal eating was first reported by Stunkard et al. in 1955 as the "night-eating syndrome" (NES) affecting 20 adults with refractory obesity who complained of nocturnal hyperphagia, insomnia, and morning anorexia[1]. Over the next 35 years, abnormal nocturnal eating received scant attention in the literature, with polysomnographic data being reported in only four cases[2-3], until the formal identification of "sleep related eating disorder" (SRED) by Schenck et al. in 1991[4]. The initial reported series of 19 consecutive patients was expanded to a larger series of 38 patients that was reported in 1993[5]. SRED usually features partial consciousness during eating episodes, with limited subsequent recall, whereas NES occurs in full wakefulness with overeating between dinner and bedtime, and/or during full awakenings with complete next-day

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recall. SRED is listed as a parasomnia in the recently revised International Classification of Sleep Disorders (ICSD-2)[6]. NES is regarded as a sleep maintenance insomnia disorder with inappropriate phase delay of substantial food intake, but without phase delay in the timing of nocturnal sleep onset compared to controls[7].

The onset of sleep-related eating can be sudden, associated with major life stresses, medical or neurological disorders, use of certain medications, or cessation of cigarette smoking; or it can develop gradually. The course is often progressive. In a series of 38 patients, 65.9% were women with a mean age at presentation of 38.8 years (range 18-65), and the mean age of nocturnal eating onset was 25 years (range 5-44)[5]. Common features of sleep-related eating include consumption of high-calorie foods; bingeing but not purging; elaborate but sloppy food preparation, including cooking; burns and lacerations from careless food preparation and cooking; and idiosyncratic concoctions such as cat food or salt sandwiches, or buttered cigarettes. Partial or complete amnesia for the episodes is common, and there may be associated dream-like mentation. Sometimes, however, there can be alertness and subsequent recall of the nocturnal eating. Eating may occur up to eight times nightly, and patients often describe abdominal distention with anorexia in the morning and weight gain from nocturnal eating. Almost half of patients are obese and attribute weight gain to nocturnal eating. Alcohol consumption during nocturnal episodes is rare. Psychiatric disorders, usually mood disorders are present in about half of reported patients, with the onset of sleep-related eating usually preceding the onset of the psychiatric disorder by a number of years. The prevalence of SRED across clinical and non-clinical populations is surprisingly high, in excess of 4% in the United States[8].

CASE REPORT

A 29-year-old, married Taiwanese man employed as a truck driver presented with a 6-year history of SRED. His family had witnessed his eating behavior during nocturnal sleep from the age of 23, while he was still single. He did not overeat between dinner and bedtime. The night eating behavior was much worse after he got married at the age of 27. The patient usually got up to eat about 4 or 5 times every night as witnessed by his wife, but he could not remember the night eating behavior when he woke up in the morning. One night, he arose from bed looking for something to eat, but when he could not find food he rode his motorcycle to a 24-hour convenience store to buy some food, but he only had partial memory for this event the next morning. During episodes of SRED, he had kicked the corners of various pieces of furniture several times that caused repeated toe injury, and he nearly caused a fire one time due to his smoking. On that night, he got up to eat in a partially conscious state, and then he lit a cigarette while he was eating some food in the living room, but when cigarette ash dropped on his thigh, the hot ash awakened him. Most mornings, he usually felt nauseated with abdominal fullness, and some days he had little appetite. There was no reported weight gain.

This patient presented to the sleep center because of disruptive sleep with eating behavior and daytime fatigue. Prior to the onset of SRED, there was no identified abnormal sleep history, including childhood, and there was no positive medical or psychiatric history, or any positive family history. There was no current history of restless legs or leg jerking during sleep, and he did not snore. The night eating behavior developed gradually since its onset 6 years prior, and became progressively worse eventually occurring nightly during his 2 years of marriage. There was no precipitating medical or psychosocial event preceding the emergence of the night eating behavior. He had no drug use history or any caffeine intake, but smoked 1 pack of cigarettes daily. Alcohol consumption consisted of a small half-cup of Chinese herb wine every night. His usual sleep time was from midnight to 8:30 am, with an added hour of sleep during holidays. His childhood nocturnal sleep pattern was from 10 pm to 7 am.

The patient completed a comprehensive questionnaire covering lifetime sleep-wake, medical and psychiatric history, and review of systems. He and his wife were interviewed. Neurological exams and psychiatric interviews were conducted. Overnight, hospital-based,

polysomnographic (PSG) monitoring, utilizing standard recording and scoring methods, was then performed on this patient, and included monitoring of eye movements; expanded EEG (seizure montage) with fast paper speed; chin and leg electromyograms; airflow, chest and abdomen respiratory effort; electrocardiogram; and continuous audiovisual recording. During the overnight PSG study, the patient exhibited four episodes of eating with reduced level of consciousness that each time arose from stage 2 Non-REM sleep. The four episodes of eating occurred during the following times, with the first episode appearing 34 minutes after sleep onset at 0:38:42 am: 1st episode: 1:12:42 am to 1:28:12 am; 2nd episode: 2:15:42 am to 2:24:42 am; 3rd episode: 2:50:12 am to 3:03:12 am; 4th episode: 3:41:12 am to 3:49:12 am. The mean interval between the four eating episodes was 49.5 minutes, and the mean eating behavior duration was 11.4 minutes, unaccompanied by seizure-like EEG activity or dream recall. While eating, his EEG remained in stage 2 sleep or else became admixed with or converted to a wakeful EEG, but without full alertness, as observed by the sleep technologist. Neither bruxism nor rhythmic masticatory-muscle activity occurred during the PSG study. With the morning awakening, the patient had no memory of any eating episodes the previous night. Fig. illustrates one such episode from stage 2 sleep. Total sleep time was 4.0 hours; time spent eating from stage 2 sleep was 45.5 minutes during a mixed sleep/wakefulness state that accounted for over 14% of the sleep architecture; total time in bed was 5 hours,

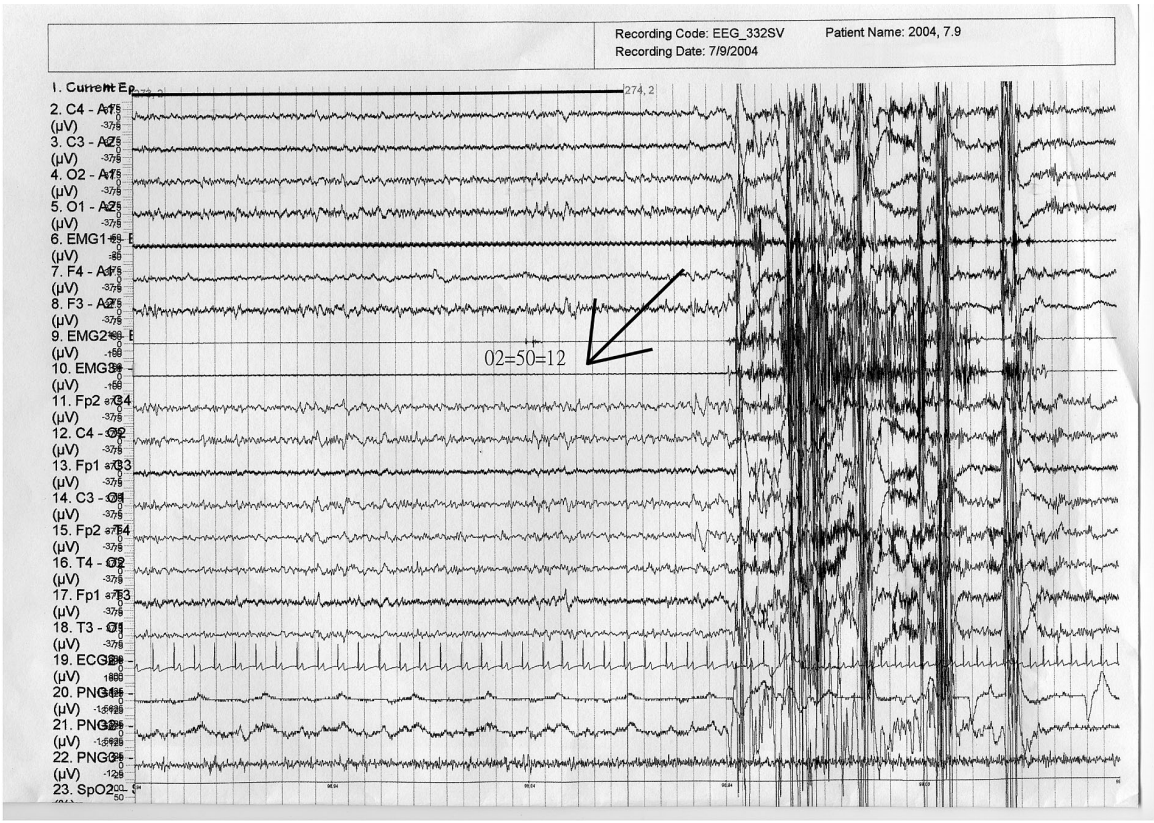

Figure. Nocturnal PSG (60s epoch, Ep 273 and 274) during the stage 2 sleep period and the emergence of an eating-behavior episode with EMG artifact (PSG takes 30 seconds as an epoch, Ep273.2 is mean the 273rd epoch and this epoch is in stage 2 sleep). Arrow indicates the time (02:50:12 am) of the episode beginning. EEG montage (channels 10-17) detects no seizure activity. The electrocardiogram (channel 18) does not show an increase in heart rate during the motor activation with behavior release. The channels 20-23 represent the nasal/oral airflow, chest respiratory effort, abdomen respiratory effort and O2 saturation, that do not show any sleep apnea or oxygen desaturation.

22.5minutes. Sleep architecture: stage 1, 2.4%; stage 2, 50.6%; stage 3/4, 13.1%; REM sleep, 8.7%; eating behavior time, 14.2%; wake time, 9.6%; movement time, 1.4%. There was no snoring, apneas/hypopneas, oxygen desaturations, periodic breathing, periodic limb movements, or excessive arousals during the PSG study, apart from the 4 episodes of eating.

On the basis of his clinical history and the diagnostic PSG study, the diagnosis of SRED was confirmed[6]. Clonazepam therapy (a standard therapy for slow-wave sleep parasomnias, such as sleepwalking[9]), 0.5 mg at bedtime was initiated, but the patient did not respond and discontinued the therapy after two weeks, and was lost to follow-up. Two years later the patient was contacted and he reported modest benefit from restricting access to food before bedtime.

DISCUSSION

This is a case of a 29 year-old man who fulfills the ICSD-2 diagnostic criteria of definitive SRED that had both typical and atypical features. A non-REM sleep parasomnia was well documented during 4 eating episodes in his PSG study. However, in contrast to the Disorders of Arousal, including sleepwalking, that predominantly emerge from slow-wave (delta) sleep, his SRED (sleepwalking with eating) consisted of a stage 2 Non-REM sleep parasomnia. For this patient, the multiple-times and nightly episodes of involuntary eating emerged from sleep with reduced consciousness and subsequent partial or complete amnesia. Since there was no prior history of sleepwalking, restless legs syndrome, and no other sleep disorder was found during the PSG study, and he was not taking any medication, his SRED was considered to be idiopathic. Being male gendered is atypical for SRED, which is considered a female-predominant disorder[5,10]. Also, the frequency of his nocturnal eating episodes (20-35 episodes weekly), is considerably higher than average for SRED. This atypical feature combined with a history of reduced consciousness with amnesia for the episodes, would raise the possibility of nocturnal seizures, but there was no other clinical or any EEG evidence supporting such a diagnosis. There were no identified triggers for nocturnal eating, such as periodic limb movements or sleep-disordered breathing. Likewise, there was no rhythmic masticatory-muscle activity during the PSG study; in contrast, in a recently reported series of SRED patients from Italy, such activity was found in 29 of 35 patients during stages 1 and 2 non-REM sleep[11], and these patients had nocturnal eating in the sleep lab during full alertness, which is atypical compared to previously reported SRED series.

Other possible causes of SRED could easily be excluded in this case, such as nocturnal bulimia nervosa, nocturnal psychogenic dissociative disorder (with a "night-eating" personality), medical triggers, or cessation of cigarette smoking[5]. He was not taking any medication; recently, zolpidem therapy of insomnia has been reported to induce de novo onset of SRED with profound subsequent amnesia[12,13]. Therefore, SRED can be viewed as a "final common pathway disorder" that can be accessed by a variety of sleep disorders, medical-neurological disorders, and medications. The consequences of chronic, longstanding SRED can be severe, including excessive weight gain, obesity, metabolic syndrome, dental complications, sleep-related injury from cutting or burning oneself in the kitchen, daytime tiredness, and interpersonal/social consequences.

Therapy with clonazepam was not effective, which is in contrast to the high success rate in controlling sleepwalking without eating[9]. One of the early and effective therapies of SRED consisted of bedtime levodopa, at times combined with a minor opiate such as codeine or propoxyphene. This therapeutic modality (a standard treatment for restless legs syndrome/periodic limb movement disorder--RLS/PLMD) was initially found to be effective in patients with SRED emerging together with RLS and PLMD. This then led to the recognition that the same bedtime levodopa therapy, at times combined with opiate (and clonazepam) therapy also controlled cases of SRED that were either idiopathic or associated with sleepwalking[5]. The efficacy of this therapy for SRED[14] may be comparable to the efficacy of topiramate therapy (to be described below). Patients with SRED and histories of chemical dependency can also respond to combined levodopa, trazodone and

bupropion (i.e. dopaminergic antidepressant) therapy at bedtime[15]. The dopamine receptor agonist pramipexole may also be a promising therapy[16]. The mechanism of action remains unknown, but dopaminergic dysfunction may promote SRED beyond disorders such as RLS/PLMD with known dopamine-related dysfunction.

Recently, in two separate series, the anticonvulsant topiramate was reported to be effective in controlling SRED in two-thirds of treated patients, presumably on the basis of suppressing the urge to eat rather than an anticonvulsant effect or a direct effect on sleep[17,18]. The starting dose is 25 mg at bedtime, with increases by 25 mg increments every 5 days up to the typical therapeutic range of 50-150 mg (which can be extended to 200-400 mg). At times topiramate will control the nocturnal eating but not the awakenings, in which case the addition of low dose trazodone or clonazepam at bedtime can be effective. Paresthesias and various other side-effects have been reported, which at times result in cessation of therapy.

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