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Original Article

New data on psychological traits and sleep profiles of patients affected by nocturnal eating

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ABSTRACT

Objective: Nocturnal eating behavior is shared by patients affected by a parasomnia, sleep-related eating disorder (SRED), and several eating disorders such as night eating syndrome (NES) and binge-eating disorder (BED); however, the differential clinical features of these patients have been poorly studied, with persisting difficulties in defining the borders between these pathologies. The aim of this study was to evaluate polysomnographic and personality characteristics of nocturnal eaters to further differentiate the syndromes.

Methods: During a period of six months, consecutive patients complaining of nocturnal eating were asked to participate to the study. Twenty-four patients who were found to eat during the polysomnographic recording (PSG) study, and gender-matched control subjects were included. All subjects underwent a full-night video-PSG study and a psychometric assessment including the Eating Disorder Inventory (EDI-2), the self-rating Bulimic Investigatory Test–Edinburgh (BITE), the Temperament and Character Inventory (TCI), and the Barratt Impulsivity Scale (BIS).

Results: Nocturnal eaters showed a mild reduction in sleep efficiency and duration due to a moderate sleep fragmentation, whereas the percentage of each sleep stage was not significantly affected. Nocturnal eaters scored higher at many subscales of the EDI-2, at the BITE symptoms subscale, and at the BIS attentional impulsivity subscale.

Conclusion: The psychological characteristics found in our patients with NES seem to be typical for patients affected by eating disorders, and support the hypothesis that the nocturnal behavior of these individuals is due to an eating disorder; however, specific traits also allow differentiation of NES from BED.

1. Introduction

Recurrent nocturnal eating episodes represent the main symptom in patients affected by night eating syndrome (NES) as well as in those affected by sleep-related eating disorder (SRED). Although NES is considered to be an eating disorder, SRED is classified among parasomnias [1]. Moreover, patients affected by other eating disorders, such as bulimia nervosa (BN), anorexia nervosa (AN), and binge-eating disorder (BED), may also eat at night [2]. Only recently have sleep experts and eating disorders specialists agreed on a better definition of clinical and polysomnographic features of nocturnal eaters. This joint venture effort led to the publication of standard diagnostic criteria for NES [3] (Table 1) and to the revision of the diagnostic criteria for SRED, included in the last version of the International Classification of Sleep Disorders, Third Edition (ICSD-3) [4] (Table 2). However, the boundaries of these two nocturnal disorders are still blurred, especially in patients presenting with isolated SRED episodes in context of a NES condition [5]; also, the relationship of NES with other eating disorders is still poorly studied. Delving deep into the psycho-behavioral aspects of patients with NES might help to better define the borders of this particular eating disorder and, hopefully, shed some light on its psycho-pathogenesis.

Several studies have evaluated the psychopathological characteristics of patients affected by nocturnal eating, focusing mainly on anxiety and depression [6]. A positive history of anxiety was mentioned in the first published study on the syndrome in 1955 [7] and more recently confirmed by Schenck and Mahowald [8]. In patients with NES, evening hyperphagia, mood, and sleep disturbances

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Table 1
Proposed diagnostic criteria for night eating syndrome.

I. The daily pattern of eating demonstrates a greatly increased intake in the evening and/or night-time, as manifested by one or both of the following:
A. At least 25% of daily food intake is consumed after the evening meal
B. At least two eating episodes per week occur upon awakening during the night
II. The clinical picture is characterized by at least three of the following features:
A. Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week
B. Presence of a strong urge to eat between dinner and bedtime and/or during the night
C. Sleep onset and/or sleep maintenance insomnia are present four or more nights per week
D. Presence of a belief that one must eat in order to get to sleep
E. Mood frequently depressed and/or worsens in the evening
III. Awareness and recall of evening and nocturnal eating episodes is present.
IV. The disorder is associated with significant distress and/or impairment in functioning.
V. The disordered pattern of eating has been maintained for a minimum of 3 months.
VI. The disorder is not secondary to substance abuse or dependence, a general medical disorder, a medication, or another psychiatric disorder.

Note: Criteria III, IV, V, V all have to be satisfied in addition to at least one of the two criteria of items I and three out of five criteria of item II.
Based on Allison et al. [3].

Table 2
Diagnostic criteria for sleep-related eating disorder (SRED).

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

Based on International Classification of Sleep Disorders, Third Edition [4].

are positively correlated with their nocturnal mental anxiety [9] and depression is a risk factor for NES [10,11]. Compared to healthy controls, NES patients report more pronounced eating disorder pathology as well as general psychopathology and chronic social stress [12]. The highest prevalence of NES is found among obese individuals [13,14], but not all subjects with NES are obese or overweight [15–18]; very limited data are available on the personality traits of nonobese NES patients [6,15]. Marshall et al. [16] did not find differences between a group of 40 obese NES subjects and a group of 40 nonobese NES patients with respect to morning appetite, evening hyperphagia, insomnia, mood, perceived need to eat to fall asleep, or cravings and control over eating in the evening and upon awakening during the night. Lundgren et al. [6] reported that nonobese night eaters were younger, significantly more nocturnal awakenings and nocturnal ingestions of food, and scored significantly higher on all subscales of the eating disorder examination, compared to controls (dietary restraint, weight concern, shape concern, eating concern, and Eating Disorder Examination global score). Moreover, these investigators reported more depressed mood, higher perceived stress, lower quality of life, and a more frequent lifetime history of criteria for Axis I disorders; 26% of night eaters, and none of the controls, met lifetime criteria for substance abuse or dependence.

Studies on hormonal characteristics of NES patients report significant changes in the timing and amplitude of various physiological circadian markers involved in appetite and neuroendocrine regulation; in general, the neuroendocrine circadian patterns of NES patients are characterized by an attenuated nocturnal rise in the plasma concentrations of melatonin and leptin and a greater increase in the concentration of cortisol [19]. Disturbances in the hypothalamic–pituitary–adrenal axis with an attenuated ACTH and cortisol response to corticotrophin-releasing hormone (CRH) were found. Compared to controls, CRH-induced ACTH and cortisol response were significantly decreased to 47% and 71%, respectively [19]. Among night eaters, an increased level of cortisol throughout a 24-h period has been reported, especially from 08:00 to 02:00 [15], although other studies found no differences either between NES and controls [20] or between nonobese and obese night eaters; however, in this study, NES patients showed reduced amplitudes in the circadian rhythms of cortisol [21]. Geliebter et al. found that NES patients had higher baseline cortisol levels than controls, but not when controlling for baseline cortisol levels [22]. NES patients have an alteration in the circadian rhythm of both leptin and ghrelin [23], but the research is far from completely clarifying the influence of these hormones on the onset and maintenance of NES [15]. A delay of 1.0–2.8 h has been reported in the circadian rhythms of leptin (with a trend for a delay in the circadian cortisol rhythm), whereas circulating levels of ghrelin were phase advanced by 5.2 h. It must be noted that altered food intake could cause the observed hormonal patterns or vice versa; the patterns could be in a reinforcing cycle; and/or a third factor (eg, disturbed sleep) might cause both changes simultaneously [21]. Allison et al. [20] reported that the plasmatic levels of leptin did not differ between NES and controls, but these results are in contrast to those of Birketvedt et al. [15], who reported that plasma leptin levels were lower in nonobese than in obese night eaters. Also, studies on the plasmatic levels of ghrelin yielded controversial results: one study reported that they were significantly lower in NES than in controls [20], but other researchers [24] found that a more or less flat curve of the nocturnal increase of ghrelin was observed not only among NES patients (both normal weight and overweight) but also among overweight non-NES participants; there was a significant independent lowering effect of overweight on ghrelin. Geliebter et al. [22] measured plasma ghrelin concentration in response to a physiological laboratory stressor (cold pressor test [CPT]), in overweight women with and without NES; ghrelin levels increased, as did stress and hunger ratings, across all subjects (NES and non-NES). Plasmatic levels of melatonin did not differ between NES patients and controls, but delays of 1.0–2.8 h were found in its circadian rhythm, similar to those of leptin and insulin [21]. The glucose rhythm of NES patients shows an inverted circadian pattern, together with reduced amplitudes in the circadian rhythms of insulin [21]. Allison et al. [20] reported that insulin and glucose levels were higher in NES patients than in controls, and that these differences were closely associated with nocturnal food intake. There were independent significant lowering effects of overweight and NES on the serum GH levels; no changes in the serum levels of insulin-like growth factor-1 (IGF-1) or insulin-like growth factor-β3 (IGFB-3) were observed [24]. Independent significant lowering effects of overweight and NES on the levels of IGF-1 were found, whereas a nearly significant reduction in the global levels of IGFBP-3 was observed in both NES groups [21]. Finally, among NES patients an increased amplitude in the circadian rhythm of thyroid-stimulating hormone (TSH) was also described [21]. The differences in the experimental conditions can provide a partial explanation of the different results of the two main neuroendocrine data sets [15,20] of NES patients. Subjects in the study conducted by Birketvedt et al. [15] were observed during a single 24-hour period with no run-in time; in contrast, patients in the study by Allison et al. [20] were observed on the third day, allowing for two days of run-in time. In addition, subjects in the study by Birketvedt et al. [15] were fed four controlled meals of 300 kcal