Early Sleep Psychiatric Intervention for Acute Insomnia: Implications from a Case of Obsessive-Compulsive Disorder

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CASE REPORTS

Insomnia is a common problem among patients with obsessive-compulsive disorder (OCD), and patients suffering from acute insomnia with psychiatric comorbidity are more likely to develop chronic insomnia without appropriate intervention. Here we report a case of obsessive-compulsive disorder with acute insomnia, successfully treated with early sleep psychiatric non-pharmacological intervention. The augmentation of medication runs a risk of exacerbating daytime impairment. Clinicians usually prescribe medication, such as antidepressants and hypnotics without reflections for such complaints. However, the use of these sedative

agents is often problematic, especially when patients have kept a good QOL activity in daily life. The rapid recovery from acute insomnia in this case suggests that the appropriate use of actigraphy is a favorable non-pharmacological intervention in acute insomnia.

Keywords: Insomnia, Obsessive-Compulsive Disorder, actigraphy, subjective-objective discrepancy

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Obsessive-compulsive disorder (OCD) is characterized by recurrent obsessive thoughts, images, or impulses that evoke anxiety and compulsive behaviors (e.g., handwashing) or mental acts (e.g., ritualistic praying) aimed at decreasing discomfort.¹ The lifetime prevalence is comparatively high at 2% to 3.5% of the population.² While neither the core syndromal manifestations nor prominent associated features of OCD include sleep disturbances, patients suffering from OCD often complain about sleep disturbance. Clinical observations show that their complaints are nonspecific and persist. Previous sleep studies among patients with OCD are sparse and results inconsistent, often confounding with their comorbid depressive illness.³

Psychiatric disorders, such as neurotic disorders including OCD, are often associated with sleep disorders, especially insomnia, which is a crucial element in clinical practice.^{1,4} Characteristically, insomniacs often report their sleep to be worse than is objectively measured.^{5,6} It means that the discrepancy between subjective daytime complaints and objective performance in individuals with insomnia is common.⁶ Therefore, we have to organize specific remedies for co-occurring acute insomnia itself.⁷ Here we present a case of OCD involving a 25-year-old male. The purpose of this single case study is to show the possible efficacy of early sleep psychiatric intervention, mainly focusing on the subjective experience of the individual with acute insomnia.

REPORT OF CASE

Mr. T., a 25-year-old man, had a long history of OCD with recurrent obsessive thoughts of touching dirt and compulsive cleaning since preadolescence. At the age of 18, he first consulted a psychiatrist for the purpose of treating his depressive symptoms

after his father's sudden death. His depressive symptoms improved and then stabilized for several years with the aid of pharmacologic treatment (sulpiride 30 mg, clorazepate 7.5 mg, and paroxetine 20 mg). After graduating from professional school, he was able to work as a computer engineer in an urban company, despite persistence of his obsessive symptoms. One winter, he was referred to our outpatient clinic by his general practitioner. His symptoms had already stabilized because of the same medication as a long-term maintenance treatment for OCD.

After 4 months of our follow-up, he was transferred to another section in his company. This change of social environment made him cogitate about his interpersonal relationship with other colleagues, which provoked acute insomnia symptoms, such as difficulty falling asleep and nighttime awaking. Additionally, he also suffered from daytime impairment related to his insomnia, especially hypersomnia and daytime sleepiness. He said, "I can't concentrate on my work because I made efforts to get to sleep last night", "I have to fight to stay awake during my work" and "I feel afraid of falling asleep tonight because of my insomnia". Typically, the fear of insomnia was exacerbated.² In other words, he was very afraid of losing his career position in his new section caused by the daytime impairments (e.g., losing concentration and diminished performance), which he attributed to his insomnia.

In order to improve acute insomnia symptoms, we treated him mainly with an early sleep psychiatric approach as a non-pharmacological intervention. Intentionally, we avoided increasing medication, because his principal concerns were strongly related with daytime impairment of insomnia. Adding another medication to improve sleep might run a risk of exacerbating daytime consequences of insomnia. In this situation, we treated him, making use of home-monitoring actigraphy and an oxy-

gen saturation tool. After two days of monitoring, the actigraphy measured total sleep time and number of nighttime awakenings; the data showed that he had slept sufficiently during the night, contrary to his complaints. As a first step, we supported him by showing him recorded sleep data, which suggested that his objective quality of sleep was different from his subjective complaints. This manifestation explained by itself a typical psychopathology of insomnia.^{5,6} This monitoring continued for a week. During this week, he was encouraged to visit our clinic 3 times for evaluation. His nighttime actigraphic records always suggested longer and more consolidated sleep than indicated by the subjective intensity of insomnia complaints. As a next step, one night he was asked to get installed a portable oxygen saturation tool. The obtained data showed that some presence of hypoxemia during his sleep, which could partially explain the fragility of his sleep function. Also, he was found drinking alcohol and smoking just before going to bed. Moreover, he often surfed the Internet, eating snacks during the night on weekends. We considered this information as important evidence to strongly stop him from smoking and drinking before bedtime, and urging him to keep regular habits, even at the weekend. With this intervention equipped with the home-monitored objective data, also based on sleep hygiene education (e.g., avoid bedside drinking, smoking, snacking, and surfing the Internet), his anxiety and fear of insomnia diminished dramatically, and he spontaneously recovered from acute insomnia.

DISCUSSION

Recently, sleep psychiatry (psychiatric therapeutic approach, both biologically and psychologically, based on sleep science) has gathered much attention worldwide.⁴ In this case, we attempted an early intervention in the vicious cycle of acute insomnia.⁶ This early sleep focused intervention prevented him from entering the chronic vicious cycle of psycho/physiological hyperarousal, which was supposed to play a central role in the pathophysiology of insomnia.⁵⁻⁸ Possible other reasons explaining his diminished quality of sleep in this case, were as follows: (1) presence of co-occurring subclinical depressive symptoms, (2) negative consequences of core OCD symptoms of sleep habits, and (3) concurrent diurnal side effects of long-term prescribed medication. Clinically, these aspects must always be taken into consideration for managing sleep disturbance comorbid with neurotic disorders, including OCD.³

We emphasize several suggestions about acute insomniac state. “I can’t sleep,” “I don’t get enough sleep”: This kind of complaint has often led to the easiest solution of direct prescriptions of hypnotics. The accumulation of hypnotics has eventually had negative consequences in their everyday QOLs, such as daytime sleepiness and diminished concentration. Traditionally, Morita Therapy, a unique psychotherapy originated in Japan was devised for treating classical neurotic disorder.^{5,9,10} That concept has evolved the phenomenology of insomniacs as a subjective fabricated nature, claiming that clinicians are liable to make an error by just giving hypnotics to help the patient’s feeling of sleeplessness without attempting radical cure on him.¹⁰

In this case, theoretically we applied some conventional concepts of Morita therapy to the treatment, utilizing the latest

home monitoring instruments. We have to understand the fundamental phenomenology of diminished quality of sleep, and then give feedback to the acute insomniacs themselves in an appropriate way. To explain this process in the Morita theory, we attempted to stop exacerbating “psychic interaction” of acute insomniacs.^{10,11} This method of feedback may have something in common with the current well-developing biofeedback and mindfulness-based cognitive behavioral therapy for insomnia.¹¹ Despite their nature of subjective-objective discrepancy,^{5,6} individuals suffering from acute insomnia are situated under a subjectively perceived overwhelming threat.

We may stress that focusing on how sleep state misperception could be a central aspect of insomnia within the context of OCD. From this case study, it appears that the treatment was largely successful because the actigraphic records helped to correct the patient’s misperceptions. Overestimation of performance deficits may also be explained by psychophysiological hyperarousal model of insomnia.^{6,8} Perhaps such a focused intervention has a nonspecific and positive psychotherapeutic effect. This could also have implications about the possible application of actigraphy to treat sleep problems within anxiety disorders.

While actigraphy has been used in research studies for many years, methodological issues had not been systematically addressed in clinical research and practice.¹² A home monitoring system, such as actigraphy, not only provides satisfactory objective evaluation, but also a supportive psychotherapeutic effect in diminishing fear and anxiety related with acute insomnia. Getting an individual to recognize at an early stage, and providing him with treatment pathway guided by actigraph to deal with, were crucial in this case.

REFERENCES

1. American Academy of Sleep Medicine. *International classification of sleep disorders, 2nd ed.: diagnostic and coding manual*. Westchester, IL: American Academy of Sleep Medicine, 2005.
2. Angst J, Gamma A, Endrass J, et al. Obsessive-compulsive severity spectrum in the community: prevalence, comorbidity, and course. *Eur Arch Psychiatry Clin Neurosci* 2004;254:156-64.
3. Insel TR, Gillin JC, Moore A, et al. The sleep of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry* 1982;39:1372-7.
4. Goblin A, Kravitz H, Keith L, eds. *Sleep psychiatry*. London: Taylor & Francis, 2004.
5. Endo S. The psychophysiological study of neurotic insomnia. *Psychiatria et Neurologia Japonica* 1962;64:673-707.
6. Orff HJ, Drummond SP, Nowakowski S, et al. Discrepancy between subjective symptomatology and objective neuropsychological performance in insomnia. *Sleep* 2007;30:1205-11.
7. Ellis JG, Gehrman P, Espie CA, et al. Acute insomnia: current conceptualizations and future directions. *Sleep Med Rev* 2011 <http://dx.doi.org/10.1016/j.smrv.2011.02.002>.
8. Basta M, Chrousos GP, Vela-Bueno A, et al. Chronic insomnia and stress system. *Sleep Med Clin* 2007;2:279-91.
9. Gibson HB. Morita therapy and behaviour therapy. *Behav Res Ther* 1974;12:347-53.
10. Kora T. Morita therapy. *Int J Psychiatry* 1965;1:611-45.
11. Yamadera W, Itoh H, Ozono M, Ushijima S. Morita therapy and sleep hygiene. *Journal of Morita Therapy* 1999;10:107-15.
12. Ancoli-Israel S, Cole R, Alessi C, et al. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep* 2003;26:342-92.

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