

Scientia Psychiatrica

Journal Homepage: <u>www.scientiapsychiatrica.com</u>

eISSN (Online): 2715-9736

A Review on Insomnia: The Sleep Disorder

Atika Pusparani^{1*}

¹ Department of Neurology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

ARTICLE INFO

Keywords: Insomnia Insomnia severity index

Melatonin Treatment

*Corresponding author: Atika Pusparani

E-mail address: pusparaniatika@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/scipsy.v3i2.53

1. Introduction

Insomnia is the inability to acquire enough sleep to feel relaxed and rejuvenated when we wake up. Insomnia is defined by the quality of sleep and how we feel after sleeping, not by the number of hours we sleep or how quickly we doze off, because different people require various amounts of sleep. Even though we sleep for eight hours every night, if we remain drowsy and tired throughout the day, we may be suffering from insomnia.¹ Sleep issues are common among the elderly. While the prevalence of sleep problems is 20– 40% in the overall population, it reaches 50% in those aged 65 and up.²

According to studies conducted on general populations, roughly one-third of adults suffer from insomnia symptoms, with about 10% -15 percent reporting daytime impairments and 6% -10% experiencing symptoms of insomnia disorder, the most frequent of all sleep disorders. Although insomnia can be classified as a symptom or a separate disorder, it is

ABSTRACT

Insomnia is the inability to fall or stay asleep, even when one has the opportunity to do so. People who suffer from insomnia may be unsatisfied with their sleep and exhibit one or more of the following symptoms: weariness, low energy, problems concentrating, mood swings, and poor work performance. Insomnia can be classified based on how long it lasts. Acute insomnia is a type of insomnia that lasts only a few hours and is caused by a variety of factors. Many people have had this form of temporary sleep disruption, which usually goes away without medication. Chronic insomnia is defined as sleep disruption that lasts at least three months and occurs at least three evenings per week. Insomnia can be caused by a variety of factors. Insomnia is caused by a misalignment of sleep-inducing neurotransmitters. The Insomnia Severity Index and the Pittsburgh Sleep Quality Index are two methodologies and indices that have been used to research sleep disturbances in insomnia sufferers. Many medicines, such as antidepressants and benzodiazepines, are used in the treatment, and drinking Melatonin-rich milk has also been found to be useful.

most commonly thought of as a co-morbid condition that occurs in conjunction with another medical condition or mental problem.³

Seligman emphasized the prospect that a patient may believe that a specific treatment is advantageous, and that this perception may influence commitment and persistence to the treatment, resulting in a better outcome. Patients' willingness to seek treatment and the type of treatment they seek may also be influenced by folk ideas.4 Patients with insomnia had modest to moderate impairments in attention, episodic memory, working memory, and executive function when compared to healthy controls, according to largesample meta analyses.⁵

In general, insomnia refers to a lack of sleep quality or quantity despite having enough time to sleep. Insomnia, when characterized as a sleep disorder, is defined as a difficulty falling or staying asleep, which might include sleep maintenance issues or early morning awakenings despite attempts to sleep. As an insomnia complaint, a complaint of non-refreshing sleep may be included in the sleep disorder nosologies. Daytime repercussions or functional impairment must also be evident for an insomnia condition to be diagnosed. Fatigue, inability to concentrate, and irritation are some of these symptoms. Insomnia affects over 30% of the general population on a regular basis, and for about 10% of the population, it is a serious or chronic problem.⁶

Pathophysiology of the disease

Insomnia is caused by an imbalance between the neurotransmitters sleep-inducing gammaaminobutyric acid (GABA) and adenosine, which are found in the hypothalamic ventrolateral preoptic nucleus, and the arousal neurotransmitters (noradrenaline, serotonin, acetylcholine, orexin, and dopamine).7 Orexm, also known as hypocretin, is a neuropeptide produced by a cluster of neurons located in the lateral hypothalamus. It also appears to play a role in regulating wakefulness. Caffeine's sleepinducing action is assumed to be related to adenosine A2-receptor blockage. Specific brain areas with broad projections across the brain manufacture many of the chemicals involved in sleep-wake control. However, there is emerging evidence that many sleep regulating chemicals have a local effect on neurons in the locations where they are created. Sleep is described as a fundamental emergent property of strongly linked neurons, or cortical columns, according to Krueger et al's.⁸ local sleep theory.

The buildup of sleep-regulatory chemicals (tumor necrosis factor- and IL-1) arising from past neuronal usage is thought to be responsible for local sleep propensity and slow wave amplitude.^{9,10} Slow wave activity is thought to spread from cortical columns to adjacent regions via humoral and electric connections, eventually leading to a "global" sleep state in the entire organism. In this light, insomnia might not be a "whole-brain" issue (i.e, a simple matter of imbalance between global amounts of sleep and wake). An animal model of insomnia showed synchronous localized activity in both sleep-promoting and wake-promoting regions during global sleep.¹¹ In patients with insomnia, spectral EEG methods have identified increased regional electrical brain activity during nonrapid eye movement (NREM) sleep. 12,13 Merica et al 14 postulated that separate neuronal groups remaining active during PSG-defined sleep could explain the lack of objective sleep disruption in many insomnia patients. Many people who suffer from insomnia may mistake this brain dynamic for alertness, which is considered as "normal" sleep by traditional PSG standards.15 Symptoms of insomnia are consisted of general tiredness, problems with concentration or memory, difficulty falling asleep at night, sleepiness during the day, waking up during the night, waking up too early, after a night's sleep, do not feel properly rested, daytime tiredness or sleepiness, irritability, depression or anxiety, paying attention, focusing on duties, or remembering is difficult, increased errors or accidents, ongoing worries about sleep.

Cause of type -2 diabetes

Insomnia (difficulty falling or staying asleep) has been related to an increased risk of type 2 diabetes. Because there are probable biochemical pathways linking sleep deprivation to the development of type 2 diabetes via increases in insulin resistance and hunger, insomnia can be effectively treated and may be a feasible option for medicines to lower the prevalence of type 2 diabetes. Sleep issues may be linked to psychiatric distress and being overweight or obese, with each aggravating the other.

A neurology clinic enlisted patients with insomnia and volunteers. A clinical interview, laboratory blood tests, and a cognitive assessment were among the exams that the subjects underwent. Before the study, the participants signed consent forms, and the ethics committee approved the study procedure. All individuals were subjected to a thorough medical and neurological examination, as well as conventional laboratory testing and a battery of neuropsychological tests, which included the Pittsburgh Sleep Quality Index, Insomnia Severity Index, Hamilton Anxiety Scale, Hamilton Depression Rating Scale, Mini-Mental State Examination, Montreal Cognitive Assessment, and Clinical Dementia Rating. Patients with CID also underwent polysomnography.¹⁶ In a nutshell, a 1.5T superconductor MR imaging scanner was used to perform MR imaging. This study's MR imaging parameters and scanning mode can be found in a previously published study.¹⁷

Sleep-related scales Insomnia severity index

The ISI takes into account the severity of sleep start and maintenance problems, contentment with present sleep patterns, difficulty with daily functioning, the appearance of impairment ascribed to the sleep problem, and the level of anxiety induced by insomnia. The five-point Likert Scale is used to rate each item (0 = not at all, 4 = excessively). The score range is 0–28, and the total score is divided into three categories: 0– 7 indicates no clinically significant insomnia; 8–14 indicates sub-threshold insomnia; 15–21 indicates moderately severe clinical insomnia; and 21–28 indicates severe clinical insomnia.

Pittsburgh sleep quality Index

The PSQI assesses sleep quality and disruptions in the past. Individual self-report items examine a wide range of sleep quality dimensions, including sleep wake patterns, sleep duration, sleep latency, the frequency and severity of specific sleep-related issues, and the perceived impact of poor sleep on daily performance. This index is made up of item scores ranging from 0 to 3. The following is a breakdown of the overall score: Mild depression is defined as ages 10-15; moderate depression is defined as ages 16–23; and severe depression is defined as ages 24–63. Insomnia is a common symptom of depression and other psychiatric conditions. As a result, the subject's depressive symptoms were assessed.¹⁸

They looked at sleep investigations done on 106 chronic insomnia patients at the Singapore General Hospital's multidisciplinary sleep disorder unit (SDU) between 2006 and 2010. The PSG indices and diagnoses of patients were tabulated and compared to medical records to determine why sleep tests were performed. The efficacy of focused PSG in the evaluation of chronic insomnia was investigated by comparing pre and post PSG diagnosis. This study excluded patients who were referred for examination of their insomnia due to suspected primary sleep disorders or other causes. This allowed us to see if the referring doctors missed any underlying sleep abnormalities in chronic insomnia patients during the clinical history and physical examination.

The sleep investigations were carried out in 12channel attended PSG suites, and scores were assigned using the Recht schaffen and Kales criteria. Patients undergoing PSG were advised not to take any psychoactive medicines or hypnotics, as these could impair sleep physiology and cause false results. If the patients couldn't sleep, zolpidem was administered because it has the fewest side effects in terms of sleep architecture and muscle tone.

The unit, which is staffed by psychiatrists, physicians, neurologists, respiratory otolaryngologists, clinical psychologists, polysomnographic technologists, and respiratory therapists, accepts referrals from community primary care physicians and other medical professionals (e.g. psychiatrists from psychiatric hospitals without PSG facilities), as well as self-referrals. The results of this study can represent the management of insomnia patients in a real-world situation because the patients in this study had completed standard examinations and treatment. The hospital's institutional review board gave its approval to the study. The study was approved by the hospital's institutional review board, and consent was not required because no direct patient interaction was required.

Treatment

Pharmacological therapy of insomnia

Some antidepressants, such as trazodone, are particularly effective at treating insomnia and anxiety. When you need an insomnia drug that stays in your system longer, try Emazepam and other older sleeping medications. For example, they have been shown to be useful in treating sleep disorders including sleepwalking and night terrors. However, these drugs can make you tired during the day and can lead to dependence, which means you may need to take the drug all of the time to sleep. Zolpidem is effective in assisting you in falling asleep, however some people wake up in the middle of the night. Anti-histamines make up the majority of these sleeping medicines. There is no evidence that they help with insomnia, and they can make you sleepy the next day. They're considered safe enough to sell without a prescription. However, if you're taking other prescriptions that contain antihistamines, such as cold or allergy meds, you might take too much.

Insomnia treatment with melatonin rich milk

Melatonin is a non-sedating hormone that regulates the sleep-wake cycle and is secreted by the pineal gland in the brain. Only duri ng the hours of darkness, from dusk to morning, is it synthesized and released. Melatonin in the blood inhibits awakening by keeping the body temperature low and producing sleep. In a research of older people with low sleep efficacy due to low blood melatonin levels, those who took 0.3 mg of melatonin had their serum melatonin levels stabilized and their sleep efficacy greatly enhanced.

There was no significant difference between groups who consumed 0.3 mg melatonin and those that consumed 3.0 mg melatonin, which is known to be the initiating pharmacological serum level. The group that took 3.0 mg melatonin had higher serum melatonin levels. As a result, taking more dosages of melatonin than necessary could result in unfavorable side effects. Another study found a significant increase in daytime activity in an aged population that received melatonin-rich milk having 10 times more melatonin (10–40 ng/L, 0.5 L) than regular milk, without a commensurate increase in serum melatonin or a decrease in core body temperature. As a result, it was found that higher sleep quality caused by melatoninrich milk consumption resulted in increased daytime activity.

2. Conclusion

Insomnia disrupts cognitive and physical functioning and is linked to a variety of decreased daytime functions in the emotional, social, and physical realms. Persons with persistent sleep problems are more likely to be involved in accidents, have greater rates of work absenteeism, lower job performance, lower quality of life, and need more health care than people who sleep well. To properly analyze the causes and mechanisms of insomnia, studies that expand our understanding of the neurobiological systems controlling sleep homeostasis, circadian rhythms, physiological hyperarousal, genetics, stress, and cognition are essential. Accurate neurobehavioral and neurobiological data is required for effective pharmaceutical and behavioral therapies to treat insomnia. Because there are several approaches for identifying insomnia, it is possible to gain a better understanding of the patient's symptoms and treatment There potential options. are pharmacological treatments available, and drinking melatonin-rich milk before bedtime may help people recover from their illness.

3. References

- Ellis JJ, Hampson SE, Cropley MM. Sleep hygiene or compensatory sleep practices: An examination of behaviours affecting sleep in older adults. Psychology, Health & Medicine. 2002; 7(2): 156-161.
- Agargun MY, Kara H, Anlar O. Validation and reliability of Pittsburgh sleep quality index. Turkish Journal of Pyschiatry. 1996; 7(2): 107-115.
- Abdel Khalek AM. Prevalence of reported insomnia and its consequences in a survey of 5, 044 adolescents in Kuwait. Sleep. 2004; 27(4): 726-731.
- 4. Daley M, Morin CM, Le Blanc M, Gregoire JP, et al. The economic burden of insomnia: direct and

indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. Sleep. 2009; 32(1): 55-64.

- Lim AS, Kowgier M, Yu L, et al. Sleep fragmentation and the risk of incident Alzheimer's disease and cognitive decline in older persons. Sleep. 2013; 36: 1027-32.
- Michael JT. American Association of Sleep Disorders (eds.) The International Classification of Sleep Disorders: diagnostic and coding manual. 2nd edition. American Association of Sleep Medicine, Westchester, Illinois, 2005.
- Arya SN, Rajiv K, Singh R. Practical Approach to the Diagnosis and Management of Insomnia, Ch114, Sec16, 519.
- Krueger JM, Rector DM, Roy S, Van Dongen HP, et al. Sleep as a fundamental property of neuronal assemblies. Nat Rev Neurosci. 2008; 9(12): 910-919.
- Yoshida H, Peterfi Z, Garcia F, et al. Statespecific asymmetries in EEG slow wave activity induced by local application of TNF alpha. Brain Res. 2004; 1009 (1-2): 129-136.
- 10.Yasuda T, Yoshida H, Garcia-Garcia F, et al. Interleukin-1beta has a role in cerebral cortical state- dependent electroencephalographic slow-wave activity. Sleep. 2005; 28(2): 177-184.
- 11.Cano G, Mochizuki T, Saper CB. Neural circuitry of stress-induced insomnia in rats. J Neurosci. 2008; 28(40): 10167-10184.
- 12.Merica H, Blois R, Gaillard JM. Spectral characteristics of sleep EEG in chronic insomnia. Eur J Neurosci. 1998; 10(5): 1826-1834.
- Perlis ML, Merica H, Smith MT, Giles DE. Beta EEG activity and insomnia. Sleep Med Rev. 2001; 5(5): 363-374.
- Borbely AA. Secrets of Sleep. New York, NY: Basic Books, Inc, 1986.
- 15.Buysse DJ, Germain A, Hall M, et al. A neurobiological model of insomnia. Drug Discov Today Dis Models. 2011; 8(4): 129-137.

- 16.Mander BA, Marks SM, Vogel JW, et al. Amyloid disrupts human NREM slow waves and related hippocampus-dependent memory consolidation. Nat Neuroscience. 2015; 18: 1051-57.
- 17.Kao CC, Huang CJ, Wang MY, et al. Insomnia: prevalence and its impact on excessive daytime sleepiness and psychological well-being in the adult Taiwanese population. Qual Life Res. 2008; 17: 1073-80.
- Hui DS. Craniofacial profile assessment in patients with obstructive sleep apnoea. Sleep. 2009; 32:11.