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THE CLINICAL SPECTRUM OF EXCESSIVE SLEEPINESS: NEUROBIOLOGICAL MECHANISMS, ASSESSMENT, AND TREATMENT- A NARRATIVE REVIEW

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ABSTRACT

Hypersomnolence disorders constitute a heterogeneous group of conditions characterized by excessive daytime sleepiness (EDS), impaired alertness, and reduced quality of life. These disorders represent a significant medical and social challenge due to their impact on cognitive functioning, occupational performance, interpersonal relationships, and the increased risk of accidents. The regulation of sleep and wakefulness is a complex process involving interactions between circadian and homeostatic mechanisms, as well as numerous neurotransmitter systems, including orexin, dopamine, serotonin, histamine, acetylcholine, and gamma-aminobutyric acid (GABA). Advances in polysomnography, neuroimaging, and neurophysiology have improved understanding of the mechanisms underlying sleep regulation and central disorders of hypersomnolence.

This review summarizes the physiological basis of sleep, current methods used in the assessment of excessive sleepiness, and the clinical characteristics of the major hypersomnolence disorders. Particular attention is given to narcolepsy type 1 and type 2, idiopathic hypersomnia, Kleine–Levin syndrome, and hypersomnia associated with neurological, psychiatric, metabolic, endocrine, inflammatory, vascular, genetic, and traumatic conditions. Diagnostic approaches, including sleep questionnaires, actigraphy, polysomnography, and the Multiple Sleep Latency Test, are discussed in the context of differential diagnosis.

The article also reviews current therapeutic strategies, emphasizing both non-pharmacological interventions, such as sleep hygiene, scheduled naps, and behavioral modifications, and pharmacological treatments including modafinil, armodafinil, stimulants, sodium oxybate, pitolisant, and emerging therapeutic approaches targeting the orexin system. Early recognition of hypersomnolence disorders and appropriate management are essential for improving patient outcomes, reducing disease burden, and enhancing overall quality of life.

KEYWORDS

Hypersomnia, Excessive Daytime Sleepiness, Narcolepsy, Idiopathic Hypersomnia, Sleep Disorders, Sleep Physiology, Orexin, Polysomnography, Sleep Medicine

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Introduction

Sleep is considered a physiological state characterized by the cessation of motor activity, a typical posture, and reduced responsiveness to stimuli. It occurs spontaneously and periodically. In a standard polysomnogram, core parameters are recorded, including: the brain's bioelectrical activity (EEG), electrooculogram (EOG), and the recording of muscle bioelectrical activity (EMG). Based on these, it has been determined that sleep consists of two states: REM sleep (rapid eye movement) and NREM sleep (non rapid eye movement). These states alternate, and NREM sleep consists of 4 stages. As NREM sleep deepens, there is a progressive decline in skeletal muscle tone. During REM, in contrast to wakefulness, EOG shows rapid eye movements, while EMG registers muscle atonia. This does not affect only the inner ear muscles, respiratory muscles, and extraocular muscles. In healthy adult patients, sleep begins with NREM sleep. This state constitutes 75-80% of the main daily sleep period, and REM sleep—20-25%. One such cycle lasts about 90-110 minutes [1].

Mapping of the brain's bioelectrical activity and metabolic neuroimaging of the brain in recent years have made it possible to assess brain function during sleep. Brain activity during sleep is diverse and dependent on topography. During NREM sleep, a decrease in synaptic metabolism is observed compared to wakefulness or the REM phase of sleep. This primarily concerns the brainstem, thalamus, and basal forebrain—structures that sustain wakefulness. Additionally, it has also been recorded in subcortical nuclei, the cerebellum, and in the frontal, parietal, and temporal cortical regions. The greatest decrease in activity is found in the prefrontal cortex, mainly within the orbital gyri, and in the inferior parietal lobule. Interestingly, areas responsible for preliminary stimulus processing remain active. This distribution is characteristic of sleep in general and

appears as early as falling asleep, maintaining continuity throughout the entire NREM/REM sleep cycle. The heterogeneity of cortical activity allows for a rapid return to wakefulness. At this time, significant stimuli are processed, while less significant stimuli do not cause awakening. The feeling of refreshment after sleep is due to deactivation of the anterior cingulate gyrus and anterior insula, as well as the simultaneous independence of vegetative and endocrine functions during slow-wave sleep from stimuli originating in the environment. Dreaming and the emotional involvement during dreams are associated with activation of the limbic system in the REM phase (especially increased activity of the tegmentum and thalamus, as well as limbic and paralimbic areas). [1] The alternating occurrence of wakefulness and sleep is governed by centers extending from the medulla oblongata to the forebrain. Their maintenance is mediated by synaptic transmitters such as: noradrenaline, serotonin, dopamine, acetylcholine, histamine, GABA, glutamates, glycine, and orexins. [1], [9]

The ability to stay awake and the sense of sleep need exhibit circadian fluctuations. The generator of these rhythms (the biological clock) is the suprachiasmatic nuclei located in the anterior hypothalamus. They manage sleep timing via the circadian mechanism and regulate a constant amount of sleep through the homeostatic mechanism. The homeostatic mechanism increases sleep propensity after prolonged wakefulness. With extended wakefulness, adenosine accumulates, which activates the VLPO, responsible for shutting down the systems maintaining wakefulness. Hence the action of caffeine and theophylline, which are adenosine antagonists, promoting arousal and suppressing sleep. [1]

Assesment

Subjective assessment

Clinical evaluation of patients begins with a subjective assessment of their sleepiness. Many sleep questionnaires are used to assess subjective sleepiness. The most commonly used is the Epworth Sleepiness Scale, which offers a maximum score of 24 and estimates the likelihood of falling asleep in eight different scenarios. Other assessment tools include the Pittsburgh Sleep Quality Index (PSQI), Athens Insomnia Scale (AIS), Barcelona Sleepiness Index, Stanford Sleepiness Scale, Karolinska Sleepiness Scale, Daytime Sleepiness Scale, Leeds Sleep Evaluation Questionnaire, STOP-BANG scale, and Berlin Questionnaire. [3]

Clinical evaluation

Subjective assessment of sleepiness should be supplemented with a comprehensive sleep history and appropriate examinations. The patient's sleep and wake rhythms should be evaluated using a sleep diary. Objective assessment of circadian rhythms can be conducted using actigraphy. Actigraphy involves monitoring periods of activity and inactivity over a 2–4 week period to provide an overview of sleep and wake times and determine whether the patient has a delayed or advanced sleep phase rhythm.

Objective assessment of sleepiness

The Multiple Sleep Latency Test (MSLT) is a type of polysomnogram that can help identify patients with narcolepsy. In this test, the patient is allowed to fall asleep during five scheduled naps. The sleep-onset REM period is recorded if REM sleep appears within 15 minutes of falling asleep. The presence of two or more SOREMs along with an average sleep-onset latency of eight minutes or less is diagnostic for narcolepsy. The MSLT is preceded by a baseline sleep study, during which the patient is allowed to sleep to ensure that sleep deprivation or other sleep disorders do not affect the results of the five naps. It is also important to evaluate the patients' sleep-wake rhythm over the previous one to two weeks, preferably using actigraphy. If a SOREM occurred during the preceding night, it may be included toward the total of two SOREMs required for diagnosis. [11]

Clinical Spectrum

Narcolepsy type 1

The pathological basis of narcolepsy type 1 is the loss of hypocretin-1 (also known as orexin-A)–producing neurons in the hypothalamus. In addition to excessive daytime sleepiness, another important feature is REM sleep dissociation. About 90% of these patients have low or undetectable hypocretin-1 levels in the cerebrospinal fluid (CSF). Cataplexy is a transient state of decreased muscle tone, triggered by emotional stimuli, and represents the most specific REM sleep disorder observed in narcolepsy. Cataplexy does not always occur in all patients with type 1 narcolepsy and is closely related to low hypocretin levels in CSF. A significant number of patients with type 1 narcolepsy also experience nocturnal sleep disturbances. Although not characteristic, hypnagogic and hypnopompic hallucinations may also occur together with sleep paralysis. Increased rates of obesity, other sleep disorders, and coexisting psychiatric illnesses are also noted. Narcolepsy with cataplexy is closely linked to HLA subtypes. [4]

This significant association led to the hypothesis that autoimmunity is a likely causative mechanism, with great potential to explain the selective damage to hypothalamic neurons. If the HLA test result is negative, the level of hypocretin in the cerebrospinal fluid is most likely normal. The usual age of onset is between 10 and 25 years. Sleepiness is typically the first symptom, while cataplexy usually appears within a year of symptom onset. Secondary narcolepsy may also occur in the course of inflammatory diseases such as encephalitis, as well as tumors or other lesions of the hypothalamus. [10]

Narcolepsy type 2

Patients with narcolepsy type 2 (NT 2) do not have cataplexy and account for about 25% of the narcolepsy population. This disorder shares many features with NT 1. The exact cause of this disorder is unknown. Here as well, the genetic and environmental factors underlying NT 2 in patients are unknown. It typically appears during adolescence. Cataplexy develops later in the course of the disease in about 10% of these patients, at which point the disease should be reclassified as NT 1. [4] Similarly, a low level of hypocretin in the cerebrospinal fluid (<110 pg/ml or $<1/3$ of the mean population level) should prompt reclassification of the disease as NT 1. Both types of narcolepsy are characterized by early onset of REM phase after sleep onset. [7] The REM sleep phase (SOREM) is defined as REM sleep that begins within 15 minutes of falling asleep. In patients suffering from narcolepsy, at least two SOREM episodes should be recorded as part of MSLT/nocturnal PSG examinations. [10]

Idiopathic hypersomnia

Idiopathic hypersomnia is also characterized by EDS syndrome, but without cataplexy and without REM sleep attacks. The most typical phenotype includes prolonged nocturnal sleep episodes, which are not restorative, drowsiness, and daytime naps that are typically long and unrefreshing. Other patients with a less specific course resemble patients with narcolepsy type 2, except for the absence of early REM sleep. Recently, it has been suggested that these two groups should be classified together as a “narcolepsy spectrum disorder,” but this terminology has not been included in any formal classification. [12]

Kleine–Levin syndrome

This is a rare sleep disorder, usually seen in adolescent males. It is characterized by alternating periods of recurrent and remitting hypersomnia, cognitive impairment, perceptual disturbances, eating disorders (hyperphagia or anorexia), low mood, and disinhibition, including hypersexuality. The first episode most commonly appears after an infection or excessive alcohol intake. Episodes may last from 2 days to 5 weeks and occur at least once every 18 months, with periods of normal cognitive functioning and sleepiness between episodes. During episodes, patients may sleep up to 20 hours a day, waking only to eat and urinate. The disorder often resolves spontaneously by age 30. Sometimes a similar syndrome is also observed in the context of the menstrual cycle and is considered a variant of Kleine–Levin syndrome. [2]

Hypersomnia due to a medical disorder – sleepiness despite adequate treatment of obstructive sleep apnea

Sleepiness in obstructive sleep apnea (OSA) is usually attributed to chronic sleep deprivation caused by sleep fragmentation. For the same reason, sleepiness is expected to resolve when OSA is properly treated. However, a subset of patients remains sleepy despite adequate treatment. The exact mechanism of this condition is unclear, although animal studies have shown that intermittent nocturnal hypoxia may lead to irreversible damage to neuronal regions involved in the regulation of sleep and wakefulness. [8]

Posttraumatic hypersomnia

Hypersomnia can be a complication of head injury in up to 27% of individuals. In the acute phase of moderate to severe traumatic brain injury, hypocretin levels in the cerebrospinal fluid may also be low. Imaging studies usually show no significant changes, but sometimes they may reveal lesions in the hypothalamus and brainstem.

Hypersomnia in neurodegenerative diseases

Excessive sleepiness affects 16–50% of patients with Parkinson's disease and may occur in up to 25% of patients with multiple system atrophy. Similarly, other dementias, spinocerebellar degeneration, and Huntington's disease are associated with hypersomnia. [8]

Hypersomnia in genetic diseases

Muscular dystrophy, particularly myotonic dystrophy in adults, can cause excessive sleepiness in up to about 33% of patients. Other genetic disorders associated with primary sleepiness of the central nervous system include Niemann–Pick disease type C, Norrie disease, Prader–Willi syndrome, Smith–Magenis syndrome, Moebius syndrome, and fragile X syndrome.

Hypersomnia in inflammatory, vascular, and neoplastic processes

Cerebrovascular disorders, infections, tumors, and other inflammatory diseases can also cause sleepiness, especially when the hypothalamus or paramedian midbrain is involved.

Hypersomnia in endocrinological and metabolic disorders

The most common endocrinological cause of excessive sleepiness, especially in women, is hypothyroidism, which affects one in three women. Hepatic encephalopathy, adrenal and pancreatic insufficiency, chronic renal failure may be associated with sleepiness. Possible causes of fatigue and sleepiness also include iron and vitamin D deficiency, but comprehensive characterization of objective measures of sleepiness and alertness is lacking. [8]

Hypersomnia in psychiatric disorders

Depression and central hypersomnia share many common symptoms. The prevalence of depressive symptoms in narcolepsy type I ranges from 15% to 37%, while in IH it ranges from 15% to 25%. For the same reason, the prevalence of hypersomnia in major depressive disorder (MDD) occurs in over 70% of adult patients. Hypersomnia symptoms may range from 23% to 78% among patients with depression in the course of bipolar disorder (BD).

Excessive sleepiness is one of the main features of seasonal affective disorder and occurs in up to 70% of patients. Patients with MDD experience shortened REM sleep latency, increased REM density and duration, which may be related to increased cholinergic activity of the central nervous system. Patients with BD may experience circadian rhythm disturbances, especially delayed sleep phase. Stimulants should be used with caution, as there is an increased risk of psychotic symptoms at higher doses of these medications. Preclinical data suggest that H3 receptor antagonists may alleviate depression symptoms. Sodium oxybate may induce or worsen symptoms of depression. [11]

Treatment

Treatment of hypersomnia should primarily target the underlying cause, if one exists. Before initiating therapy, all factors negatively affecting sleep quantity and quality should be eliminated. Therefore, treatment of hypersomnia is both pharmacological and non-pharmacological. [5]

Non-pharmacological methods include: – proper sleep hygiene, regular daytime naps, regular physical activity, behavioral therapy, and distraction techniques.

Sleep Hygiene

This includes any actions and practices that improve sleep quality, including but not limited to avoiding stimulants such as caffeine and nicotine, avoiding heavy meals and alcohol before bedtime, ensuring quiet and comfort in the sleeping environment, fresh air, and maintaining a regular sleep schedule. [8]

Scheduled Naps

It has been proven that they reduce daytime sleepiness without negatively affecting nighttime sleep quality. Naps should last about 15–20 minutes, no longer. Extended naps carry a risk of disorientation. Scheduled naps are very effective in the treatment of narcolepsy (although they are rarely sufficient on their own), but have not been well studied in other types of hypersomnia.

Physical Activity

It has been shown in animal models that physical activity improves wakefulness in narcolepsy.

Distraction Techniques

Distraction techniques such as chewing gum have moderate effectiveness in promoting wakefulness. [11]

Pharmacological Therapies

Originally, treatment of narcolepsy included methods such as the use of bromides, strychnine, and amyl nitrite. Later, the stimulant effect of caffeine was discovered. Among more invasive approaches, attempts were made to help by hypothalamic X-ray irradiation, intrathecal air injection, and removal of cerebrospinal fluid. Currently, the most commonly used drugs in treatment of EDS share one feature: they enhance dopaminergic tone. Modafinil and armodafinil have milder effects and are often the drugs of choice at the beginning of treatment. They act on the dopamine transporter (DAT) and prevent dopamine reuptake. Headache and nausea are the most common adverse effects. [9] Amphetamine salts (methylphenidate, dextroamphetamine, etc.) are significantly stronger and more effective in treating EDS, unfortunately with significant addictive potential and greater side effects. They cause the release of noradrenaline and dopamine in the synaptic cleft and act on DAT, preventing their reuptake. [10]

Sodium oxybate (the sodium salt of gamma-hydroxybutyrate (GHB)) is a GABAB receptor agonist and is approved for the treatment of both daytime sleepiness and cataplexy. It promotes slow-wave sleep. The mechanism of action is not fully understood. Pitolisant is an inverse agonist of the H3 receptor. Histaminergic H3 receptors inhibit autoreceptors and are abundant in the central nervous system. Pitolisant increases histamine release in the cortex and hypothalamus. Studies have shown its effectiveness in the treatment of EDS as well as cataplexy, and it was better tolerated than modafinil. Flumazenil is an antagonist of the benzodiazepine binding domain on GABAA receptors. [12] It has been studied in patients with idiopathic hypersomnia and has been shown to improve psychomotor and subjective alertness. Clarithromycin is another negative allosteric modulator of the GABAA receptor and demonstrates subjective improvement in sleepiness. Levothyroxine has also proven effective in patients with IH in improving ESS.

Caffeine is the most commonly used stimulant in the world and can be used to treat hypersomnia. Higher doses of caffeine have comparable efficacy in treating EDS as modafinil but are limited by side effects. [5]

Other currently investigated treatment methods include intranasal orexin for type I narcolepsy, immunologically based treatment of narcolepsy, prostaglandin D2 receptor antagonists, and thyrotropin-releasing hormone.

Conclusions

In summary, hypersomnia is a state of reduced alertness that affects daytime functioning and negatively impacts quality of life. Excessive sleepiness is a social problem associated with numerous physical and mental diseases. It carries a risk of complications, traffic accidents, poorer functioning of patients in their daily lives, and consequently, emotional disturbances, difficulties in relationships, and lower work performance. The level of alertness depends on circadian and homeostatic processes, which in turn operate through sleep-promoting and arousal systems. Any imbalance in these two opposing neural systems leads either to reduced alertness and increased sleepiness or to difficulty falling asleep and insomnia.

In cases of hypersomnia caused by sleep disorders, hormonal or metabolic disorders, as well as chronic sleep deprivation, addressing the underlying cause is usually sufficient to alleviate sleepiness. On the other hand, in disorders such as narcolepsy, which result from orexin deficiency, treatment options are limited to symptomatic improvement of sleepiness. A thorough medical history together with a good understanding of hypersomnolence disorders are essential for identifying these patients and providing them with appropriate care.

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