The Correlation between Obsessive-compulsive Disorder, Schizophrenia and Sleep Disorder

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Abstract. Under the influence of the quick development of today's society, mental and psychological disorders, becoming a serious global public health problem. Approximately one billion people worldwide suffer from mental disorders. Obsessive-compulsive disorder and schizophrenia are two common types, often accompanied by sleep disorders. The two are mutually causal, but there are relatively few related studies. This article, by reviewing relevant literature from the past 3 to 5 years, sorts out the correlations among obsessive-compulsive disorder, schizophrenia and sleep disorders. Among patients with obsessive-compulsive disorder, 40% to 70% are accompanied by sleep disorders. Common types include delayed sleep phase syndrome (with an incidence rate of 17% to 42%), abnormal sleep structure (increased N1 stage, decreased N3 stage, and reduced efficiency), and REM sleep disorders (shortened latency and increased density) Anxiety sensitivity, excessive activation of the prefrontal cortex - cingulate gyrus - amygdala network, and circadian rhythm disorder are the mechanisms by which the two influence each other. In addition, over 80% of schizophrenia patients have significant sleep abnormalities throughout the entire process. The sleep characteristics of patients vary at different stages such as the prodromal stage, acute stage, and chronic stage. This includes multiple influencing mechanisms such as neurotransmitter disorders (excessive activation of dopamine, insufficient function of serotonin and GABA), abnormal brain region functions (defects in the prefrontal cortex, disorders in the thalamic-cortical pathway), circadian rhythm gene abnormalities, as well as secondary interference from antipsychotic drugs and poor treatment compliance.

Keywords: Obsessive-compulsive disorder, schizophrenia, sleep disorder.

1. Introduction

Mental disorders include obsessive-compulsive disorder, depression, anxiety disorder and so on. In today's society, whether at work or in study, people are all facing social pressure. Academic performance and achievements seem to have become the only measurement criteria. Under such pressure, it has transformed into psychological and mental stress, which has now become a serious global public health problem. GBD showed that mental disorders remained among the top ten leading causes of burden worldwide [1]. It can be seen from this that mental illness should not be ignored, especially obsessive-compulsive disorder and schizophrenia.

Obsessive-compulsive disorder (OCD) is a common mental illness. In fact, it is a kind of mental disorder that is easily misunderstood by non-professionals, perhaps because everyone has a preference for keeping things neat and orderly. But true obsessive-compulsive disorder sufferers are those who have persistent and intrusive obsessive thoughts or highly purposeful compulsive behaviors that strictly follow a series of rules repeatedly due to the uncertainty. The latest research shows that its prevalence rate among the general population is 1% to 3%, and it is on the rise. The simplest example is in the movie "The Aviator". Howard Hughes, a famous American aerospace engineer, was a patient with obsessive-compulsive disorder. Afraid of coming into contact with bacteria, he washed his hands over and over again with soap. Even if he didn't want to continue this behavior, no matter how hard he tried, he couldn't eliminate the ideas that invaded his mind. This is the characteristic of obsessive-compulsive disorder.

Schizophrenia is a very serious mental disorder that can even affect people's lives, impacting approximately 24 million people worldwide, or one in every 300 [2]. Although it is not as common as many other mental disorders, it is currently facing human rights violations in many health

institutions and social environments and thus cannot be ignored. Common symptoms include confused thinking, persistent fantasies, and extremely disordered behavior, etc. The onset of the disease is most common in late adolescence and people in their twenties, and the onset in men often occurs earlier than that in women. Schizophrenia is often closely associated with significant suffering in personal, family, social, educational, occupational and other important areas of life [2].

No matter what kind of mental and neurological disorder it is, it may be accompanied by sleep disorders. A variety of factors may lead to sleep disorders. For instance, patients with obsessive-compulsive disorder not only feel anxious, uneasy and distressed, but also experience various sleep problems due to anxiety. Patients with schizophrenia often suffer from insomnia, circadian rhythm disorders and narcolepsy. Sleep not only affects the quality of each day but also becomes even more important and challenging under the influence of mental illness. Under this premise, it is also a precursor to mental illness, that is, mental illness can also be affected by sleep. The two are mutually causal. However, there are relatively few studies elaborating on the association between the two types of mental and neurological disorders and sleep disorders. Therefore, this article, by consulting relevant literature in recent years, systematically sorts out the correlation between two common mental disorders and sleep. In order to provide some theoretical reference basis for future treatment.

2. The Correlation between OCD and Sleep Disorder

Previous research on OCD has shown that there is a close relationship between specific sleep behaviors and symptom characteristics, such as the severity of OCD symptoms, treatment resistance, and the age of onset of the disease. Among them, the most common types are sleep phase lag syndrome in insomnia and poor sleep quality (low subjective sleep satisfaction), followed by abnormal sleep structure, such as REM sleep disorder.

Sleep disorders are very common in obsessive-compulsive disorder, accounting for as high as 40% to 70%, significantly higher than the 10% to 15% in the general population [3]. Moreover, sleep problems are not only "accompanying symptoms" of OCD but may also be involved in the maintenance and deterioration of symptoms, forming a vicious cycle. Previous research on obsessive-compulsive disorder (OCD) has shown that there is a close relationship between specific sleep behaviors and symptom characteristics, such as the severity of OCD symptoms, treatment resistance, and the age of onset of the disease. Among them, the most common type is sleep phase shift syndrome in insomnia, followed by abnormal sleep structure and REM sleep disorder, etc.

Delayed sleep phase syndrome is mostly a disorder of sleep between day and night. Its symptoms are that the start and wake times of sleep are uncontrollably later than the ideal situation, that is, the time to fall asleep each day is basically the same, but once asleep, it is not easy to wake up, and the sleep phase cannot be advanced. The incidence of this symptom in OCD is 17% to 42%, which is also significantly higher than the 0.2% to 10% in the general population [4]. Additionally, for OCD patients who do not have sleep phase lag syndrome, when they have this symptom, the OCD-related symptoms are more serve and the age of onset is younger. The possible reason is that 70% of OCD patients suffer from daytime sleepiness and inattention. Insufficient sleep leads to fatigue, and fatigue in turn reduces control over obsessive-compulsive symptoms, creating a vicious cycle.

Abnormal sleep structure is also common in obsessive-compulsive disorder, such as an increased proportion in N1 stage (light sleep) (averaging 20%-25% vs). Among healthy people, 15%-20% have a reduced proportion in N3 stage (deep sleep) (equal 15%-20% vs. 20%-25%), indicating insufficient sleep depth. The final research results only showed significant findings in the non-rapid eye movement stage, with obsessive-compulsive disorder spending significantly less time in stage 2 sleep. Sleep efficiency declines, with an average of 60% to 70% (over 85% in healthy individuals), and is negatively correlated with the frequency of compulsive behaviors ($r=-0.43$, $p<0.01$) [5]. If compulsive behaviors (such as repeated hand washing) have coercive characteristics, patients must strictly follow a fixed procedure (such as washing hands 10 times).

The latency of REM sleep is shortened (average < 60 minutes, 90-120 minutes for healthy people), and the REM density (the number of eye movements per unit time) increases, leading to frequent dreams and easy awakening. A systematic review published in the Journal of Sleep Research comprehensively analyzed 17 studies to explore the occurrence of sleep disorders in patients with OCD. The results show that adult patients with OCD generally have delayed circadian rhythm disorder (DSWPD) and insomnia, which are characterized by difficulty falling asleep, sleep maintenance disorders and decreased sleep quality, and the delay of circadian rhythm is related to the severity of OCD symptoms. Children with OCD also show significant sleep problems, including reduced sleep duration, increased nocturnal awakenings, nightmares and sleepwalking, etc. Sleep problems are positively correlated with the severity of OCD [6]. Meanwhile, the REM latency of OCD patients is significantly shortened, and there is a correlation between the severity of obsessive-compulsive disorder and the shortening of REM latency, that is, the more severe the condition, the shorter the REM latency. This discovery not only quantifies the damage that OCD causes to sleep structure, but also reveals the close connection between the severity of the disease and sleep abnormalities, providing an important empirical basis for subsequent exploration of the mechanism by which OCD affects sleep. The reason for the shortened REM latency may be related to the dysfunction of the neurotransmitter system (such as serotonin) in the brain of OCD patients. Serotonin is involved in both sleep regulation and emotion control, further explaining the internal logic of the interweaving of OCD symptoms and sleep disorders.

Anxiety is a common unpleasant emotional state that is easily triggered by potentially threatening things, mainly manifested as tension, unease and worry. The "high anxiety state" caused by the core symptoms of OCD will increase physiological arousal (such as accelerated heart rate and elevated cortisol levels), while sleep requires a "decrease in physiological arousal". The conflict between the two leads to difficulty falling asleep. Anxiety sensitivity (AS) is a common risk factor for both, reflecting a fear tendency towards physical sensations related to anxiety arousal. It consists of one higher-order factor (AS) and three lower-order factors, respectively, the fear of the physical, social, and cognitive consequences of coping with anxiety. Among them, the cognitive concern sub-scale of AS has a unique connection with the unacceptable thought domain in obsessive-compulsive disorder (such as sex, religion or aggressiveness related). In addition to the relationship between AS cognitive problems and OC symptoms, the study also found a significant association between AS cognitive problems and insomnia symptoms. The relationship between AS and sleep-related disorders was studied in a sample of patients with chronic insomnia, and it was found that the cognitive concern dimension of AS was related to sleep-related disorders. It can be seen from this that obsessive-compulsive symptoms may influence an individual's insomnia behavior through the mediating effect of anxiety [7].

Overactivation of the prefrontal cortex - cingulate gyrus - amygdala network is a typical feature of OCD. This network is also involved in "sleep-wake regulation". Overactivation of the prefrontal cortex may inhibit sleep initiation, while overactivation of the amygdala enhances nocturnal alertness [8]. It can also to some extent indicate that there is a common connection between the two. In addition, circadian rhythm disorder is also one of the key influencing factors. For instance, the peak secretion of melatonin in OCD patients is delayed (with an average delay of 1-2 hours) , leading to a "backward shift of the biological clock", which conflicts with social routines and exacerbates difficulty in falling asleep [9]. A clinical controlled study also fully confirmed this point. Compared with the normal general population, the Pittsburgh Sleep Quality Index (PSQI) score of OCD patients was significantly higher (8-12 vs. <5 points), especially with more awakenings at night (an average of 3-4 times per night).

3. The Correlation between Schizophrenia and Sleep Disorder

The global prevalence of schizophrenia is approximately 0.7% to 1% [10]. Epidemiological investigations show that over 80% of schizophrenia patients have significant sleep disorders, and

these sleep disorders may persist throughout the entire course of the disease, from the prodromal stage to the chronic stage. Another meta-analysis found that the overall incidence of sleep disorders among schizophrenia patients was 70% to 90%, significantly higher than that in the general population (10% to 15%) [11]. Delayed sleep phase syndrome accounts for approximately 40%. In addition, research shows that compared with other mental disorders (such as depression), sleep problems in patients with schizophrenia are more likely to be accompanied by abnormal perceptions (such as bedtime auditory hallucinations causing difficulty falling asleep) and bedtime thoughts related to cognitive disorders, such as uncontrollable associations.

On the one hand, the core symptoms of schizophrenia, such as hallucinations, delusions and cognitive disorders, can disrupt the sleep structure. On the other hand, long-term sleep disorders such as insomnia and fragmented sleep may exacerbate psychotic symptoms and increase the risk of schizophrenia recurrence by 2 to 3 times.

It is worth noting that sleep disorders are dynamically associated with the course of schizophrenia and have different characteristic manifestations at different stages of the disease. During the prodromal stage, abnormal sleep often serves as an early warning signal of disease onset. Research shows that approximately 60% of patients will experience insomnia 1-2 years before the appearance of psychotic symptoms, with difficulty falling asleep being particularly prominent. At the same time, sleep efficiency will also drop below 70%. At this stage, Polysomnography (PSG) can detect a reduction in deep sleep (N3 stage) [12]. After entering the acute stage, the degree of sleep disorders is the most severe. 85% of patients will experience sleepless nights or fragmented sleep, such as a single sleep duration of less than 2 hours. Moreover, the sleep problems at this time are positively correlated with the severity of psychotic symptoms such as hallucinations and delusions. For instance, auditory hallucinations were significantly correlated with the insomnia complaints, the sleep duration and also the ease of getting up [13]. In the chronic stage, sleep problems are characterized by persistent poor sleep quality. The average Pittsburgh Sleep Quality Index (PSQI) score of patients reaches 10-14 points (usually less than 5 points for healthy people), which is specifically manifested as prolonged sleep latency (more than 60 minutes) and excessive daytime sleepiness due to insufficient sleep at night.

The impact of schizophrenia on sleep is mainly manifested as abnormalities in the sleep-wake regulatory network, involving multiple aspects such as neurotransmitters, brain region functions, and core genes of circadian rhythms. Among them, neurotransmitter disorder is one of the important mechanisms. Excessive activation of the dopamine system, a core pathological feature of schizophrenia, can inhibit sleep initiation (especially affecting the N3 stage), thereby leading to difficulty falling asleep and reduced deep sleep. Meanwhile, the insufficiency of serotonin and gamma-aminobutyric acid (GABA) functions can also have adverse effects, being unable to participate in maintaining sleep stability and exerting central inhibitory effects, thereby exacerbating sleep fragmentation and nocturnal awakening [14]. Abnormal brain region functions should not be ignored either. The prefrontal cortex is involved in the "active regulation of the sleep-wake cycle"[15]. When its function is defective, patients will be unable to maintain their sleep rhythm independently, such as having difficulty suppressing abnormal thoughts before sleep. The thalamus is the "sensory information filtering center". If the thalamic-cortical pathway is disrupted, it will interfere with falling asleep, causing patients to have "excessive sensory input" before sleep, such as being overly sensitive to slight sounds and light [16]. In addition, abnormalities in the core genes of the circadian rhythm are also relatively common in patients with schizophrenia. Abnormal expression of the Clock gene and Per2 gene can cause a delay in the peak secretion of melatonin (with an average delay of 3-4 hours) and an extension of the circadian rhythm cycle (>25 hours), directly leading to sleep phase disorders.

The evidence-based impact of schizophrenia on sleep can be clearly presented through the subjective reports of patients and the objective data of polysomnography (PSG), both of which jointly quantify the degree of sleep disruption caused by the disease. In terms of the relevant sleep indicators reported subjectively, the patient's abnormal manifestations are relatively prominent: 65% of the

patients reported taking 1 to 2 hours to fall asleep each day, and 70% reported having difficulty falling asleep again after waking up at night. Moreover, the patients' "subjective satisfaction" with sleep was significantly lower than that of healthy people, with an average Visual Analogue Scale (VAS) score of only 30 to 40 points, while healthy people usually score 70 to 80 points [17]. In addition, 80% of patients experience daytime fatigue and inattentiveness due to insufficient sleep, and the decline in cognitive function, in turn, aggravates psychotic symptoms, such as making it more difficult to distinguish between reality and hallucinations. From the objective monitoring data of PSG, the changes in sleep indicators are equally significant: the sleep structure shows a fragmented feature, and the sleep efficiency is significantly reduced, averaging only 50%-60% (while it exceeds 85% in healthy people). The average number of awakenings at night is 6-8 times, and the proportion of N1 stage (light sleep) is as high as 40%-50%, which is much higher than the 15%-20% in healthy people. Deep sleep (N3 stage) is severely absent, with an average proportion of only 5% to 8%, and this indicator is positively correlated with cognitive decline (such as memory decline) ($r=0.46$, $p<0.01$). There are also abnormalities in REM sleep. In the acute stage, the REM latency is shortened (less than 60 minutes, while in healthy people it is 90-120 minutes), and the REM density (frequency of eye movement) increases. In contrast, in the chronic stage, the REM latency may be prolonged (exceeding 180 minutes). This change indicates the chronicity of the disease [18].

Unlike other mental disorders, drug treatment in schizophrenia may also cause secondary disturbances to sleep. Among them, the impact of antipsychotic drugs is dual: first-generation antipsychotic drugs (such as the haloperidiol), due to their strong dopamine-blocking effect, may cause restlessness (manifested as subjective anxiety), thereby exacerbating the patient's difficulty in falling asleep. Second-generation antipsychotic drugs (such as quetiapine and olanzapine) can improve sleep in the short term due to their sedative effects, but long-term use may lead to abnormal sleep structure, such as reduced REM sleep and daytime sleepiness [19]. At the same time, the chain reaction caused by poor treatment compliance cannot be ignored. If patients stop taking their medication on their own, the recurrence of psychotic symptoms will directly lead to the deterioration of sleep conditions, and the decline in sleep quality will further reduce the patients' medication compliance, thus forming a vicious cycle [20].

In conclusion, the impact of schizophrenia on sleep involves multiple dimensions, such as neurotransmitter disorders (such as excessive activation of dopamine), cognitively behavioral abnormalities (such as hallucinations and pre-sleep intensification), and drug effects, which in turn disrupt sleep structure (reduced deep sleep, fragmentation) and circadian rhythms. Moreover, sleep abnormalities can exacerbate psychotic symptoms and cognitive impairment.

4. Conclusion

Mental illness has become a major global public health issue, with obsessive-compulsive disorder and schizophrenia being particularly prominent. And these two diseases are often accompanied by sleep disorders. Its correlation is based on a shared neurobiological matrix and complex psychological behavioral mechanisms. Sleep disorders play multiple roles in the course of these two diseases: they are not only potential risk factors and prodromal markers, but also comorbidity symptoms reflecting the severity of the diseases. Moreover, they are key factors that induce and exacerbate core mental symptoms, hinder treatment responses, and predict poor prognosis. Therefore, in clinical practice, the impact of sleep disorders on diseases needs to be considered and effectively managed.

However, current research also has certain limitations. First, there are many studies on the association between obsessive-compulsive disorder and schizophrenia and sleep disorders respectively, but there is a lack of comparison between the two in terms of abnormal sleep characteristics and influencing mechanisms, making it difficult to reveal the specificity of the association. Second, the exploration of mechanisms mostly focuses on single factors such as neurotransmitters and brain region functions, while there is insufficient research on the interaction of multiple factors (such as the synergistic impact of anxiety sensitivity and circadian rhythm disorders)；

Thirdly, there are relatively few intervention studies on sleep disorders associated with the two diseases, especially a lack of long-term follow-up data, making it difficult to clearly define the specific role of improving sleep in the prognosis of the diseases.

Future research can be advanced from three aspects: conducting comparative studies of the two diseases to clarify the differences in their manifestations and mechanisms of sleep disorders, integrating multidisciplinary techniques to deeply explore the molecular pathways of multi-factor interactions, designing targeted intervention trials to evaluate the impact of sleep improvement measures on disease symptoms, and verifying the causal relationship between the two through long-term cohort studies. Provide more accurate basis for clinical treatment.

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