Original Article
Changes in sleep architecture and quality in minimal hepatic encephalopathy patients and relationship to psychological dysfunction

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Abstract: Objectives: We examined changes in sleep quality and architecture in patients with minimal hepatic encephalopathy (MHE) and the impacts of sleep disruption on patient physical and psychological health. Methods: Ninety-eight MHE patients were examined by polysomnography (PSG) and the Pittsburg sleep quality inventory (PSQI). In addition, patients completed the SAS, SDS, and SCL-90 to examine the relationship between sleep quality and psychological health. Results: Mean relative durations of Stage 1 and Stage 2, sleep latency, microarousal frequency, and total sleep time (TST) were all lower in MHE patients compared to healthy controls (P<0.05 for all). Similarly, SWS and REM stage durations, REM latency, sleep maintenance rate, and sleep efficiency were lower than controls (P<0.01 for all). Mean PSQI scores were lower in MHE patients. Total SAS, SDS, and SCL-90 scores, as well as all SCL-90 subscores, were significantly higher in the MHE group (P<0.05), indicating significant psychological dysfunction. Longer SWS, longer REM, and lower microarousal frequency were associated with improved sleep quality (P<0.05), while shorter SWS and REM led to dyssomnia and daytime functional disturbance (P<0.05, P<0.01). Longer REM latency and higher microarousal frequency were associated with higher PSQI scores (P<0.05, P<0.01), while longer SWS, longer REM, and higher sleep maintenance rate were associated with lower PSQI scores (P<0.05, P<0.01). Finally, total PSQI score and sleep efficiency subscore were positively correlated with total SCL-90 and most SCL-90 subscores (P<0.05). Conclusions: MHE patients suffer from multiple subjective dyssomnias and changes in sleep architecture that are strongly correlated with psychological dysfunction.

Keywords: Minimal hepatic encephalopathy sleep architecture, pittsburgh sleep quality index, psychological symptoms

Introduction

Minimal hepatic encephalopathy (MHE) refers to the mild encephalopathy afflicting patients with chronic liver disease that shows none of the obvious clinical manifestations of hepatic encephalopathy (HE), including abnormal blood chemistry, and that is not within the scope of HE according to the Four-Level classification. Rather, MHE can only be diagnosed by subtle intellectual and mental state examinations and/or electrophysiological tests. According to published reports from 1991-2003, the incidence of MHE in hepatic cirrhosis patients ranges from 38.8%-85.0% in China [2] and from 30%-84% internationally [3]. Chronic liver disease and hepatic encephalopathy are almost always accompanied by dyssomnia. Sleep can be divided into non-rapid eye move-ment (NREM) and rapid eye movement (REM) sleep, and NREM can be further divided into four stages (Stages 1-4). The pattern of repeated NREM-REM cycles constitutes the sleep architecture [4]. Bajaj [5] report highlighting an association between loss of slow-wave sleep and reduced levels of ghrelin in a small group of cirrhotic patients with MHE, a recent study comparing the polysomnographic characteristics of patients with obstructive sleep-apnea with and without cirrhosis [6]. To date, no clinical reports have investigated whether there is a correlation between sleep architecture and sleep quality and psychological dimensions or psychiatric symptoms. We conducted subjective sleep quality surveys, polysomnography, and a battery of psychological tests to investigate changes in sleep quality and architecture in MHE patients and the relationship between these

changes and psychological dimensions/symptoms. Identification of MHE-specific changes in sleep architecture and associated psychiatric symptoms may reveal pathogenic mechanisms and possible strategies for prevention and treatment of MHE.

Materials and methods

Study groups

The MHE group: The diagnosis of MHE was made according to the recommendation by the working party of 11th world congress of gastroenterology [7]. The neurological manifestations of MHE are can be detected by the number connection test (NCT) and the digit symbol test (DST). The NCT measures the time required by the subject to connect the numbers 1-25 in sequence on a randomly dispersed number array (including the time for the rectification of errors). In the DST, the subject is presented with 9 digit-symbol pairs and from memory must write down the symbol corresponding to the digit as quickly as possible. A score on the NCT at least two standard deviations higher than controls or a DST score two or more standard deviations lower than the control average is a sign of neurological dysfunction.

In the present study, the MHE group consisted of 98 cases, 57 males and 41 females, ranging in age from 30-65 (average, 46.3±9.3). Routine tests for serum bilirubin concentration, blood albumin concentration, and prothrombin time were conducted. We also determined whether the patient suffered from abdominal dropsy and hepatic encephalopathy and the severity of both. The five different indexes were scored as 1, 2, or 3 according to severity. From the total score, three overall degrees of MHE severity, A, B, and C, were defined. The minimum score of the five indexes is 5, and the maximum score is 15 according to the Child-Push improved classification system. According to this classification, 43 cases were Level A and 55 cases were Level B. Elimination criteria were severe cardiopulmonary diseases, renal inadequacy, diabetes, a pre-existing neuropsychiatric disease, ethylism, or an inability to comprehend the self-report inventories. In addition, patients taking ammonia decreasing medications or branched-chain amino acids (BCAAs), antibiotics, sleep-promoting agents, or other medications were also excluded. Finally, pregnant women, women in lactation, and other patients with poor compliance were eliminated.

Control group: The control group consisted of 20 healthy volunteers with no chronic diseases and included 12 males and 8 females ranging in age from 30-60 (average, 47.6±8.3). The A-E hepatitis virus scores were all negative and no abnormities were detected on pretest examinations. Blood biochemical indicators of liver function were all normal. No control subject had a history of alcoholism and all consumed less than 40 g per week. No control subject had been on any medication for the month prior to testing.

There were no significant differences in age, sex ratio, or pre-existing disease conditions (except MHE) between the two groups. The study was approved by our hospital and the Ethics Committee of HongKou WeiSheng. All research subjects gave informed consent by voluntarily signing or press their fingerprint on the consent form.

Polysomnography

Polysomnography was conducted using a WanTai PSG system and included EEG, an eye movement electrode, a snore electrode, breast and abdominal breathing monitor, submaxilla myoelectricity electrode, mouth and nose airflow monitor, electrocardio-electrode, leg movement electrode, and blood oxygen electrode, as well as a body position monitor. Subjects were acclimated to the sleep clinic environment to reduce anxiety and ensure adequate sleep. Subjects avoided naps on the day of examination and abstained from tea, coffee, chocolate, colas, alcohol, and other drinks that affect sleep. All men shave and all subjects showered before PSG.

Analysis of sleep architecture

Sleep stage classification was conducted according to the International Human Sleep Staging Criteria established by Rechtschaffen-Kales Sleep architecture parameters measured were the percentage of time spent in Stages 1, 2, 3 and 4 (Stage 3 and Stage 4 are both regarded as SWS and treated as a single statistical unit), the percentage time spent in REM, the REM latency, the total REM duration, and the sleep latency. Sleep parameters included the total monitoring time, total sleep time, total

Table 1. Differences in sleep architecture between healthy controls and MHE patients

	Control group	MHE group
Stage 1 (%)	8.75±1.67	12.37±2.58*
Stage 2 (%)	48.63±11.37	57.21±21.76*
SWS (%)	21.52±3.65	13.67±6.27**
REM stage (%)	21.1±4.17	16.75±8.35*
Sleep latency (min)	15.24±2.58	35.78±9.26**
REM latency (h)	1.12±0.38	0.97±0.33*
Microarousal frequency	16.28±3.27	30.62±7.32**
Total sleep time (h)	6.4±0.8	8.6±1.2**
Total awareness time (min)	27.7±6.3	59.1±8.7**
Total monitoring time (h)	7.6±1.2	8..0±2.1
Sleep maintenance rate (%)	97.5±1.39	87.7±5.23*
Sleep efficiency (%)	88.36±2.65	79.56±3.57**

*$P<0.05$, **$P<0.01$ compared to the control group.

Table 2. Comparisons of PSQI item scores between groups

	MHE	Control	Z	P
Sleep quality	1.45±0.68	0.91±0.52	-3.826	0.000
Time to fall asleep	1.75±0.83	1.06±0.64	-3.571	0.000
Hours of sleep	1.06±0.97	0.67±0.57	-1.753	0.067
Sleep efficiency	0.76±0.89	0.17±0.0.21	-4.012	0.000
Dyssomnia	1.57±0.76	0.95±0.55	-4.312	0.000
Hypnotics	0.28±0.33	0.11±0.32	-1.146	0.073
Daytime functional disturbances	1.13±0.73	0.62±0.57	-3.183	0.000
Total scores	8.03±4.32	4.35±2.18	-4.724	0.000

awareness time, sleep latency, microarousal frequency, sleep efficiency, and sleep maintenance rate. Each parameter was measured automatically by the PSG system.

The evaluation of subjective sleep quality

The Pittsburg sleep quality index (PSQI) probes seven aspects of sleep [8]: overall sleep quality, time to fall asleep, hours of sleep, sleep efficiency, dyssomnia, use of hypnotics, and daytime functional disturbances. Each component is scored 0-3 for a total score from 0-21. A higher total score is indicative of poorer sleep quality.

Psychological dimensions and mental health

The SAS, SDS, and SCL-90 psychological inventories were used to evaluate the psychological health of the patients and controls. The SCL-90 contains 90 items that probe 10 psychological dimensions: somatization, compelling symptoms, sensitivity in interpersonal relationship,

depression, anxiety, hostility, phobophobia, bigotry, and psychosis. Each item is scored from 0 to 4 and higher scores are indicative of maladaptive behaviors or poor mental health.

Statistical analysis

The statistical software package SPSS 16.0 was used to conduct tests of normality for each variable. Variables with a normal distribution are expressed as mean ± SD, and variables with a non-normal distribution are expressed by the median and range. Means of normally distributed data sets were compared by t-test or Wilcoxon rank-sum test. Correlations were tested using Spearman correlation analysis. Analysis of variance was used to compare multiple group means from normally distributed data sets. The SNK-q test was adopted for pair-wise comparisons between data with homogeneity of variance while Dunnet's T3 was used for pair-wise comparisons of means with heterogeneity of variance. The Mann-Whitney U test was used to conduct comparisons between two non-parametric group means while the Kruskal-Wallis H test was used to compare multiple non-parametric group means. A $P<0.05$ was regarded as statistically significant.

Results

Changes in architecture in MHE patients

There was no age or sex ratio difference between the control group and the MHE group ($P>0.05$). In the MHE group, the proportion of Stage 1 and Stage 2 sleep was higher than in the normal group ($P<0.05$), while the proportions of SWS and REM sleep were lower ($P<0.01$). Sleep latency was longer in the MHE patients compared to healthy controls ($P<0.01$), but REM latency was shorter ($P<0.05$). Microarousal frequency was higher in MHE patients and total monitoring time longer than

Table 3. The comparisons of SCL-90 score between MHE and Control groups

	MHE	Control group	Z	P
Total scores	51.61±37.86	28.18±0.52	-3.869	0.000
Somatization	31.54±20.87	21.28±17.69	-3.278	0.001
Compelling symptoms	1.57±0.77	1.35±0.32	-1.357	0.082
Sensitivity in interpersonal relationships	0.61±0.58	0.41±0.24	-2.156	0.048
Depression	0.57±0.52	0.35±0.39	-3.321	0.001
Anxiety	0.59±0.43	0.28±0.38	-4. 416	0.000
Hostility	0.53±0.37	0.22±0.46	-2.141	0.037
Phobophobia	0.36±0.42	0.15±0.13	-3. 247	0.001
Bigotry	0.42±0.47	0.29±0.32	-1.337	0.158
Psychosis	0.45±0.37	0.21±0.31	-2.280	0.025
Others	0.68±0.0.47	0.35±0.0.41	-3.761	0.000

Table 4. Comparison of SAS and SDS scores between MHE and control groups

	MHE	Control group	t	P
SAS	53.64±12.88	39.17±9.52	6.275	0.000
SDS	45.66±11.25	37.78±9.67	3. 723	0.027

in the control group (P<0.01). Both sleep maintenance rate and sleep efficiency were lower in the MHE group (P<0.01). All sleep parameters measured by polysomnography in MHE patients and healthy controls are summarized in **Table 1**.

Analysis of subjective sleep quality using the PSQI

Except for total hours of sleep and use of hypnotic medications, all other scores on the PSQI were higher in the MHE group (P<0.05; **Table 2**).

Psychological dysfunction in MHE patients

Except for the "compelling symptom" factor and the "bigotry" factor, all subscores on the SCL-90 inventory were higher in the MHE group (**Table 3**), indicating maladaptive responses and signs of psychological dysfunction. Similarly, total scores on the SAS and SDS were significantly higher in MHE patients (SAS: P<0.05; SDS; P<0.01; **Table 4**).

Correlations between PSG sleep parameters, subjective sleep quality, and psychological dimension scores

The sleep quality subscore was correlated only with "compelling symptoms". There was a positive correlation between the time to fall asleep and the total score on the SCL-90, as well between time to fall asleep and the individual SCL-90 factor subscores "sensitivity in interpersonal relationships", "hostility", "phobophobia", and bigotry. There were positive correlations between all SCL-90 subscores except "compelling symptoms" and "bigotry" with both PSQI sleep quality score and total PSQI score (**Table 5**). There was no correlation between "Daytime functional disturbance" and any item on the SCL-90. There was a positive correlation between the total SAS score and sleep quality, time to fall asleep, sleep efficiency, dyssomnia, and total PSQI score. Finally, there was a positive correlation between the total SDS score and time to fall asleep, total sleep hours, sleep efficiency, and total PSQI score.

Correlations between PSG and PSQI in MHE patients

Increased SWS and REM stage durations and decreased microarousal frequency were associated with improved sleep quality (P<0.05). Stage 1, Stage 2, and total sleep time (TST) were longer, and sleep maintenance rate lower in patients with higher PSQI scores, while microarousal frequency was higher, total awareness times shorter, and the subjective hours of sleep higher in patients with higher PSQI scores (P<0.05, P<0.01). In contrast, longer REM sleep, higher sleep maintenance rate, and lower microarousal frequency correlated with better subjective sleep efficiency (P<0.05, P<0.01). In MHE patients, the longer sleep latency, shorter REM latency, higher microarousal frequency, and the lower proportions of SWS and REM sleep led to dyssomnia and day-

Table 5. Correlations between PSQI, SCL-90, and SAS/SDS scores in MHE patients

	Sleep quality	Time to fall asleep	Total sleep hours	Sleep efficiency	Dys-somnia	Hyp-notics	Daytime functional disturbance	PSOI total score
SCL-90 total score	0.187	0.268*	0.286*	0.385**	0.201	0.217	0.091	0.342**
Somatization	0.105	0.251	0.223	0.337**	0.135	0.296*	0.065	0.302*
Compelling symptoms	0.289*	0.112	0.217	0.124	0.223	0.120	0.137	0.168
Sensitive in interpersonal relationship	0.185	0.279*	0.235	0.367**	0.217	0.297*	0.047	0.359**
Depression	0.076	0.230	0.173	0.270*	0.156	0.185	0.126	0.271*
Anxiety	0.057	0.146	0.243	0.301*	0.127	0.225	0.231	0.296*
Hostility	0.198	0.256*	0.261*	0.358**	0.253	0.219	0.135	0.368**
Phobophobia	0.185	0.377**	0.230	0.287*	0.105	0.474**	0.021	0.383**
Bigotry	0.183	0.267*	0.373**	0.357**	0.083	0.018	0.003	0.243
Psychosis	0.172	0.209	0.224	0.392**	0.197	0.167	0.106	0.291*
Others	0.196	0.152	0.218	0.305*	0.100	0.143	0.073	0.241
SAS	0.357**	0.315*	0.217	0.424**	0.323**	0.152	0.253*	0.357**
SDS	0.243	0.378**	0.292*	0.440**	0.082	0.321*	0.128	0.439**

*P<0.05, **P<0.01.

time functional disturbances (P<0.05, P<0.01). The REM latency was shorter and the micro-arousal frequency higher in patients with higher total PSQI scores (P<0.05, P<0.01). Finally, the proportions of SWS and REM stage sleep as well as the sleep maintenance rate were higher in patients with lower total PSQI scores (P<0.05, P<0.01; **Table 6**).

Discussion

Sleep is a complex series of behaviors characterized by different stages that follow characteristic cyclical patterns in healthy individuals. These patterns are altered by illness, and the associated changes may provide insight into pathological processes or treatment strategies. Each sleep stage is associated with unique patterns of brain electrical activity, and the pattern of stages and their characteristics are referred to as sleep architecture. In patients with normal sleep architecture, the durations of Stage 1, Stage 2, SWS, and REM stage account for 5%-10%, 50%, 20%, and 20%-25% of the total sleep duration, respectively. Aside from rejuvenation, the NREM sleep component of the sleep cycle increases attention, the capacity for logical thinking, language, and foresight. It can also improve behavior flexibility in response to environmental changes and shorten response times. There is a strong correlation between REM stage sleep and memory because REM sleep can facilitate synaptic plasticity [4, 9]. Bajaj [5] found significant disruptions in sleep architecture. SWS occurred in all controls but not in 80% of MHE (P = 0.04). Median SWS time was lower (0% vs. 15%, P =

0.016) and REM time was higher (19% vs. 7%, P = 0.02) in MHE. This study found in the MHE group, the fractional times in Stage 1 and Stage 2 of NREM sleep were higher than in controls (P<0.05), while MHE patients spent proportionally less time in SWS and REM stage sleep (P<0.01). In addition, sleep latency was longer (P<0.01) and the REM latency shorter (P<0.05), while microarousal frequency, TST, and total monitoring time were all higher in MHE patients (P<0.01). Finally, sleep maintenance rate and sleep efficiency were lower compared to controls (P<0.01). The increase in Stage 1 and Stage 2 sleep and concomitant decrease in SWS sleep indicate that MHE patients suffer with deep dyssomnia, which interferes with the rejuvenating effects of sleep. The increased microarousal frequency, longer TST, higher total monitoring time, and decreased sleep maintenance rate, together with decreased sleep efficiency, are also indicative of poorer sleep quality. The characteristics of REM are relatively stable in healthy adults, but are altered markedly in patients with brain injuries. In this research, when the latency to REM stage was shorter, REM stage duration also decreased. This decrease in REM stage sleep can lead to mood instability, difficulty in concentration, and depression, which further exacerbate the physical effects of poor sleep. Sleep architecture disorder is a prominent characteristic of MHE that can induce further endocrine, neural, and cognitive dysfunction in patients.

Sleep architecture is controlled and regulated by multiple centers in the brainstem, thalamus, hypothalamus, and preoptic region [10]. REM

Table 6. Analysis of the correlation between PSQI and sleep architecture in MHE patients

	Sleep quality	Time to fall asleep	Hours of sleep	Sleep efficiency	Dyssomnia	Hypnotics	Daytime functional disturbance	Total PSQI score
Stage 1 (%)	0.156	0.258*	0.276*	0.076	0.221	0.272*	0.096	0.143
Stage 2 (%)	0.121	0.101	0.289*	0.107	0.153	0.268*	0.068	0.202
SWS (%)	0.252*	0.121	0.198	0.224	0.264*	0.126	-0.273*	-0.268*
REM stage (%)	0.267*	0.152	0.163	0.358**	0.271*	0.153	-0.361**	-0.327**
Sleep latency (min)	-0.026	-0.256*	0.132	0.106	0.256*	-0.306**	0.148	0.116
REM latency (h)	0.175	0.165	0.137	0.314**	0.268*	0.215	0.261*	0.258*
Microarousal frequency	-0.293*	0.174	-0.271*	-0.367**	0.264*	0.185	0.335*	0.315**
TST (h)	0.176	0.106	0.350**	0.139	0.197	0.465**	0.061	0.205
Total awareness time (min)	-0.181	0.152	-0.257*	-0.145	-0.138	-0.278*	0.083	0.217
Sleep maintenance rate (%)	0.157	0.089	0.284*	0.382**	0.207	0.267*	0.137	-0.263*
Sleep efficiency (%)	0.169	0.125	0.265*	0.202	0.253*	0.253*	0.173	0.235

*P<0.05, **P<0.01.

sleep is controlled principally by cholinergic neurons in the brainstem (mainly in the mesencephalon and pontine). Acetylcholine can depolarize inhibitory GABAergic neurons in the nucleus reticularis, suppressing rhythmic discharge, which induces asynchronous electric discharges in the cortex that manifest as low wave amplitudes on PSG. Noradrenergic neurons of the locus ceruleus, serotonergic neurons of the raphe nuclei, and histaminergic neurons in the posterior hypothalamus are deactivated [11], and this quiescence is also associated with the characteristic cortical activity, autonomic nerve activity, and disappearance of muscle tension during REM sleep. There is also a correlation between GABA and REM sleep. Experimental hepatic encephalopathy disrupts 5-HT, γ-aminobutyric acid, and catecholamine titers. The changes in REM stage in MHE patients indicate that there is a correlation between MHE-induced neuronal dysfunction and neurotransmitter changes. Significant decreases in blood flow in both the right and left hemispheres have been detected by SPECT in MHE patients, while the blood flow in the frontal cortex increased [12]. Thus, changes in REM sleep (and other aspects of sleep architecture) reflect brain injury induced by MHE.

As the analysis of sleep architecture requires specific sleep monitoring equipment, the prevalence of disrupted sleep architecture in MHE patients is difficult to estimate. For this reason, we conducted the more accessible PSQI survey in addition to PSG. Results confirmed that MHE patients suffer from poor sleep quality, take a longer time to fall asleep, exhibit a lower sleep efficiency, and show daytime functional disturbances, indicating that MHE patients suffer from multiple subjective dyssomnias. However, there were no significant differences in hours of sleep or hypnotic use compared to healthy controls, indicating chronic somnolence in these patients. Moreover, patients with liver diseases are generally not recommended hypnotics. The results are consistent with the PSG results; Stage 1 and Stage 2 sleep are increased by MHE while deep SWS is inhibited.

There was a strong correlation between many aspects of subjective sleep quality and PSG results in MHE patients, although some differences were noted. There was a positive correlation between subjective sleep quality and the durations of SWS and REM stage sleep, as well as a negative correlation between sleep quality and higher microarousal frequency (P<0.05). Thus SWS, REM sleep [4, 9], and low microarousal frequency effectively maintain normal sleep architecture and result in optimal energy recovery. Indeed, these three factors constitute the core elements of subjective sleep quality. The decrease in SWS and REM, sleep together with the increase in microarousal frequency, interfere with the normal progression and stage transition of the sleep cycle, leading to disordered sleep architecture and dyssomnia. Moreover, the ensuing daytime functional disturbance contribute to dissatisfaction as reported on subjective evaluations, increasing the total PSQI score (P<0.05, P<0.01). The evaluation of agrypnia resulted from the increase in microarousal frequency and total awareness time, and from the decrease in Stage 1, Stage

2, TST, and the sleep maintenance rate (P<0.05, P<0.01). Patients believed that sleep should be maintained for about eight hours with few disruptions and dreams [13], but PSG results demonstrated a correlation between poor sleep evaluation by MHE patients and the durations of Stage 1, Stage 2, and other Shallow sleep According to some reports, even healthy adults may show erroneous subjective evaluations of sleep that can even lead to subjective agrypnia [14].

Mild hepatic encephalopathy patients suffer from a variety of health problems that reduce quality of life [15] but there is no report on the psychological dysfunction in this patient group. The total score on the SCL-90 and the severity of "compelling symptoms" were significantly higher than in the control group, indicating poor psychological health in MHE patients. Furthermore, all item scores on the SCL-90 were higher in MHE patients, except for the "compelling factor" and the "bigotry" factor (P<0.05, P<0.01). Specifically, there were highly significant differences in the degrees of somatization, depression, anxiety, and phobophobia in MSE patients (P<0.01). Phobophobia and anxiety have a common psychological basis, so anxiety and depression were more common in MSE patients. Results from the SCL-90 were confirmed by the SAS and SDS. The main reasons for these psychological problems in MHE patients include fear of relapse, isolation, and financial stress [16]. Liver disease is prone to relapse and in some cases not easily treated. Mild HE can lead to serious complications such as hepatic cirrhosis and liver cancer, so patients may be pessimistic about their treatment and recovery prospects. Liver disease may induce discrimination, so patients may suffer from poor self-esteem, loneliness, and alienation from society. Chronic disease patients may have fewer opportunities to communicate with other people and may lack a social support network. Furthermore, social and family roles are altered by chronic illness. Finally, there is the economic burden of MSE treatment.

The coefficient of correlation between the total PSQI score and the total score on the SCL-90 was 0.342 (P<0.01), and there was a positive correlation between the PSQI sleep quality factor and severity of SCL-90 positive symptoms. In addition, there were positive correlations between the PSQI score and other symptoms of psychological dysfunction. In general, those patients with the most severe psychiatric symptoms had the worst sleep. Aside for anxiety and depression, other psychiatric factors negatively impacted the sleep quality of MHE patients, such as compelling symptoms, sensitivity in interpersonal relationships, hostility, and phobophobia.

These correlations suggest a vicious cycle; poor psychological health negatively affects sleep, and sleep problems lead to or exacerbate psychological health problems. It should be noted, however, that these correlation do not necessary imply causality. Although this research cannot explain the specific causal relationships between sleep and psychological factors in MHE patients, we do demonstrate that MHE patients suffer from dyssomnia and that dyssomnia is strongly correlated with psychiatric symptoms. Treatment of the psychological problems associated with MHE may improve sleep quality, which in turn may aid in recovery. Future studies will observe the sleep quality and architecture in MHE patients of different ages, genders, and neurological severity to provide greater insight into treatment strategies for psychological problems and dyssomnia in MHE patients.

Disclosure of conflict of interest

None.

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