Sleep Apnea and its Effect on Ascitic Patients

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ABSTRACT

Introduction: Obstructive sleep apnea syndrome has been recently described as a complication of ascites. This occurs due to elevated mechanical loads on the upper airway and defects in compensatory neuromuscular responses. Sleep disturbances, daytime hypersomnolence and neurocognitive dysfunction are common complications of cirrhosis and sleep apnea syndrome. The severity of these complications increases with the presence of both pathologies and improve markedly with the improvement of apnea after tapping of ascites. The aim of this study was to determine the type of apnea associated with ascites. The effect of apnea on the condition and the level of improvement in sleep disturbances and neurocognitive complications after tapping of ascites. Subjects and Methods: The study was conducted on 40 patients with chronic liver disease, divided into 3 groups according to the extent of ascites. Group I with tense ascites (n=20), Group II with mild to moderate ascites (n=10), Group III without ascites (n=10). Assessment was done through clinical examination, minimental state examination (MMSE), laboratory investigations, abdominal ultrasound and polysomnography. Results: Sleep efficiency and neurocognitive functions as measured by MMSE were significantly affected with the increased degree of ascites. Apnea was found to be of the obstructive type. Apnea hypopnea index (AHI) was clearly higher in group I than groups II and III. MMSE and sleep efficiency showed a statistically significant correlation with the presence of significant apnea. AHI, sleep efficiency and MMSE improved markedly after tapping of ascites. Conclusion: Obstructive sleep apnea syndrome (OSAS) can be a complication of tense ascites. OSAS causes more deterioration of sleep efficiency and neurocognitive functions and these findings improved after tapping of ascites. (Egypt J. Neurol. Psychiat. Neurosurg., 2008, 45(2): 459-468)

INTRODUCTION

Cirrhotic patients with hepatic encephalopathy have significant impairment of their daily activities. Sleep disturbances are commonly encountered in this clinical entity and are emerging as a sensitive sign of subclinical encephalopathy in cirrhotic patients¹.

Cirrhosis may also affect pulmonary functions and it might be involved in the development of sleep apnea syndrome in patients with ascites².

There are 2 types of sleep apnea syndrome (SAS): obstructive sleep apnea syndrome (OSAS) and central sleep apnea syndrome (CSAS). OSAS is a prevalent disorder while CSAS is much less common. The latter condition is common in patients with heart failure and cerebral neurologic diseases³.

There was a trial to determine if a low PaCO₂ is a predictor of CSAS in patients with cirrhosis of the liver and with normal left ventricular systolic function. The results of this study showed that hypocapnia does not predict CSAS in patients with cirrhosis, an observation that contrasts to that in patients with heart failure⁴.

OSAS is a highly prevalent disorder with significant clinical consequences including, fragmented sleep, daytime hypersomnolence and neurocognitive dysfunction. The major risk factors include obesity, male gender, age and craniofacial abnormalities as retrognathia or micrognathia⁵.

Current understanding of the pathophysiologic basis of the disorder suggests that a balance of anatomically imposed mechanical loads and compensatory neuromuscular responses are important for maintaining upper airway potency during sleep. OSAS develops in the presence of both elevated mechanical loads on the upper airway and defects in compensatory neuromuscular responses⁶.

OSAS was found to be associated with reduction in vigilance and attentional abilities being responsible for traffic or occupational accidents⁶.
**Aim of The Work**

To determine the type of apnea associated with ascites. The effect of apnea on the condition and the level of improvement in sleep disturbances and neurocognitive complications after tapping of ascites.

**PATIENTS AND METHODS**

This work was done in Ain Shams University Hospitals over 2 years from June 2005 to April 2007.

**Patients:**

Three groups of patients with chronic liver disease were the subject of this study. All the patients were matched as regards age and sex.  
**Group I:** Twenty (20) patients with tense ascites the age was ranging from 32 to 54 years. Fourteen were males and six were females.  
**Group II:** Ten (10) patients with mild to moderate ascites. The age was ranging from 28 to 50 years, seven were males and three were females.  
**Group III:** Ten (10) patients without ascites the age was ranging from 24 to 45 years six were males and four were females.

**Exclusion criteria:**

- Manifestations of hepatic encephalopathy.  
- Other causes of ascites.  
- Other causes of obstructive sleep apnea (e.g. cardiac, pulmonary or renal disease).  
- Other systemic diseases especially cardiac and pulmonary diseases, diabetes mellitus and hypertension.

**Methods:**

The patients were subjected to full history taking. Symptoms of chronic liver disease were asked upon as abdominal pain, dyspepsia, hematemesis, bleeding tendency, fatigue and loss of weight. Also symptoms of hepatic encephalopathy were enquired upon as tremors, sleep disturbances, irritability, amnesia, personality changes and drowsiness. General examination was done with special emphasis on general manifestations of liver cell failure as jaundice, spider naevi, foetor hepaticus, gynecomastia in males, palmar erythema and oedema of lower limbs to exclude patients with hepatic encephalopathy. Abdominal examination was done to determine the size of the liver and the spleen and to examine for the presence of ascites. Full medical neurological history and examination was done for all cases.

Ain Shams Sleep disorders questionnaire (a modified questionnaire of Akran general medical hospital and sleep disorders center of Lankenau hospital sleep questionnaire with special stress on history of apnea. Only cases with history of apnea or snoring were included in the study. Minimental state examination was done for all cases.

**Laboratory studies included:**

CBC, ESR, blood sugar level, serum Na and K, BUN and serum creatinine, liver function tests as s. bilirubin, s. albumin, AST, ALT, S.alkaline phosphatase and P.T. Abdominal ultrasound was done for all patients. Polysomnographic overnight study was done for all cases. For those patients in group 3 with significant apnea follow up polysomnography was done after tapping of ascites.

**Polysomnography (PSG):**

Polysomnography was performed for all patients. Overnight recordings were performed on 16-channel computerized EEG systems (SASSY, Telefactor Corp., West Conshohocken, PA), with split-screen video EEG in a bipolar chains using the international 10-20 system to monitor nocturnal EEG changes. Electrooculogram, submental EMG, nasal-oral airflow, respiratory effort (using intercostals EMG), pulse oximetry [Nellcor 2000], and anterior tibialis EMG were also recorded.

All patients came to the sleep laboratory of the Neurology Department, Ain Shams University, about two hours before their usual bedtime. This allow sufficient time for electrode application. The EEG electrodes are attached to the scalp using small patches of gauze soaked in collodion and dried with compressed air after adding the conducting media in the electrode cup.

The recording included four EEG channels (C3-O1, C3-O2, A1- C1 and A2- C2). Electro oculogram (EOG). Standard EOG placement include the right outer canthus (ROC) and the left outer canthus (LOC) electrodes, with one of them one centimeter above and the other is one centimeter below the outer canthi to record eye movements. Electromyogram (EMG) is monitored by two electrodes placed on and below the chin. Nasal-oral airflow is monitored by a sensor and
displayed in a separate channel, respiratory effort is monitored using intercostals EMG electrode. Pulse oximetry [Nellcor 2000] is used to monitor oxygen saturation changes. Anterior tibialis EMG were also recorded in a separate channel, body position and snoring assessment.

All PSG recordings were scored manually according to the standard manual for staging normal sleep to generate a report supplying us with data including parameters of sleep statistics, respiratory events, leg movements, and arousals.

Apneas were defined as >50% reduction in naso-oral airflow lasting >10 seconds. A hypopnea was scored when there was an obvious reduction, usually >20% in airflow and/or effort, lasting >10 seconds and accompanied by an arousal or a fall in SaO2 of >4%. The number of respiratory events per hour was defined as the apnea hypopnea index (AHI) or the respiratory distress index (RDI).

**Data processing and statistical analysis:**

Data collected were revised, coded, tabulated and introduced to PC for statistical analysis. All data manipulation and analysis were performed using the 11th version of SPSS (Statistical Package for Social Sciences). Qualitative data is presented in the form of frequency tables (numbers and percent), while quantitative data is presented in the form of mean ± standard deviation. The statistical tests used included: independent sample-T test, ANOVA test for more than two groups, and Wilcoxon signed Ranks test, and correlation coefficient test.

**RESULTS**

The study included 40 patients, 27 males and 13 females with a mean age of 41.02±5.88. Group I with severe ascites included 20 patients 14 males and 6 females with a mean age of 39.16±3.60. Group 2 with mild to moderate ascites included 10 patients 7 males and 3 females with a mean age of 43.7±5.81 and Group 3 without ascites included 6 males and 4 females with a mean age of 39.4±6.20 with no statistical difference between the 3 groups (Table 1).

Concerning the sleep efficiency, there was statistically significant correlation with the degree of ascites (f=6.85, p=0.001). Also the MMSE correlated significantly with the degree of ascites (f=10.452, p=0.000) (Table 2). MMSE correlated significantly with the decrease in sleep efficiency (f=0.760, p=0.000) (Table 3), apnea count (T=-0.740, p=0.000) (Table 4) and AHI (f=0.813, p=0.000) (Table 5).

The results show that the snoring episodes, the number of significant desaturations and apnea hypopnea index (AHI) were clearly more prevalent in Group I than Group II and III (AHI between groups f=2.895, p=0.048, apnea count between groups f=2.953, p=0.046 O2 desaturations f=11.534, p=0.000) (Table 6).

The sleep efficiency correlated significantly with all apnea parameters. An increase in AHI was associated with a decrease in sleep efficiency (Table 7).

On comparing the 6 cases of significant AHI with the 14 cases with insignificant AHI in Group I, it was found that the MMSE and the sleep efficiency showed a statistically significant correlation with the presence of significant apnea (Table 8).

Follow up of the patients with definite sleep apnea was done after abdominal paracentesis. The apnea count, AHI, oxygen desaturations was found to be markedly improved after paracentesis (Table 9) (Fig. 1a,b).

Also the sleep efficiency and the MMSE improved markedly after the paracentesis (Table 10).

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Sex</th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age</td>
<td>Male</td>
<td>Female</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>---------</td>
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<td></td>
</tr>
<tr>
<td>Group I</td>
<td>39.16±3.6</td>
<td>14</td>
<td>6</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>43.7±5.81</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Group III</td>
<td>39.4±6.203</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>41.025±5.876</td>
<td>27</td>
<td>13</td>
<td>40</td>
<td></td>
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</tbody>
</table>

Table (1): Age and sex distribution among different groups of patients.
**Table 2.** Impact of degree of ascites on sleep efficiency and MMSE.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sleep efficiency</th>
<th>MMSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (N=20)</td>
<td>56.505 ± 16.222</td>
<td>23.7 ± 2.867</td>
</tr>
<tr>
<td>Group II (N=10)</td>
<td>74.18 ± 12.463</td>
<td>25.9 ± 1.952</td>
</tr>
<tr>
<td>Group III (N=10)</td>
<td>75.340 ± 8.766</td>
<td>28.5 ± 1.08</td>
</tr>
<tr>
<td>F</td>
<td>6.850</td>
<td>10.452</td>
</tr>
<tr>
<td>P</td>
<td>0.001</td>
<td>0.000</td>
</tr>
</tbody>
</table>

P<0.05 → significant

**Table 3.** Impact of decrease of sleep efficiency on MMSE.

<table>
<thead>
<tr>
<th>Group</th>
<th>MMSE</th>
<th>Sleep efficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>23.7±2.867</td>
<td>56.50±16.222</td>
</tr>
<tr>
<td>Group II</td>
<td>25.9±1.952</td>
<td>74.18±12.463</td>
</tr>
<tr>
<td>Group III</td>
<td>28.5±1.08</td>
<td>75.34±8.706</td>
</tr>
<tr>
<td>T</td>
<td>0.760</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

P<0.05 → significant.

**Table 4.** Impact of increase in apnea count on MMSE.

<table>
<thead>
<tr>
<th>Group</th>
<th>MMSE</th>
<th>Apnea count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>23.7±2.867</td>
<td>47.25±61.33</td>
</tr>
<tr>
<td>Group II</td>
<td>25.9±1.952</td>
<td>7.83±3.54</td>
</tr>
<tr>
<td>Group III</td>
<td>28.5±1.08</td>
<td>3.7±1.56</td>
</tr>
<tr>
<td>T</td>
<td>0.740</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

P<0.05 → significant

**Table 5.** Impact of increase in AHI on MMSE.

<table>
<thead>
<tr>
<th>Group</th>
<th>MMSE</th>
<th>AHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>23.7±2.867</td>
<td>17.39±24.01</td>
</tr>
<tr>
<td>Group II</td>
<td>25.9±1.952</td>
<td>1.316±4.49</td>
</tr>
<tr>
<td>Group III</td>
<td>28.5±1.08</td>
<td>0.88±0.364</td>
</tr>
<tr>
<td>T</td>
<td>0.813</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>0.000</td>
<td></td>
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</tbody>
</table>

P<0.05 → significant.
Table 6. Impact of degree of ascites on all apnea parameters.

<table>
<thead>
<tr>
<th>Group</th>
<th>AHI</th>
<th>Apnea count</th>
<th>Oxygen saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>17.39±24.01</td>
<td>47.25±61.33</td>
<td>81.165±7.54</td>
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<tr>
<td>Group II</td>
<td>1.316±0.449</td>
<td>7.83±3.54</td>
<td>89.166±2.78</td>
</tr>
<tr>
<td>Group III</td>
<td>0.88±0.364</td>
<td>3.7±1.56</td>
<td>93.6±2.716</td>
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</table>

F = 2.895  
P = 0.048

P<0.05 → significant

Table 7. Impact of severity of apnea on sleep efficiency.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sleep efficiency</th>
<th>AHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>56.505±16.222</td>
<td>17.39±24.01</td>
</tr>
<tr>
<td>Group II</td>
<td>74.18±12.463</td>
<td>1.316±0.449</td>
</tr>
<tr>
<td>Group III</td>
<td>75.34±8.706</td>
<td>0.88±0.364</td>
</tr>
</tbody>
</table>

F = 0.78  
P = 0.000

Table 8. Effect of significant apnea on MMSE and sleep efficiency.

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Insignificant apnea n=14</th>
<th>Significant apnea n=6</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMSE</td>
<td>25.28±1.48</td>
<td>20.00±1.41</td>
<td>7.373</td>
<td>0.000</td>
</tr>
<tr>
<td>Sleep EFF</td>
<td>64.88±8.41</td>
<td>36.95±12.66</td>
<td>5.852</td>
<td>0.002</td>
</tr>
</tbody>
</table>

P<0.05 → significant.

Table 9. The effect of paracentesis on apnea count, AHI and oxygen desaturation.

<table>
<thead>
<tr>
<th></th>
<th>Apnea count</th>
<th>AHI</th>
<th>Oxygen saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparacentesis</td>
<td>128.66±53.88</td>
<td>52.11±11.31</td>
<td>74.65±10.51</td>
</tr>
<tr>
<td>Post paracentesis</td>
<td>16±3.27</td>
<td>5.56±1.57</td>
<td>90.48±1.79</td>
</tr>
</tbody>
</table>

Z = -2.201  
P = 0.028

P<0.05 → significant

Table 10. MMSE and sleep efficiency improvement after paracentesis.

<table>
<thead>
<tr>
<th></th>
<th>MMSE</th>
<th>Sleep off</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparacentesis</td>
<td>20±1.41</td>
<td>36.95±12.66</td>
</tr>
<tr>
<td>Postparacentesis</td>
<td>25±0.89</td>
<td>57.55±12.60</td>
</tr>
</tbody>
</table>

T = 0.949  
P = 0.004

P<0.05 → significant
Fig. (1a): A histogram of a male patient 41 years old with tense ascites, AHI is 64.9, apnea count is 181, and average oxygen saturation is 74.7.

Fig. (1b): A histogram of the same patient after tapping of ascites, AHI is 6.2, apnea count is 24, and average oxygen saturation is 92.
DISCUSSION

Sleep apnea syndrome (SAS), a common disorder, is characterized by repetitive episodes of cessation of breathing during sleep resulting in hypoxemia and sleep disruption. The consequences of the abnormal breathing during sleep include daytime sleepiness, neurocognitive dysfunction, development of cardiovascular disorders, metabolic dysfunction, and impaired quality of life.

Sleep disturbances and neurocognitive dysfunction are also classic signs of hepatic encephalopathy. There are limited data regarding its prevalence in patients with chronic liver disease without overt hepatic encephalopathy. Obstructive sleep apnea syndrome (OSAS) could give rise to similar symptoms to hepatic encephalopathy and has been previously assessed in cirrhotic patients in only few studies.

We studied patients with different stages of chronic liver disease with history of snoring with or without sleep apnea to find out if these patients had sleep apnea or not and the type of apnea present. Also we studied the effect of apnea on sleep and cognitive functions and whether or not this is corrected after tapping of ascites.

In this study group I of patients with severe ascites showed significant deterioration of sleep efficiency and neurocognitive functions in comparison to other groups.

These results go with those results found by Sherlock et al., who found out that a disturbance of sleep is recognized as one of the early signs of hepatic encephalopathy. Tarter et al. also stated that sleep disturbance was significantly higher in cirrhotic patients than other subjects with another chronic illness. Also Quero et al. found out that up to thirty five percent of cirrhotic individuals had difficulties in the area of sleep and rest. The mechanism responsible for these findings is poorly known. It could be due to an abnormality in circadian function or a delay in the increase in diurnal plasma melatonin and its peak nocturnal level.

Neurocognitive dysfunction has long been known to be associated with cirrhotic patients without evidence of encephalopathy thirty percent to seventy percent of cirrhotic patients show neuropsychological abnormalities. Memory and attention alterations and also impairment of non verbal intelligence were found to be the highest abnormalities in those patients.

Neurocognitive impairments are caused by several mechanisms. Such mechanisms include direct effects of the ammonium ion on excitatory and inhibitory neurotransmission and a deficit in cerebral energy metabolism due to ammonia-induced inhibition of alpha-ketoglutarate dehydrogenase. In addition, ammonia disrupts neuron astrocyte trafficking of amino-acids and monoamines in the brain.

Then we searched for the presence of apnea in these patients and its relation to sleep efficiency and neurocognitive functions.

Severe apnea (RDI>30) was found in 6 patients of group 1 (tense ascites). This group had a history of snoring ± apnea.

A RDI from 5 to 14 is considered as minimal OSASA, a RDI from 15 to 29 as mild OSAS, and a RDI at 30 or above as moderate to severe OSAS.

Apnea was found to be of the obstructive type. Obstructive sleep apnea is the periodic reduction (hypopnea) or cessation (apnea) of breathing due to the narrowing of the upper airways during sleep. These results go with Shahrokh et al. and Crespo et al. Shahrokh stated that low PaCO2 in patients with cirrhosis is not a predictor of SAS. While Crespo stated that OSAS is a potential complication in cirrhotic patients with high volume ascites. They suggested that the enlarged abdominal perimeter plays a role in the development of this ascites-related complications. Diaphragmatic elevation produces a decrease in functional residual capacity and perhaps modifies the upper airway system. Golpe et al. thought that the ascites induced diaphragmatic elevation can produce a decrease in functional residual capacity and this can induce oxygen desaturation because of ventilation perfusion dysmatch. However they thought that it is less probable that the enlarged abdominal perimeter can modify by an unknown mechanism the upper airway system and they suggested that the possible oedema of the pharyngeal and laryngeal soft tissues in those patients could increase the collapsibility of the upper airway, thus facilitating the appearance of obstructive apneas.
Patients in group II and III (with mild to moderate ascites and without ascites), showed non significant apnea (RDI less than 5) these results are in accordance to those obtained by Nikaina et al., who concluded that early stage cirrhosis is not associated with sleep disorders and OSAS.

The sleep efficiency and the neurocognitive functions (by MMSE) showed a statistically significant correlation with the apnea which was more clear in the group with severe RDI (Table 8).

These results go with those found by Bonnet (26) who stated that OSAS was found to produce sleep fragmentation and disruption (brief arousals from sleep that prevent consolidated sleep) and nocturnal hypoxemia (intermittent oxygen desaturation). Both of these complications have been related to behavioral phenomena of day time sleepiness and neurocognitive impairments during the subsequent day.

Patients may demonstrate arousals with concomitant symptoms such as choking and gasping sensation that help to support a diagnosis of OSA rather than another sleep disorder(17).

OSA may promote and exacerbate insomnia symptoms through psychophysiological conditioning in response to repeated complete arousals(27). These findings are evident by reduced sleep efficiency, total sleep time, and sleep latency(29). Such awakenings may lead to ruminations and a state of hyperarousability that result in difficulties in initiating or maintaining sleep(30).

In this group of patients there is chronic liver disease with tense ascites and obstructive sleep apnea. Both of these pathogenic factors could explain the more significant sleep deficiency and neurocognitive dysfunction present in this group.

The group of patients with tense ascites and OSAS underwent tapping of ascites. Polysomnographic recording and MMSE were repeated after tapping.

There was a marked improvement in the RDI and the average oxygen saturation after the tapping (Table 9) which confirms that the main cause of apnea was the ascites as found by Crespo et al.24, they found that OSAS disappears after removal of ascetic fluid. This suggests that the enlarged abdominal perimeter plays a role in the development of this ascites-related complication (diaphragmatic elevation produces a decrease in the functional residual capacity and perhaps modifies the upper airway system).

The presence of residual mild apnea is in concordance with the opinion of Golpe et al.25. There can be additional oedema of the laryngeal and pharyngeal soft tissues that could increase the collapsibility of the upper airway, thus facilitating the appearance of OSAS.

Marked improvement in the sleep efficiency and neurocognitive functions measured by MMSE (Table 10) prove that OSAS has a great impact on those functions. Those findings are in concordance with those found by Ryan and Bradley 31, who found that recurrent episodes of upper airway obstruction has a wide spectrum of clinical consequences including daytime hypersomnolence and neurocognitive dysfunction.

However the patients did not improve to the extent to have sleep efficiency and MMSE results as those of group II and III. This confirms that chronic liver disease affects sleep and neurocognitive functions and this affection increases with the progression of the disease as stressed by Cordoba14.

In conclusion, Obstructive sleep apnea syndrome (OSAS) can be a complication of tense ascites. OSAS causes more deterioration of sleep efficiency and neurocognitive functions and these findings improved after tapping of ascites.

REFERENCES


الملخص العربي

انقطاع النفس النومي ومؤثراته في مرض الاستسقاء

انقطاع النفس الاسبادي النومي وصف مؤخرًا كمضاعفة للاستسقاء. يحدث ذلك نتيجة ارتفاع الحمل الميكانيكي على مجرى التنفس العلوي مع قصور في الاستجابة العصبية العضيلية المعدلة. إضطرابات النوم وزيادة النوم أثناء النهاية والاضطراب في الوظائف العقلية من المضاعفات كثيرة الموجودة في مرض تليف الكبد ومرض اقطاع النفس النومي. تزيد شدة هذه المضاعفات مع وجود كلا المرضى وتحسن بصورة ملحوظة مع تحسن اقطاع النفس النومي بعد بزل الاستسقاء. 

الهدف من البحث كان تحديد نوع اقطاع النفس النومي المصاحب للاستسقاء وتأثيره على حالة المريض ومدى تحسن إضطرابات النوم والاضطرابات في الوظائف العقلية بعد بزل الاستسقاء. أشتمل البحث على 40 مريضا من مرضى الكبد المرضى مقاتلين إلى 3 مجموعات بداء على درجة الأستسقاء. المجموعة الأولى مكونة من 20 مريضا بحالات شديدة والمجموعة الثانية مكونة من 10 مرضى بحالات خفيفة أو متوسطة والمجموعة الثالثة مكونة من 10 مرضى بدون استسقاء. تقييم المرضى من خلال فحص أكليتريكي شامل وفحص القدرات القهارية الصغرى (MMSE) والفحوصات الباطنية. الصورية على البطن ورسم النوم الليلي المتبقي.

أظهرت النتائج أن جودة النوم والوظائف العقلية تتأثر تأثيرًا ذات دالة إحصائية بزيادة الاستسقاء. وجد أن اقطاع النفس النومي من النوع الاسبادي أعلى بوضوح في المجموعة الأولى من المجموعة الثانية والثالثة. ووجد أيضا أن جودة النوم والوظائف العقلية تتأثر تأثيرًا دون دالة إحصائية بوجود اقطاع النفس النومي الملاحظ وقد تمت نسبة اقطاع النفس النومي وجودة النوم والوظائف العقلية بصورة ملحوظة بعد بزل الاستسقاء، والخلاصة من هذا البحث أن اقطاع النفس الاسبادي النومي من الممكن أن يكون أحد مضاعفات الاستسقاء الشديد، وله امراض النوم الاسبادي النومي تدهوراً أكبر في حالة النوم والوظائف العقلية وتحسن هذه الوظائف بعد بزل الاستسقاء.