Original Article

Association of Obstructive Sleep Apnea with Nocturnal Hypoxemia in Metabolic-Associated Fatty Liver Disease Patients: A Cross-sectional Analysis of Record-based Data

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Abstract

Rationale: Obstructive sleep apnea (OSA) is often seen among obese individuals and the obesity has a linear association with MAFLD. The contribution of chronic intermittent nocturnal hypoxia of OSA and association of MAFLD with OSA is an unmet need. The present study aimed to determine the etiology, impact and association of OSA severity and nocturnal hypoxemia among patients of Chronic liver disease (CLD). **Methods:** In this study, analysis of the medical records and clinical details of the patients of CLD who had undergone polysomnography were analyzed after appropriate inclusion in study as per inclusion and exclusion criteria. After assessing the eligibility criteria, a total of 78 patients were included in the final analysis. Nocturnal hypoxemia was gauged from the baseline oxygen saturation record of study. Presence and severity of OSA were graded as per American Academy of Sleep Medicine (AASM) criteria. The primary objective of the study was to determine the association between OSA severity and nocturnal hypoxemia to the presence of Non-alcoholic Fatty Liver Disease (NAFLD). Secondary objectives were to assess the association of OSA severity and extent of nocturnal hypoxemia to the BMI and to determine the proportions of NAFLD subjects with OSA. **Results:** A total of 78 patients were screened, of which only 11 (14.1%) were female. Out of these, 56 (71.8%) were classified to MAFLD group while 22 (28.2%) were to the non-MAFLD group. The patients in MAFLD group with mean age of 56.02 years were older as compared to non-MAFLD with mean age of 51.05 years but that was not statistically different. Patients were categorized into MAFLD ($n = 56$) and non-MAFLD, representing other etiologies of CLD ($n = 22$; ethanol, chronic Hepatitis B virus (HBV), chronic Hepatitis C virus (HCV), cryptogenic, Non-cirrhosis portal fibrosis (NCPF), Primary sclerosing cholangitis (PSC), Autoimmune hepatitis (AIH), sarcoidosis, Wilson's disease). The mean BMI was significantly higher in MAFLD in comparison to non-MAFLD (34.51 ± 8.79 vs. 25.47 ± 5.75; $P = 0.000$) and also the median AHI of MAFLD group was significantly higher than the non-MAFLD 4.95 {(1.85, 25.47) vs. 0.85 (0.30, 2.72) (P value < 0.000)} [Table 1]. Among the desaturation indices, the number of desaturations >3% {median of 122.50 (75.00, 241.25) vs. 63.00 (13.75, 158.00), P value 0.009} and average desaturation {mean of (5.04 ± 2.16) vs. (3.78 ± 1.226)%, P value 0.002} were significantly higher in MAFLD versus non-MAFLD group [Table 2]. The AHI and all desaturation parameters, although not statistically significant, were worst in Child B [Table 3]. **Conclusion:** MAFLD patients have higher prevalence and greater severity of OSA and worse nocturnal desaturation parameters as compared to non-MAFLD patients. OSA is independent of obesity among patients of CLD, but prevalent among NAFLD group. Further prospective studies are needed among MAFLD and OSA patients to elucidate the mechanism linking pathophysiology of OSA-MAFLD and guide therapy.

Keywords: BMI, metabolic-associated fatty liver disease, nocturnal hypoxemia, obstructive sleep apnea; PSG, SpO_2

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Introduction

Obesity and obstructive sleep apnea (OSA) have long been recognized to be associated with spectrum of nonalcoholic fatty liver disease (NAFLD) and NAFLD is considered as a hepatic manifestation of metabolic syndrome.[1] The chronic intermittent nocturnal hypoxia resulting from repeated upper airway obstruction in OSA patients is considered to be a contributing factor in the development of NAFLD.[2] The general population prevalence estimates for OSA vary from 9.3% in India[3] to that of around 24% and 9% in male and females, respectively, from USA.[4] For ≥5 AHI events, the overall population prevalence is estimated to vary from 9% to 38%.[5] NAFLD is an emerging liver disease and currently the most common cause of incidental abnormal liver tests.[2] The worldwide prevalence of NAFLD is estimated to be around 25% in general population and in up to 75% in obese individuals.[6,7] In patients undergoing bariatric surgery and having OSA, NAFLD is present in around 80% of cases and vice versa.[6,7] OSA and more importantly its hallmark, chronic intermittent hypoxia (CIH), are established factors in the pathogenesis and exacerbation of NAFLD.[8] OSA and CIH induce insulin-resistance and dyslipidemia which are involved in NAFLD physiopathogenesis.[8] CIH increases the expression of the hypoxia inducible transcription factor Hypoxia inducible factor (HIF1α) and that of downstream genes involved in lipogenesis, thereby increasing β-oxidation and consequently exacerbating liver oxidative stress.[8] OSA also disrupts the gut–liver axis, increasing intestinal permeability and with a possible role of gut microbiota in the link between OSA and NAFLD.[8] OSA and chronic intermittent hypoxia may be linked with the pathogenesis and the severity of NAFLD.[9] Whether OSA and consequent chronic hypoxia, apart from other shared comorbidities with NAFLD, is an independent additional risk factor in the natural history of NAFLD is yet to be conclusively proved by large prospective studies in OSA and NAFLD cohorts. Notwithstanding, many earlier human studies and experimental animal studies linking OSA and NAFLD, definite studies linking its causation to OSA independent of obesity are still lacking. The aim of the present study is to determine the etiology, impact and association of OSA severity and nocturnal hypoxemia among patients of chronic liver disease. More recently, the international panel of experts have updated its nomenclature to metabolic dysfunction-associated fatty liver disease (MAFLD), a term that describes association with metabolic dysfunction and thereby carries a positive diagnostic approach, rather than mere absence of alcohol-associated disease.[10,11]

Hereby we also propose a schematic diagram for linked pathophysiology and natural history of OSA and MAFLD [Diagram 1].

The primary objective of this study is to determine the association between OSA severity and nocturnal hypoxemia in the MAFLD patients.

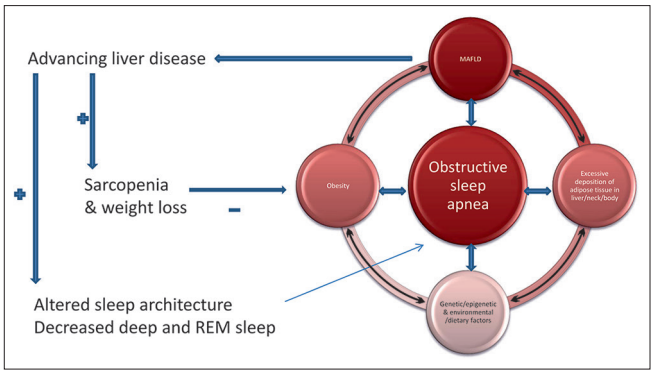

Diagram 1: Proposed schematic diagram for OSA and MAFLD-linked pathophysiology and natural history

Material and Methods

This is the cross-sectional analysis of a retrospective data which was approved by Institutional Ethics Committee (reference number: F37 (1)/9/ILBS/DOA/2020/20217/275-281), ILBS. All CLD patients who had undergone screening polysomnography (PSG) in the department of pulmonary medicine from January 2013 to February 2020 and had complete medical records available were included in the study. Patients with incomplete data were excluded.

Relevant medical records of those chronic liver disease patients were segregated to study the association of different variables. The diagnosis of NAFL/NAFLD/NASH (all included under the umbrella of MAFLD as per recent nomenclature) was based on ultrasound or Fibro scan or liver biopsy whichever was available. Presence and severity of OSA were graded as per American Academy of Sleep Medicine criteria applied to level I sleep study records using an Alice III version PSG machine (Philips, USA). AHI was defined as the mean of the number of apneas and hypopneas occurring per hour of sleep. OSA was considered to be present for a mean, AHI ≥ 5 events/h, and graded as mild for an AHI score of 5–<15, moderate for an AHI score of 15–30 and severe for an AHI score of > 30.[12] Nocturnal hypoxemia was be gauged from the baseline oxygen saturation record of study. Etiology and child class of chronic liver disease were agreed upon as per patient treatment records.

Statistics

Categorical variables were expressed as percentages while continuous variables were expressed as mean and standard deviation or median and interquartile range for variables with skewed distribution. Categorical data were analyzed with Chi-square test or Fisher's exact test wherever appropriate. For comparison, the data were grouped on the basis of etiology as those with MAFLD or not. Continuous variables were compared with independent sample *t*-test or Mann–Whitney *U* test for two groups and by one-way ANOVA or Kruskal–Wallis test for more than two groups depending on the distribution. Statistical difference was at $P < 0.05$. All statistical analyses were done using SPSS version 22 (IBM, Armonk, New York, USA).

Results

A total of 78 patients were included for the study after appropriate exclusion as per study protocols. Only 11 (14.1%) of the recruited patients were female, rest 67 (85.9%) were male. Out of these, 56 (71.8%) were classified to MAFLD group while 22 (28.2%) were to the non-MAFLD group.

The patients in MAFLD group with mean age of 56.02 years were older as compared to non-MAFLD with mean age of 51.05 years but that was not statistically different. We have analyzed the study data of same included patients with respect to MAFLD and non-MAFLD, Child categories and OSA and non-OSA. Mean age and BMI of participants were 54.62 ± 9.96 (kg/m^2) and 32.02 ± 9.01 (kg/m^2). Among sleep parameters mean sleep efficiency and light sleep were 74.81 ± 12.59% and 65.69 ± 19.15%. The median AHI, REM sleep, deep sleep, arousal of total sleep and snore episodic of total sleep were 3.65 (0.80–15.55), 10.70 (5.00–20.37)%, 17.60 (6.15–28.90)%, 29.50 (19.00–45.25) and 22.50 (9.2–37.82) (% of Total sleep time (TST)).

Comparison among OSA and non-OSA etiology

In OSA (n = 32) versus non-OSA (n = 46), the median BMI in OSA {34.43 (30.94–41.09) (kg/m^2)} was higher than that of non-OSA {28.89 (23.38–31.97) (kg/m^2)} but was not statistically significant. Baseline SpO_2 was not significantly different between OSA (95.06 ± 2.27)% and non-OSA (94.65 ± 2.25)% (P value 0.802). Here all desaturation indices but longest desaturation were higher in OSA versus non-OSA group and were statistically significant. For the parameter of longest desaturation, the mean value was significantly low in OSA {OSA (74.19 ± 22.30) vs. non-OSA (101.45 ± 44.22), P value 0.001}. Snore episodic of total sleep, although not statistically significant, was higher in OSA group as compared to non-OSA group. The number of arousals was same in both the groups [Table 1].

Comparison among MAFLD and other (non-MAFLD) etiology

The mean BMI in MAFLD group 34.51 (kg/m^2) was significantly higher than in non-MAFLD of 25.47 (kg/m^2) (P value < 0.000). The median AHI of MAFLD group was significantly higher than the non-MAFLD 4.95 {(1.85, 25.47) vs. 0.85 (0.30, 2.72) (P value < 0.000)}. Among the desaturation indices, the number of desaturations >3% {median of 122.50 (75.00–241.25) vs. 63.00 (13.75–158.00), P value 0.009} and average desaturation {mean of (5.04 ± 2.16) vs. (3.78 ± 1.226)%, P value 0.002} were significantly higher in MAFLD versus non-MAFLD group. In the sleep parameters, there was a significant increase in light sleep {(68.69 ± 18.16) vs. (58.06 ± 19.93)%, P value 0.037} and snore episodic of total sleep {26.65 (15.25–39.92) vs. 8.90 (2.52–24.50)%, P value 0.027} and there was a significant reduction in deep sleep {15.10 (3.90–27.75) vs. 25.40 (12.90–37.70)%, P value 0.051} in MAFLD versus non-MAFLD group. There was number statistically significant difference with respect to other desaturation indices like baseline SpO_2, minimum SpO_2, SpO_2 time <90%, desaturation from baseline and longest desaturation [Table 2].

Comparison among Child classes

There was significant difference with respect to number of desaturations >3% among different Child classes with median values in Child A (n = 34) {128.00 (75.00–228.00)}, Child B (n = 18) {158.50 (43.00–253.25)} and Child C (n = 17) {54.00 (22.00–89.00)} (P value 0.012). *Post-hoc* analysis among child categories further revealed that for number of desaturations >3%, there was significance between Child A versus C (0.013) and Child B vs. C (0.054). Here the number of desaturations >3% were highest in Child B (158) followed by Child A (128) and least in Child C (58). Here the median AHI, although not statistically significant, was highest in Child B {7.45 (1.52–27.20)} followed by Child A {3.90 (1.3–16.00)} and

Table 1: Comparison of BMI, AHI and other PSG parameters between OSA and non-OSA group

Parameter	OSA (n=32)	Non-OSA (n=46)	P
BMI (kg/m^2)	34.43 (30.94-41.09)	28.89 (23.38-31.97)	0.34
AHI (number index)	21.70 (8.72-49.15)	1.10 (0.475-2.72)	<0.00
Base line SpO_2 (%)	95.06±2.27	94.65±2.25	0.81
Minimum SpO_2 (%)	76.44±9.67	88.26±6.22	0.03
SpO_2 time <90% (%)	5.00 (0.85-20.40)	0.10 (0.00-2.02)	0.02
Desaturation from baseline (number)	18.00 (10.25-23.75)	7.00 (5.00-10.25)	0.01
No. of desaturations >3% (number)	230.50 (131.00-280.25)	69.50 (33.50-120.50)	0.08
Average desaturation (%)	6.07±2.36	3.72±0.92	<0.00
Longest desaturation (s)	74.19±22.30	101.45±44.22	0.01
Sleep efficiency (%)	74.34±13.53	75.13±12.03	0.15
REM sleep (%)	12.05 (6.37-17.55)	10.30 (4.77-23.92)	0.91
Light sleep (%)	71.01±16.91	61.98±19.91	0.57
Deep sleep (%)	10.50 (1.00-20.57)	22.75 (14.77-31.15)	0.82
Arousal of total sleep (numbers)	29.50 (17.25-36.00)	29.50 (20.75-47.25)	0.71
Snore episodic of total sleep time (% of TST)	36.80 (22.50-54.12)	14.55 (4.15-26.70)	0.16
Arrhythmia index (number)	10.50 (4.00-22.00)	14.00 (4.00-26.25)	0.51
Maximum systolic BP (mmHg)	157.94±25.80	153.52±30.05	0.17

Table 2: Distribution of BMI, AHI and PSG parameters among MAFLD and non-MAFLD subjects

Parameter	MAFLD (*n*=56)	Non-MAFLD (*n*=22)	*P*
Age (years)	56.02±9.28	51.05±10.93	0.68
BMI (kg/m^2)	34.51±8.79	25.47±5.75	<0.00
AHI (number index)	4.95 (1.85-25.47)	0.85 (0.30-2.72)	<0.00
Base line SpO_2 (%)	94.71±2.28	95.09±2.20	0.51
Minimum SpO_2 (%)	81.09±9.47	85.14±7.78	0.08
SpO_2 time <90% (%)	1.60 (0.10-12.22)	0.05 (0.00-3.42)	0.41
Desaturation from baseline (number)	11.00 (7.00-20.00)	7.5 (5.00-12.50)	0.12
No. of desaturations >3% (number)	122.50 (75.00-241.25)	63.00 (13.75-158.00)	0.01
Average desaturation (%)	5.04±2.16	3.78±1.226	0.01
Longest desaturation (s)	85.30 (63.62-112.05)	84.40 (51.62-109.50)	0.14
Sleep efficiency (%)	74.42±12.65	75.79±12.68	0.67
REM sleep (%)	10.05 (5.00-16.75)	13.90 (9.85-27.10)	0.21
Light sleep (%)	68.69±18.16	58.06±19.93	0.04
Deep sleep (%)	15.10 (3.90-27.75)	25.40 (12.90-37.70)	0.05
Arousal of total sleep (numbers)	30.50 (22.25-42.25)	25.50 (18.00-47.25)	0.71
Snore episodic of total sleep time (% of TST)	26.65 (15.25-39.92)	8.90 (2.52-24.50)	0.02
Arrhythmia index (number)	14.50 (5.25-31.25)	8.00 (3.00-15.25)	0.24
Maximum systolic BP (mmHg)	157.36±25.47	150.18±34.63	0.32

Table 3: Distribution of BMI, AHI and other studied PSG parameters across Child classes

Parameter	Child A (*n*=43)	Child B (*n*=18)	Child C (*n*=17)	*P*
BMI (kg/m^2)	31.22 (27.89-36.04)	31.94 (28.81-36.56)	27.48 (23.12-36.58)	0.13
AHI (number index)	3.90 (1.3-16.00)	7.45 (1.52-27.20)	0.80 (0.30-3.85)	0.27
Base line SpO_2 (%)	94.81±2.25	95.06±2.15	94.60±2.48	0.84
Minimum SpO_2 (%)	82.333±8.14	80.61±8.83	83.71±11.91	0.61
SpO_2 time <90% (%)	0.80 (0.10-9.2)	1.75 (0.00-17.80)	0.00 (0.00-5.5)	0.99
Desaturation from baseline (number)	10.00 (7.00-16.00)	13.00 (7.75-21.25)	5.00 (3.5-14.5)	0.52
No of desaturations >3% (number)	128.00 (75.00-228.00)	158.50 (43.00-253.25)	54.00 (22.00-89.00)	0.02
Average desaturation (%)	4.00 (3.7-5.1)	5.1 (3.5-6.2)	3.40 (3.35-4.85)	0.14
Longest desaturation (s)	81.30 (59.00-109.50)	97.25 (77.62-110.32)	86.30 (53.50-133.05)	0.73
Sleep efficiency (%)	75.89±14.17	72.41±11.87	74.61±8.71	0.62
REM sleep (%)	10.60 (5.00-20.30)	14.35 (9.07-19.27)	6.50 (2.80-27.50)	0.74
Light sleep (%)	67.46±14.94	63.41±25.95	63.63±21.12	0.67
Deep sleep (%)	17.00 (9.60-27.90)	12.85 (0.77-31.72)	27.30 (9.6-34.06)	0.34
Arousal of total sleep (numbers)	29.00 (20.00-52.00)	26.00 (16.75-36.50)	33.00 (23.50-37.50)	0.45
Snore episodic of total sleep time (% of TST)	21.40 (14.20-41.70)	30.55 (9.20-42.97)	18.60 (3.40-32.30)	0.35
Arrhythmia index (number)	9.00 (3.00-27.00)	14.50 (7.50-24.75)	14.00 (6.50-22.00)	0.34
Maximum systolic BP (mmHg)	157.05±27.26	156.00±25.29	150.29±34.49	0.71

Table 4: Association of MAFLD with OSA

	OSA present (*n*=32)	No OSA (*n*=46)	*P*
MAFLD	90.6%	58.7%	0.002

Table 5: Comparison of REM sleep percentage in our study with previous study

REM sleep %	Child A	Child B	Child C
Our study	10.60 (5.00-20.30)	14.35 (9.07-19.27)	6.50 (2.80-27.50)
Teodoro *et al.*	16.1±1.2	14.9±1.2	8.6±1.6

Child C {0.80 (0.30–3.85)} in that order. Similarly desaturation indices were too worst in Child B followed by Child A and Child C in that order. Among sleep parameters percentage of light sleep was highest in Child A, percentage of REM sleep in Child B and percentage of deep sleep in Child C, although not statistically significant. Total number of arousals was highest in Child C and that of snore percentage in Child B [Table 3].

In our study, more than 50% of MAFLD patients were found to be having OSA while ~90% of OSA subjects were having MAFLD which not only points toward shared risk factors and pathophysiology in causation of MAFLD and OSA but also indicates OSA to be an independent risk factor for MAFLD [Table 4 and Diagram 2].

Discussion

In the present study, we have retrospectively analyzed the sleep study parameters among studied cirrhotic patients with respect

Diagram 2: Implication of MAFLD for OSA

to MAFLD versus non-MAFLD, Child categories and OSA versus non-OSA. To the best of our knowledge, our study is first attempt to analyze sleep study parameters of same cohort of cirrhotic patient with respect to MAFLD versus non-MAFLD, OSA versus non-OSA and with respect to Child categories.

The mean age of the MAFLD, 56.02 ± 9.28 years and non-MAFLD, 51.05 ± 10.93 years was comparable to that of cirrhotic group {mean age of (50.0 ± 8.5) years} as reported by Teodoro *et al.*[13] In contrast to 69% ($n = 29$) males in *Teodoro et al.* study,[13] we had 85.95% ($n = 67$) males, which can be explained by a male preponderance in cirrhosis and gender bias in seeking health care in Indian population.

The statistically higher AHI values and worse desaturation parameters in MAFLD as compared to non-MAFLD subjects support the association of MAFLD and OSA in our study. The statistically significant higher values of BMI and snore episodic of total in MAFLD as compared to non-MAFLD have clinical implications in selection of potential candidates out of large number of cirrhotic patients with poor sleep quality, for in hospital sleep study.

The mean sleep efficiency of whole participants ($n = 78$) (74.81 ± 12.59)% with MAFLD versus non-MAFLD {(74.42 ± 12.65)% vs. (75.79 ± 12.68)%} was comparable to the mean sleep efficiency of cirrhotic group {(73.89% ±14.99)%} but considerably less as compared to that of non-cirrhotic normal cohort {with mean of (84.43% ±8.55%)} in findings by Teodoro *et al.*[13]

The median REM sleep and deep sleep in overall recruited subjects 10.70 (5.00–20.37)% and 17.60 (6.15–28.90)% and MAFLD group {10.05 (5.00–16.75)% and 15.10 (3.90–27.75)%} were less than normal value of REM (~25%) and deep sleep (~20%) in normal subjects in the report Shrivastava *et al.*,[14] thereby suggesting that as compared to normal population there is a loss of REM and deep sleep in cirrhotic patients. The median REM sleep in non-MAFLD group {13.90 (9.85–27.10)%} was comparable to mean REM sleep (14.04 ± 5.64)% in cirrhotic cohort in the study by Teodoro *et al.*[13] but higher as compared to MAFLD group {10.05 (5.00–16.75)%} suggesting that there is further loss of REM sleep in MAFLD as compared to non-MAFLD [Table 5].

The low sleep efficiency (which could partly be explained by age-related decrease) and loss of REM and deep sleep in our cirrhotic participants confirm that there is altered and poor sleep quality in cirrhotic patients as compared to normal patients.

In contrast to Teodoro *et al.*,[13] the percentage of REM sleep was highest in Child B group in our study, which also explains the highest number of desaturations > 3% and worst desaturation parameters in Child B group in our study. Whether this finding is related to large number of OSA and MAFLD patients in our cirrhotic patients or to altered sleep architecture as a natural history in case of cirrhotic patients as the disease progress or to biasing as a result of retrospective nature of our study and with low numbers of studied subjects cannot be ascertained by present study. In contrast to we couldn't find any statistically significant difference among Child classes with respect to BMI, AHI, desaturation and other sleep study parameters, but numerically AHI, most of the desaturation parameters were worst in Child Class B followed by A and C in that order. Here the paradoxical least worsened desaturation indices, least AHI could have explanation in most numbers of arousal in Child class C which tend to terminate the apnea/hypopnea and accompanying desaturation.

The median AHI of {3.65 (0.80–15.55) (kg/m^2)} our overall cirrhotic cohort cannot be compared to mean AHI (10.9 ± 8.5) (kg/m^2), with high SD values, of cirrhotic patients by Mabrouk *et al.*[15] in their study. In OSA versus non-OSA, AHI and all desaturation indices but longest desaturation were higher in OSA versus non-OSA group and were statistically significant. For the parameter of longest desaturation, the mean value was paradoxically significantly low in OSA {OSA (74.19 ± 22.30) s versus non-OSA (101.45 ± 44.22) s, *P* value 0.001} which couldn't be explained by any apparent reason.

Snore episodic of total sleep, although not statistically significant, was higher in OSA group as compared to non-OSA group but number of arousals was same in both the groups which suggests that presence of OSA can be correlated with number of snores rather than number of arousal during sleeping while evaluating a probable patient for PSG screening study.

One of the interesting findings from the study results was that there was statistically significant difference in BMI between OSA and non-OSA groups in same study subjects, which could only be explained by MAFLD rather than obesity as an associated etiology for OSA in the studied cohort.

Also, the change of nomenclature from NAFLD to MAFLD with a positive criteria-based diagnostic approach seems to have increased awareness among physicians with heightened increase in interests in the disease.[16,17]

Further step ahead and bearing on to this, India has become the first country to identify the need for action for NAFLD by Integration of NAFLD with NPCDCS (National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke[18]).

Conclusion

Our study confirms altered sleep architecture and poor sleep quality in cirrhotic patients as compared to normal population and further there was reduction in deep sleep in MAFLD group as compared to non-MAFLD. Our study not only confirms the association of OSA with MAFLD but also provides epidemiological evidence of MAFLD rather than obesity as an etiology for OSA in our study.

Limitations of the study: Our study has a few limitations apart from being retrospective in analysis. There is possibility of selection bias as the patients with MAFLD were more likely to be referred for sleep study. Basis of diagnosis of MAFLD in our study subjects was also not histopathological.

Author contribution

Arvind Tomar and Debajyoti Bhattacharya developed the study concept, acquisition of data done by Arvind Tomar and Ankit Bhardwaj, statistical analysis done by Ankit Bhardwaj, drafting of manuscript by Arvind Tomar, Debajyoti Bhattacharya and Ashok Choudhary, finalization of manuscript done by Arvind Tomar.

Ethics approval

Institutional Ethics Committee, Institute of Liver and Biliary Sciences, New Delhi, India. Reference number. F37 (1)/9/ILBS/DOA/2020/20217/275-281.

Abbreviations

Obstructive Sleep Apnea (OSA), Metabolic-Associated Fatty Liver Disease (MAFLD), Non-alcoholic Fatty Liver disease (NAFLD, Hepatitis B virus (HBV), Hepatitis virus (HCV), Non-cirrhosis portal fibrosis (NCPF), Primary sclerosing cholangitis (PSC), Autoimmune hepatitis (AIH), Chronic intermittent hypoxia (CIH), Polysomnography (PSG), Apnea-Hypopnea index (AHI), Oxygen saturation (SpO2), Non-alcoholic steatohepatitis (NASH), Child-Turcotte-Pugh (CTP) classification (Child A, Child B, Child C) used to grade the severity of liver disease.

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Nil.

Conflicts of interest

There are number conflicts of interest.

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