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EFFECT OF OBSTRUCTIVE SLEEP APNEA ON LIPID PROFILE.

Physiology

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ABSTRACT

To see the effect of obstructive sleep apnea on lipid profile.

OBJECTIVES:- To find the difference in BMI, total cholesterol(TCH), triglyceride (TG), high-density lipoprotein (HDL Cholesterol), VLDL Cholesterol, and LDL Cholesterol among participants of AHI>10 group and AHI≤ 10 group.

METHOD: Two groups were made, AHI>10 group having 25 participants and AHI≤10 group having 25 participants on bases of severity of sleep apnea recorded by polysomnography. Their height was measured with help of mounted measuring tape, weight was recorded using digital weighing machine, followed by BMI calculation. fasting blood sample was collected in morning around 6.30 am after recording of polysomnogram for lipid profile . Then comparison of all these parameters was done among the participants of the AHI>10 and AHI≤ 10 groups.

RESULT: Mean BMI of participants in AHI>10 group was more as compare to mean BMI of participants in AHI≤10 group and the difference was highly significant. Mean TG of participants in AHI>10 group was significantly higher as compare to mean TG of participants in AHI≤10 group . Mean LDL of participants in AHI>10 groups was significantly higher as compare to mean LDL of participants in AHI≤10 group.

CONLUSION: In this study we have concluded that increasing severity of OSA (obstructive sleep apnea) causes significant increase in level of TG and LDL cholesterol in serum of these participants.

KEYWORDS

Sleep apnea, Body mass index, TCH, TG, HDL, VLDL and LDL.

INTRODUCTION:-

Obstructive Sleep Apnea (OSA) is a common disorder, affecting approximately 4% of adult men and 2% of adult women in general population. [1] OSA is characterized by repeated episodes of complete or partial obstruction of the upper air way during sleep. [1] Apnea is the complete cessation of airflow lasting at least for 10 second and hypopnea is a discernible fall in airflow lasting for at least 10 second accompanied by a decrease in oxygen saturation of at least 3%.[2] Apnea hypopnea index (AHI) is the total number of apneas and hypopneas per hour of sleep.[2]

Obesity is considered as a major risk factor for the development and progression of OSA. [3, 4] The prevalence of OSA in obese or severely obese patients is nearly twice that of normal-weight adults. Furthermore, patients with mild OSA who gain 10% of their baseline weight are at a six fold-increased risk of progression of OSA, and an equivalent weight loss can result in a more than 20% improvement in OSA severity. [5]

The gold standard for diagnosis of sleep apnea is polysomnography. It is simultaneous recording of multiple physiologic parameters, namely electroencephalogram, electrooculogram, electromyogram, oronasal airflow, chest wall and abdominal motion, body position, snoring, electrocardiogram and oxyhaemoglobin saturation. [2]

In patients with sleep apnea, airway obstruction causes increases in respiratory effort, intermittent arterial oxygen desaturation, and sleep fragmentation, sleep fragmentation increases sympathetic activity, which can increase systemic and pulmonary arterial blood pressure alterations [1,6]

Chronic intermittent hypoxia leads to hypercholesterolemia and lipid peroxidation and the degree of metabolic dysregulation is dependent on the severity of the hypoxic stimulus. [7]

So in this study attempt is made to find the effect of obstructive sleep apnea on lipid profile of non-diabetic patients.

METHODOLOGY: This study was carried out in Sleep Research Center, MGM Hospital Kamothe, Navi Mumbai after the approval from the Institutional ethics committee . Age range of participants was from 20 to 70 years. Normal participants, participants having obstructive sleep apnea and free from other diseases were included in this study. Total sample size was 50 which was divided into two groups, AHI>10 and AHI≤10 on bases of severity of apnea. In AHI>10 group there were 25 participants, out of which 20 were male and 5 were female. In AHI≤10 group there were 25 participants, out of which 14 were male and 11 were female.

Methodology of each objective:

Polysomnography:

- Patients were called at 8 pm in sleep research center.
- Then procedures were explained to them.
- Followed by which their consents were taken.
- Height was recorded in cm with help of wall mounted measuring tape and weight was measured in kg with help of digital weighing machine and BMI was calculated.
- Followed by which electrodes were placed according to their positions for recording polysomnogram.
- After which the polysomnogram was recorded from 10 pm to 6 am.

Blood test:-

- Fasting blood sample is collected in morning around 6:30 am after recording of polysomnogram.
- Then blood sample is sent to the biochemistry laboratory to get all blood test done.
- Serum total cholesterol (TCH) is done by CHOD-TAP method.
- Serum triglyceride (TG) is done by GPO- TRINDER method.
- Serum high-density lipoprotein (HDL Cholesterol) is done by Phosphotungstic Acid method.
- VLDL Cholesterol was calculated by Triglyceride/five.
- LDL Cholesterol was calculated by Total cholesterol – (HDL cholesterol + VLDL)

OBSERVATION AND RESULT:-

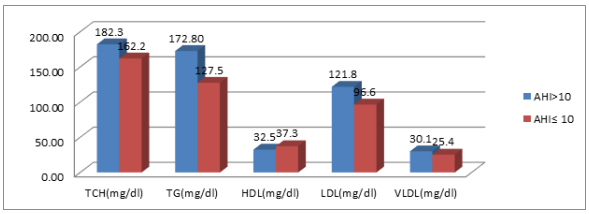
In the present study, all data collected were statistically analyzed using SPSS 19.0. Software. The data was presented using descriptive statistics such as mean, standard deviation (SD) followed by multiple bar charts. Further comparison between AHI>10 and AHI≤ 10 groups was done using independent sample t-test. The recorded values were expressed as Mean±SD. The level of significance was set at 5%. All p–values less than 0.05 were considered to be significant.

TABLE 1:- Comparison age, BMI, lipid profile of non-diabetic groups AHI>10 and AHI≤ 10.

Variables	AHI>10 (n=25)	AHI≤ 10 (n=25)	P -value
AHI	47.53±34.13	3.16±3.44	0.000001***
Age(year)	51.4±11.01	45.12±14.39	0.09
BMI(kg/m2)	32.01±5.86	25.82±3.75	0.000065***
Total Cholesterol(mg/dl)	182.3±51.05	162.2±32.77	0.105
Triglycerides(mg/dl)	172.8±97	127.5±52.82	0.048*
HDL -Cholesterol (mg/dl)	32.5±11.16	37.3±13.01	0.169
LDL-Cholesterol (mg/dl)	121.8±46.15	96.6±37.28	0.039*
VLDL-Cholesterol (mg/dl)	30.1±11.27	25.4±10.91	0.138

***significant p<0.05**
***** very highly significant p<0.00001**

Figure 1: - Comparison age, BMI and lipid profile of non-diabetic groups AHI>10 and AHI≤ 10



RESULT:

In AHI>10 and AHI≤10 groups the mean ± SD AHI was (47.53±34.13) and (3.16±3.44) respectively. (P value=0.000001)

In AHI>10 and AHI≤10 groups the mean ± SD age was (51.4±11.01) and (45.12±14.39) respectively. (p value=0.09)

In AHI>10 and AHI≤10 groups the mean ± SD BMI was (32.01±5.86) and (25.82±3.75) respectively. (P value=0.000065)

In AHI>10 and AHI≤10 groups the mean ± SD Total Cholesterol in mg/dl was (182.3±51.05) and (162.2±32.77) respectively. (P value=0.105)

AHI>10 and AHI≤10 groups the mean (mean ± SD) Triglycerides in mg/dl was (172.8±97) and (127.5±52.82) respectively. (P value=0.048*)

In AHI>10 and AHI≤10 groups the mean ± SD HDL in mg/dl was (32.5±11.16) and (37.3±13.01) respectively. (P value=0.169)

In AHI>10 and AHI≤10 groups the mean ± SD LDL in mg/dl was (121.8±46.15) and (96.6±37.28) respectively. (P value=0.039*)

In AHI>10 and AHI≤10 groups the mean ± SD VLDL in mg/dl was (30.1±11.27) and (25.4±10.91) respectively. (P value=0.138)

DISCUSSION:-

This research has been done to find the effect of obstructive sleep apnea on lipid profile of non- diabetic patients.

There was no significant difference in mean age among the participant of both the groups.

There was very highly significant(P value=0.000001) difference in mean AHI among participants of both the groups

BMI of participants in AHI>10 group was higher very significantly (P value=0.000065) as compare to BMI of participants in AHI≤10 group.

Young T in his two studies showed that an increasing BMI (in increments of 5.3 or 5.6 kg per m2) was associated with an increased risk of OSA as also observed in our study [8, 9].

Bixler EO, Bixler EO noted that BMI greater than or equal to 31.1 kg per m2 in men and 32.3 kg per m2 in women was found to be predictive of OSA (OR = 7.8 and 12.8, respectively).[10,11]

Rogers A screened 1,035 patients and noted obesity is strongest predictor of OSA risk. [12]

In our study we had a similar finding of increasing **BMI** in OSA participants which was highly statistically significant.

Studies by Martinez-Rivera C and Ibrahim AS showed that a BMI greater than 30 kg per m2 was a dependent variable for OSA. [7, 13]

Jianguo Li et al in 2007 found that chronic intermittent hypoxia leads to hypercholesterolemia and lipid peroxidation in the absence of obesity, and the degree of metabolic dysregulation is dependent on the severity of the hypoxic stimulus.[14] In our study total Cholesterol was more in participants of AHI>10 group as compared to participants of AHI≤10 group but the difference was not statistically significant.

Total Triglycerides was more in participants of AHI>10 group as compare to participants of AHI≤10 group and the difference was statistically significant.

Total HDL cholesterol was less in participants of AHI>10 group as compare to participants of AHI≤10 group and the difference was not statistically significant.

Total LDL cholesterol was more in participants of AHI>10 group as compare to participants of AHI≤10 group and the difference was statistically significant.

Total VLDL cholesterol was more in participants of AHI>10 group as compare to participants of AHI≤10 group and the difference was not statistically significant.

Ip MS et al in 200 noted that compare to control subjects with a similar BMI but without OSA, the OSA group had a significantly more dyslipidemia, and greater adiposity reflected by skinfold thickness. [15] Our study shows similar findings.

Jianguo Li in 2005 noted that exposure of lean mice to intermittent hypoxia increases fasting serum levels of total cholesterol, high-density lipoprotein (HDL) cholesterol, phospholipids (Pls), and triglycerides (TGs), as well as liver TG content. [16] Our study shows similar findings except there was decrease in HDL with increasing severity of OSA

Kawano noted that HDL-Cholesterol was correlated negatively with the lowest arterial oxyhemoglobin saturation and LDL-Cholesterol was correlated positively with apnea-hypopnea index. [17]Our study shows similar findings of decrease in HDL and increasing LDL-Cholesterol with increasing severity of OSA

Hence in our study it was seen that with increasing AHI there was a change in the lipid profile of participants with increasing levels of serum TCH, TG, VLDL, LDL and decreasing levels HDL Cholesterol . It was also observed that BMI was higher in participants with AHI > 10group.

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