Section: Biochemistry

Original Research Article

EVALUATION OF ADIPONECTIN AND OBESITY IN SLEEP DISORDER BREATHING PATIENTS WITH AND WITHOUT OBESITY

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Abstract

Background: Obesity is a pandemic condition that leads to health impairment by increasing the risk of developing diseases and it has been associated with hormonal and metabolic parameter alteration. Several conditions are associated with sleep disorder such as high BP, Insulin resistance, BMI and visceral fat deposition. Weight loss is one of the keys for not only obesity but also for abnormal sleep pattern to reduce the risk. Association of plasma adiponectin levels and obesity in patients of SDB with and without obesity. **Materials and Methods:** A total 50 sleep disorder breathing patients were collected from Sleep Medicine and Research Centre MGMIHS after getting Ethical Approval. Further subjects were classified 25 subjects SDB with obesity and 25 subjects SDB without obesity. Clinical examination was done for every participant. Plasma Adiponectin levels wereanalyzed by ELISA. **Result:** A significantly lower mean levels of plasma Adiponectin in group-I (6.48 ± 1.44 mg/l) than that of group -II (8.22 ± 1.45 mg/l) ($P<0.001$). A significantly inverse relation between BMI and adiponectin in group-I($r= -0.63$, $P<0.05$). **Conclusion:** An inverse relationship between Adiponectin and BMI suggested that weight loss could be helpful to improve the abnormal sleep pattern.

INTRODUCTION

Sleep disordered breathing (SDB), a medical condition that increasingly recognized by adverse health effects. It is a term that encompasses obstructive apneas, central apneas, hypopneas and respiratory effort-related arousals that occur during sleep. There are certain evidences that increasingly indicate that SDB is associated with adverse health effects and represents a growing public health concern.[1] Obesity is considered primary risk factor for the development of SDB and it contributes cardiovascular and metabolic abnormalities in this population. Obesity is one of the most significant risk factors for the development of SDB.[1,2] Increasing evidence suggests that short sleep durations are associated with a number of health risks, including obesity.[3] atherosclerosis, coronary artery disease, cardiovascular events. Mechanistically, through intermittent hypoxemia and reoxygenation, Obstructive sleep apnea (OSA) promotes systemic inflammation and oxidative stress which in turn causes endothelial dysfunction and atherogenesis.[4]
It is well known that obesity is an independent risk factor for SBP and with the current epidemic of obesity, the prevalenceand incidence of SBP is projected to increase dramatically in thecoming decades.[5] Therefore, it is clinically relevant to investigatethe potential biomarkers that are associated with SBP developmentin obese populations. Adiponectin is excreted by adipocytes and exerts numerous cardio-protective effects by means of antiinflammation, endothelial-protection, and anti-oxidation. Adiponectin is an anti-inflammatory and anti-atherogenic endocrine marker, which is involved in regulation of insulin sensitivity and lipid oxidation.[6,7] Adiponectin levels are significantly lower in obese, compared to non-obese, individuals and have been found to be inversely related to type 2 diabetes and cardiovascular risk, reported by many of the workers. Regarding the overlapped risk factor in terms of obesity but the opposed pathophysiological effects between OSA and adiponectin.[7] So, the present study was hypothesized to evaluate the association between plasma adiponectin levels with SBP in obese populations.

MATERIALS AND METHODS

After getting an ethical approval from institutional ethics committee(Approval No.– 2017/04/SC/51), an interdepartmental prospective type of study was carried out in the Department of Biochemistry, MGM

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Medical College & Hospital and Sleep Medicine and Research Centre MGMIHS Kamothe, Navi Mumbai from November 2016 to February 2018. Subjects with the age group of 25-65 of either gender (Male & Female) came to the sleep OPD for Polysomnography were enrolled for this study after taking a written and verbal informed consent from each and every participant. The data were collected from each subject regarding general information, clinical history (cause, symptoms, severity of diseases & drug history), socioeconomic status and family history with the help of pre-validated proforma. Anthropometric measurements and vital sign were also recorded from each and every individual.

Selection of participants: Polysomnography Evaluation: Full night polysomnography (Sandman Elite, USA) was administered to participants from 22.00 pm to 06.00 am (Second day). The following parameter were recorded (Embla Sandman Elite Sleep Diagnostic Software), EEG (electroencephalogram), electrocardiogram, Sub-mental electromyogram, chest and abdominal wall motion, airflow by nasal pressure, pulse oximetry, sleep architectures. Respiratory events were analyzed by standard criteria of American Academy of sleep medicine (AASM).[15] Apnea defined as drop of airflow ≥ 90% for at least 10 seconds, Hypopnea defined as decrease airflow ≥ 30% for at least 10 seconds accompanied by oxygen desaturation or arousal. AHI calculated by the total no. of apnea and hypopnea episode divided by total sleep time. Apnea-Hypopnea index (AHI) diagnostic criteria, that is Normal (AHI<5), Mild sleep Apnea (5 ≤ AHI ≥ 15), Moderate Sleep Apnea (15 ≤ AHI ≥ 30) and Severe Sleep Apnea (AHI ≥ 30).[8]

The subjects who havePsychiatric disorder, Epilepsy, Seizure, Pregnancy, patient with hypothyroidism, Patient with upper airway surgery, HIV and Hepatitis etc. were excluded from the current study.

The enrolled subjects for the present study were categorized into two groups on the basis of body mass index (BMI):

Group-1: 25 sleep disorder breathing patients with obesity (CASE).

Group-2: 25 sleep disorder breathing patients without obesity (CONTROLS)

All the biochemical investigations were done at MGM Medical College & Hospital, Navi Mumbai.

Blood Sample: Approximately 6 ml blood was collected after an overnight fasting from each subject by venipuncture with standard blood collection technique in a plane vial and sodium fluoride vial, wait for 10 mints and centrifuge at 3000 RPM for 15 minutes to obtain serum and plasma sample respectively. Serum sample was used for adiponectin and lipid profile analysis. Immediately plasma was used for testing fasting blood glucose. Remaining serum sample was stored at -70ºC for measurement of adiponectin and lipid profile levels.

Blood sugar was analyzed by enzymatic method (Hexokinase, glucose-6-Phosphatase dehydrogenase method) at EM-200 autoanalyzer and plasma adiponectin by sandwich ELISA were done at central clinical Biochemistry laboratory, MGM Hospital Navi Mumbai.

Statistical Analysis: Qualitative variables were presented in frequency and Quantitative data were presented in the form of Mean and SD. Statistical analysis was done by using SPSS-20 software. Significant mean value was determined by unpaired student 't' test and test of significant was p< 0.05 considered as statistically significant.

RESULTS

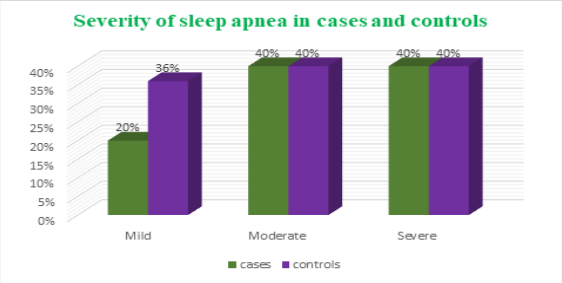

Figure 1: Distribution of subjects on the basis of Severity of sleep apnea in study and control group.

[Figure 1] showing percentage study subjects based on severity of diseases. There was an equal number of subjects who have moderate (40%) and severe (40%) respectively whereas 20% are under mild condition. The percentage of subjects with sleep disorder breathing without obesity were similar for moderate and severe and 36% with mild condition.

[Table 1] Showing statistical changes in baseline characteristic including Age and AHI. A higher mean age was observed in control group (50.60 ± 9.58 years) than that of case group (46.00 ± 11.16 years). The mean levels of AHI in cases 27.64 ± 13.90 events/hours and in controls 24.61 ± 13.46 events/hours respectively. The mean levels of BMI and FBS in case group were 34.09 ± 3.59 kg/m^2 and 133.37 ± 29.05 mg/dl respectively than that of controls 27.65 ± 3.71 kg/m^2 and 106.08 ± 19.5 mg/dl and it was found to be statistically significant (P<0.001). A significantly lower mean levels of Plasma Adiponectin were observed in cases (6.48 ± 1.44 mg/L) than that of corresponding controls (8.22 ± 1.45 mg/L) and it was found to be statistically significant (P<0.001).

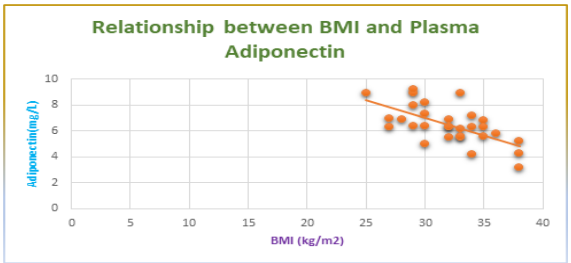

Figure 2: Correlation analysis between BMI and adiponectin in subjects with SDB with obese.

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A significantly negative correlation was observed between BMI and adiponectin in subjects with SDB with obese (r= -0.63, P<0.05).

Table 1: Showing statistical changes in baseline characteristic and biochemical parameter in Study group (SDB with Obese) and control group (and SDB without Obese).

Variables	Study group (Mean ± SD)	Control group (Mean ± SD)	p- Value
Age (years)	46.00 ± 11.16	50.60 ± 9.58	0.1244#
AHI (event/hour)	27.64 ± 13.90	24.61 ± 13.46	0.4375#
BMI (kg/m2)	32.09 ± 3.39	27.65 ± 3.71	0.0001**
FBS (mg/dl)	133.37 ± 29.05	106.08 ± 19.5	0.001**
Adiponectin(mg/L)	6.48 ± 1.44	8.22 ± 1.45	0.001**

*p ≤ 0.05 Significant, **p ≤ 0.001 Highly Significant, # p > 0.05 Not significant

DISCUSSION

Life style modification and rapid urbanization has triggered the obesity epidemic, which is associated with a number of health problems. Adiponectin is an endocrine factor synthesized and released from adipose tissue. Secretion of various bioactive substances from adipose tissue, conceptualized as adipocytokines, which has been widely recognized to have a role in insulin resistance, diabetes, cardiovascular diseases and others. In contrast to circulating inflammatory factors, adiponectin has anti-diabetic, anti-atherogenic and anti-inflammatory properties. Adiponectin is a fat-derived hormone that play a crucial role in protecting against insulin resistance/diabetes and atherosclerosis. Decreased adiponectin levels are thought to show a central protagonist in the development of type 2 diabetes, obesity and cardiovascular disease in humans.[9] So, our primary objective was to compare the levels of plasma adiponectin along with BMI and blood sugar in cases and controls and secondary to assess the association between obesity and adiponectin in the subjects with sleep disorder breathing patients with obesity.

Body mass index (BMI) screening indicates whether a person is underweight, healthy weight, overweight or they have obese. As per the WHO classification. The categories are: under low weight BMI (BMI<18.5), normal (BMI =18.5-24.9), pre obese or overweight (BMI =25-29.9) and obese (BMI>30). According to WHO publication, that there were 3 lakh deaths due to overweight/obesity in South East Asian region.[10]

The present study has two major finding; [Table 1] showing the comparison of adiponectin levels in the subjects with sleep disorder breathing patients with and without obesity, where we have observed significantly high levels of BMI and random blood glucose in the subjects with sleep disorder breathing patients with obesity than that of corresponding controls (P<0.001) [Table 1].

In the obese patients, visceral body fat affect many of health condition through an abnormal adipokines production. Adipokines play a vital role in energy metabolism, concentration of both total adiponectin and high molecular weight adiponectin decreases in subjects with obese patients and increases after weight loss.[11]

The present study assessed the mean levels of plasma adiponectin in the subjects with sleep disorder breathing patients with and without obesity and found a significantly decreased mean levels of plasma adiponectin in the subjects with sleep disorder breathing patients with obesity than that of sleep disorder breathing patients without obesity (P<0.001) [Table 1]. The present study was accordance with the study done by Masserini et al, (2006) they have that the adiponectin level was significantly reduced in obese patients compared with healthy normal weight subjects and adiponectin.[12]

The significant decrease in adiponectin levels in response to sleep restriction among the women is consistent with other evidence suggesting an inflammation-increasing response to sleep restriction, and it can also add evidence that short sleep durations are associated with risk for obesity and cardiovascular disease.

These findings indicate that decreased plasma adiponectin level is positively associated with the prevalence of SDB in obese populations. A prospective cohort study is warranted to evaluate whether the baseline and change of plasma adiponectin level over time is associated with the incidence of SDB in obese populations.[13]

A number of researchers have demonstrated a strong correlation between adiponectin levels and various metabolic abnormalities. The enlargement of adipose tissue including increase in size and cells number can also regulate the secretion of adiponectin. In the past few decades, a number of epidemiological studies have consistently showed an independent association between obesity and OSA. In our study, we have also assessed the relationship between BMI and adiponectin and found an inverse correlation between BMI and adiponectin (r= -0.63, P<0.05) [Figure 2]. Drolet et. al, (2009),[14] reported an inverse relationship between mean adipocyte diameter and adiponectin secretion. In another study Zhang et al, (2004),[15] compared the levels of adiponectin with the non-obese group and found significantly lower in the SDB with obese and adiponectin levels were also negatively correlated with AHI and BMI.

The overall prevalence of OSA was significantly higher in the obese subjects with obese patients also had equal proportion of moderate and severe SDB in patients with and without sleep disorder breathing.[16] Although the proportion of mild SDB was lower in

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cases than that of controls. [Figure 1]. This indicate that the proportion of mild OSA was significantly lower in the obese group. Pearson correlation analysis suggesting that losing weight may be favorable for SDB management which need further investigation in the future with large sample size. Despite compelling evidence supporting the independent relationship between obesity and OSA, the underlying mechanisms are still elusive. It has been speculated that compared with the lean control subjects, the obese populations commonly have more para-pharyngeal fat deposition which in turn causes upper airway caliber narrowing and collapsing. Moreover, through reduced lung volume, obesity results in decreased tracheal tug and increased airway resistance. Indeed, we observed that plasma adiponectin level was negatively correlated with BMI.[16]

CONCLUSION

Present study shows that the plasma adiponectin levels were gradually decreasing with the severity of the diseases. The plasma blood glucose and BMI were increased in the subjects of SDB with obese. Furthermore, inverse relationship between Adiponectin and obesity suggested that the low levels of adiponectin could be the predictive biomarker for the diagnosis of sleep breathing disorder with obesity.

Limitation of study:

There are two main limitations that we have considered such as availability of these kind of subjects is very difficult in rural area and another aspect is less sample size.

Abbreviation:

SDB (Sleep disordered breathing), BMI (Body mass index), PSG (polysomnography), CPAP therapy (continuous positive airway pressure).

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