Continuous Positive Airway Pressure Treatment: Effect on Serum Lipids in Patients with Obstructive Sleep Apnoea

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Abstract: Obstructive Sleep Apnoea (OSA) is a common disorder in adults. Its hallmark is repetitive episodes of partial or complete obstruction of the upper airway during sleep associated with increasing respiratory efforts. This leads to oxyhaemoglobin desaturation, sleep fragmentation, and daytime symptoms, mainly excessive sleepiness. Accumulating evidence suggests that intermittent hypoxia and oxyhaemoglobin desaturation may, irrespective of obesity, lead to elevation of serum lipids even in non-dyslipidaemic OSA patients. Continuous Positive Airway Pressure (CPAP) is the treatment of choice for OSA, since it eliminates upper airway collapse during sleep and improves sleep fragmentation, daytime symptoms and quality of life. Moreover, it has been proposed that the amelioration of breathing disturbances during sleep can improve several markers of the lipid profile, such as total cholesterol, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol as well as apolipoproteins A, B and C. Indeed, some studies have reported improvements in these parameters especially in CPAP adherent patients. However, other studies failed to confirm this beneficial effect. The present article reviews the issue whether CPAP treatment exerts a beneficial effect on lipids.

Keywords: Continuous positive airway pressure, cholesterol, lipid profile, obstructive sleep apnoea, triglycerides.

1. INTRODUCTION

Obstructive Sleep Apnoea (OSA) is a common disorder, affecting approximately 4% of adult men and 2% of adult women in the general population [1]. OSA is being increasingly recognised as a cause of morbidity and mortality [1].

OSA is characterised by repeated episodes of complete or partial obstruction of the upper airway during sleep. Airway obstruction increases respiratory effort, and leads to intermittent arterial oxygen desaturation, systemic and pulmonary arterial blood pressure alterations, and sleep fragmentation [1]. Main symptoms include nocturnal respiratory pauses, interrupted by loud snoring and excessive daytime sleepiness [1]. The gold standard for diagnosis is polysomnography, a simultaneous meticulous recording of multiple physiologic parameters, namely electroencephalogram, electrooculogram, electromyogram, oronasal airflow, chest wall and abdominal motion, body position, snoring, electrocardiogram and oxyhaemoglobin saturation [2].

The definitions of sleep-related respiratory disturbances have been clarified in recent years, particularly apnoea, hypopnoea and respiratory effort-related arousals (RERAs) [2]. Apnoea is the complete cessation of airflow lasting at least 10 sec accompanied by a decrease in oxygen saturation of at least 3% or by an EEG-recorded arousal; RERA refers to increasing respiratory effort, leading to an arousal from sleep, which does not meet the criteria for apnoea or hypopnoea [2]. Apnoea-hypopnoea index (AHI) is the total number of apnoeas and hypopnoeas per hour of sleep, and respiratory disturbance index (RDI) is the total number of events (e.g. apnoeas, hypopnoeas and RERAs) per hour of sleep [2]. RDI is generally higher than AHI, because the former includes RERAs, whereas the latter does not [2].

OSA is associated with increased cardiovascular morbidity and mortality [1, 2]. Although it was previously assumed that this was due to its relation with obesity, recent data suggest that OSA is independently associated with the cardiovascular risk factors that comprise metabolic syndrome (MetS); one of them being dyslipidaemia [1, 2].

Continuous Positive Airway Pressure (CPAP) is the treatment of choice for OSA [3]. It was proven to be efficacious in eliminating obstructive respiratory events during sleep, ameliorating sleep architecture, improving daytime sleepiness and quality of life [4,5], and reducing serum levels of cardiovascular risk factors, such as total cholesterol [6] and markers of systemic inflammation [7]. Unfortunately, patient adherence to CPAP treatment remains suboptimal and its use during sleep time shows substantial variation [8, 9].

Not only is CPAP the established treatment for OSA, but it may also have a favourable effect on the lipid profile in such patients. It has been postulated that CPAP use ameliorates in-
termitative hypoxia and reduces sympathetic nerve activity [10], which may account for an improved lipid profile. Furthermore, effective treatment of OSA may result in increased physical activity and a reduction of hyperinsulinenence during the day [11]. It is well known that physical activity can also improve serum lipid levels [12]. This additional therapeutic benefit is attracting considerable interest, but it is still under debate. Findings from numerous studies on the effect of CPAP treatment on lipids in OSA populations have been rather conflicting. This can be attributed to differences between the studied populations (i.e. dyslipidaemic, non-dyslipidaemic, obese or non-obese patients), the primary outcomes, the period of CPAP application (ranging between 1 month and 1 year) and patient adherence to CPAP use.

The present article provides a review of the current evidence whether CPAP treatment exerts a beneficial effect on lipid profile.

2. METHODOLOGY: SEARCH STRATEGY AND ENDPOINTS

Our search strategy was based on the PubMed, Medscape, Embase and Google scholar databases up to May 2011 using combinations of the following keywords: Continuous positive airway pressure; lipid profile; dyslipidaemia; cholesterol; obstructive sleep apnoea. It included all types of articles (randomised controlled trials, original studies, review articles) written in English.

Treatment effect was assessed by evaluating the impact on serum lipids. Lipid parameters included cholesterol, triglycerides (TGs), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), apolipoproteins (apo) A, B and C. In many cases, lipid measurements were determined by enzymatic methods using commercial kits. Determination of cholesterol was performed after enzymatic hydrolysis and oxidation. LDL-C was derived using the Friedewald equation.

3. ASSOCIATION OF OSA WITH DYSLIPIDAEMIA

The link between OSA and lipid metabolism still remains to be defined. The repetitive episodes of upper airway obstruction that are characteristic of OSA result in intermittent hypoxia and large swings in intra-thoracic pressure that trigger autonomic responses, and sympathetic overactivity has been reported in patients with OSA [2]. There is a direct link between the adrenergic system and lipid metabolism and the chronic elevated sympathetic activity in OSA patients may lower HDL-C and increase serum TG levels [13, 14]. Metabolic and atherosclerotic changes have been previously shown in mice exposed to chronic intermittent hypoxia (IH) [15]. It has been demonstrated in mice that IH induces hypercholesterolaemia by increasing lipoprotein secretion via upregulation of a key hepatic enzyme, stearoyl-coenzyme A desaturase-1 (SCD-1) [16-18].

The association between OSA and lipid profile has been studied with conflicting results. Some findings suggest that OSA is independently associated with lipid abnormalities, while others show that dyslipidaemia is associated with obesity and not directly to OSA.

In a cross-sectional analysis of a selected group of males with and without OSA, Kono et al. [19] reported that OSA was associated with dyslipidaemia in non-obese patients, independent of visceral fat obesity. Borgel et al. [20] demonstrated an influence of OSA on HDL-C levels. In their study, an independent association was found between the change in AHI and the change in HDL-C or triglycerides, respectively. Can et al. [21] found that OSA is associated with increased lipid levels. Total cholesterol, LDL-C, TGs and apolipoprotein B values were increased in patients with OSA compared with controls (without OSA, p<0.05). Iesato et al. [22] reported that circulating lipoprotein lipase concentrations, which are negatively correlated with TG concentrations and positively correlated with HDL-C concentrations, were lower in OSA patients compared with those in non-OSA patients. Tan et al. [23] demonstrated that OSA subjects had greater degree of HDL dysfunction (p<0.01) and increased oxidised LDL levels (p<0.05) compared with controls and that AHI was the main determinant of HDL dysfunction in OSA. In the Sleep Heart Health Study (SHHS), there was an independent association between the severity of OSA and HDL-C levels in females only, while there was only a minor but significant association with total cholesterol levels in males. These findings were evident in the age group <65 years after adjustment for co-variates [24].

Gruber et al. [25] reported that OSA is associated with MetS independently of obesity, predominantly due to increased TG levels. Zgierska et al. [26] found an independent, positive correlation (p<0.05) between TG levels and AHI, suggesting that OSA increases the risk of coronary artery disease by increasing plasma TGs, independently of obesity. Limited case-control data suggest that OSA is associated with a pattern of dyslipidaemia typical of the MetS. Coughlin et al. [27] demonstrated that OSA was independently associated with decreased HDL-C, increased cholesterol:HDL-C ratio and higher TG concentrations. Previous studies have also demonstrated that OSA is independently associated with dyslipidaemia [28, 29].

On the other hand, Mc Ardle et al. [30] reported an increase in LDL-C and total cholesterol among patients with OSA, a result partially explained by the presence of central obesity in these patients. Furthermore, Sharma et al. [31] found that OSA has no independent association with lipid abnormalities and that obesity was the major determinant of metabolic abnormalities in obese subjects with OSA.

4. EFFECT OF CPAP ON SERUM LIPIDS: POSITIVE STUDIES

Several studies have provided evidence for the benefits of CPAP treatment on the lipid profile of OSA patients (Table 1). Chin et al. [32] studied the changes in lipid levels before and after 8 months of CPAP treatment in 22 OSA patients in correlation with weight reduction, defined as BMI change >1 kg/m² from baseline. In the group without body weight reduction (n=13), significant changes in HDL-C (p=0.013) and LDL-C (p=0.046) levels occurred. In the group with body weight reduction (n=9), there was a significant improvement in HDL-C (p=0.025), LDL-C (p=0.011), TGs (p=0.028), Apo A-II (p=0.008) and Apo C-II (p=0.021) serum levels [32].

Ip et al. [29] examined the effect of 6-month CPAP treatment on leptin, TGs and cholesterol levels among patients with
Table 1. Effect of CPAP Treatment on Serum Lipids: Positive Studies

| Author, Year   | No of patients | Control Group/ Adherence to CPAP (h/night) | Baseline Lipid Profile | Age (Years) | BMI (Kg/m²) | AHI/h) | ESS | Duration | Methods | Results                                                                 |
|----------------|----------------|---------------------------------------------|------------------------|-------------|-------------|---------|-----|----------|---------|-------------------------------------------------------------------------|
| Chin et al. 1999 | 22             | No body weight reduction (n=13), body weight reduction (n=9) | Baseline lipid profile | 46.2±3.7, BWR: 50.8±3.7 | 28.5±0.8, BWR: 31.2±1.7 | 52.7±5.8, BWR: 63.5±5.8 | 8 months | TC, triglycerides, HDL-C, LDL-C, apoA-I, apoA-II, apoB, apoC-II, apoC-III, apoE | NBWR: improvement in HDL-C and LDL-C, BWR: improvement in triglycerides, HDL-C, LDL-C, apoA-II, apoC-II, apoC-III, apoE |
| Ip et al. 2000   | 60             | Yes                                         | Baseline lipid profile | 43.6±10.1, control: 41.9±10.4 | 27±2.9, control: 26.5±2.1 | 35.7±18, 1.8±1.9 | 6 months | TC, triglycerides, HDL-C, LDL-C, TC/HDL-C ratio | Decrease in triglycerides levels after CPAP treatment |
| Buechner et al. 2001 | 95         | Effective treated group/ ineffective treated group | LDL-C levels ≥ 130 mg/dl | 56.6±9.5 | 35.9±6.3 | 35.6±7.6 | 16.2±3.3 | 6 months | TC, triglycerides, HDL-C, LDL-C, apoB, and lipoprotein (a) levels | Decrease in TC and LDL-C in effective treated group |
| Robinson et al. 2004 | 220        | Yes/ Subtherapeutic CPAP 4.1±2.4, therapeutic CPAP 5.0±1.9 | Mean baseline levels of TC within the normal range in both groups | 49.1±10.3, 49.7±10.3 | 35.9±6.3 | 35.6±7.6 | 16.2±3.3 | 1 year | TC, triglycerides | Reduction in TC in therapeutic CPAP group |
| Borgel et al. 2006 | 127         | 9.4% were under lipid lowering medication | Baseline lipid profile | 55.7±10.6, 31.6±5.9 | 32.9±21.5 | 16.2±3.3 | 6 months | TC, triglycerides, HDL-C, LDL-C | Improvement in TC, triglycerides, HDL-C, LDL-C levels in patients with initial abnormal lipids levels |
| Dorkova et al. 2008 | 32           | No/ Good adherence group (5.1±1.2), Poor adherence group (1.9±1.1) | Baseline lipid profile | 53.7±9.6, 35.1±6.1 | 64±20.8 | 13.3±4.6 | 8 weeks | TC triglycerides, HDL-C, LDL-C, apoA-I, apoB | Reduction in TC and apoB in the good adherence group |
| Barcelo et al. 2008 | 44            | Yes/ OSA with EDS (n=22), OSA without EDS (n=22) | Baseline lipid profile | 50±7.4 | 32.3±4.7 | 11.3±5.7 | 8 weeks | TC, triglycerides, HDL-C, LDL-C, VLDL-C | Reduction in TC and LDL-C |
| Oktay et al. 2009 | 20             | No                                           | Baseline lipid profile | 50±7.4 | 32.3±4.7 | 11.3±5.7 | 8 weeks | TC, triglycerides, HDL-C, LDL-C, VLDL-C | Reduction in TC and LDL-C |
| Cuhadaroglu et al. 2009 | 31         | Mean CPAP use >4h/night                     | Baseline lipid profile | Range: 28-76 | Range: 16-90 | 11.3±5.7 | 8 weeks | TC, triglycerides, HDL-C, LDL-C, VLDL-C | Reduction in TC and LDL-C |
severe OSA (AHI: 35.7±18). They found a significant decrease in serum leptin (p=0.012) and TGs (p=0.017), while no significant change was observed in serum cholesterol.

Buechhner et al. [33] studied the treatment effect on lipid levels in patients with OSA. They demonstrated a significant decrease in total cholesterol and LDL-C levels (p<0.001) after 6 months, but this occurred only in patients effectively treated with CPAP.

In a meta-analysis of 2 randomised placebo-controlled trials in OSA patients, Robinson et al. [34] compared the effect of therapeutic and subtherapeutic CPAP treatment on cholesterol and TGs. Despite the short treatment duration (1 month), there was a significant reduction in total cholesterol levels among patients receiving therapeutic CPAP (p=0.001), whereas no such change was observed among those receiving subtherapeutic CPAP. No significant changes were observed in serum levels of TGs in either group [34].

In a large population-based study, Borgel et al. [20] demonstrated a small but significant increase in HDL-C (p<0.013) after 6 months of CPAP therapy in 127 OSA patients (AHI: 32.9±21.5). Furthermore, the authors observed significant improvements in serum levels of HDL-C (p<0.001), LDL-C (p<0.001), total cholesterol (p=0.001) and TGs (p=0.01) in OSA patients with baseline abnormal lipid/lipoprotein serum levels [20].

Dorkova et al. [35] examined the effect of 8-week CPAP therapy in 32 patients with severe OSA and MetS, as well as the role of adherence to therapy. Adequate adherence was defined as use of CPAP more than 4h/night, while poor adherence was defined as use of CPAP for less than 4h/night. They observed a significant reduction in total cholesterol (p=0.02) and apoB (p=0.009) in patients with adequate adherence to treatment [35]. Conversely, no significant changes were noted in the non-compliant group.

Barcelo et al. [36] studied 35 patients with OSA± excessive daytime sleepiness (EDS) who underwent CPAP treatment for 3 months. They found a significant decrease in cholesterol only in patients with EDS.

In an observational prospective cohort study, Oktay et al. [37] investigated the effect of 1-year CPAP treatment in patients with OSA. No significant difference was found in TG levels after treatment. However, a significant increase was observed in HDL-C (p=0.001) [37].

The study of Cuhadaroglu et al. [38], examining the effects of 8 weeks of CPAP therapy, reported a reduction in total cholesterol (p<0.05) and LDL-C (p<0.05) in 31 patients with moderate to severe OSA who used CPAP for > 4h/night.

Finally, in a study from our centre [6], we examined the effect of compliance to CPAP therapy on serum lipids, demonstrating significant improvements in total cholesterol (p=0.021), total cholesterol/ HDL-C ratio (p=0.018) and ApoB/ ApoA-I ratio (p=0.021) after 6 months [6]. Again, reductions were only achieved in patients who were adherent to CPAP use (≥4h use per night).

5. EFFECT OF CPAP ON SERUM LIPIDS: NEGATIVE STUDIES

There are several studies suggesting that CPAP treatment does not improve serum lipids (Table 2). Davies et al. [39] compared serum lipids of 15 not previously treated OSA patients and 18 snorers, in order to examine their association with coronary heart disease. OSA patients received CPAP treatment for more than 3 months. Levels of total cholesterol, TGs, very low density lipoprotein cholesterol (VLDL-C), LDL-C, and HDL-C of snorers and OSA patients were measured before and after CPAP treatment. There was no significant difference in the abovementioned serum lipids when OSA patients before treatment and snorers were compared [39]. Furthermore, no change was observed in OSA after CPAP treatment [39].

In another study in 43 OSA patients [40], it was reported that cholesterol levels were neither elevated at baseline compared with controls nor reduced by CPAP.

Al-Shaer et al. [41] compared the changes in lipid profile between OSA patients receiving CPAP treatment (n=34) and those not receiving CPAP (n=28). After 126±33.7 days of follow-up, there was no change in lipid levels of the CPAP group, nor any difference between the 2 groups.
Lattimore et al. [42] studied 10 patients with newly diagnosed untreated moderate to severe OSA (AHI > 15/h) under autoCPAP treatment for 3 months. Total cholesterol, LDL-C and HDL-C were all within normal limits before CPAP treatment. There were no significant changes in serum lipids after autoCPAP treatment, but this may be due, partly at least, to the small number of subjects.

Kitahara et al. [43] found that CPAP treatment had no significant effect on serum total cholesterol at 2 months and 4 months after initiation of CPAP in patients with OSA.

In a double blind randomised controlled trial of therapeutic and sham CPAP for 3 months in men with type 2 diabetes and OSA, West et al. [44] found no significant changes in any lipid parameter. No differences before and after treatment were noted in terms of total cholesterol, HDL-C, and TGs in either group.

Drager et al. [45] studied the lipid profile of 24 patients with severe OSA and mild dyslipidaemia, free of comorbidities who received either no treatment (n=12) or CPAP (n=12). No significant changes were observed in blood cholesterol, TGs, HDL-C, LDL-C in both groups, showing that effective

### Table 2. Effect of CPAP Treatment on Serum Lipids: Negative Studies

| Author, Year | No of Patients | Control Group/Adherence to CPAP (h/night) | Baseline Lipid Profile | Age (Years) | BMI (Kg/m²) | AHI (/h) | ESS | Duration | Methods | Results |
|--------------|----------------|------------------------------------------|------------------------|-------------|-------------|----------|-----|----------|---------|---------|
| Davies et al. 1994 | 10 | Yes | No dyslipidaemia | | | | | >3 months | TC, triglycerides | No difference |
| Barcelo et al. 2004 | 43 | Yes | | | | | | | TC | No difference |
| Al-Shaer et al. 2005 | 62 | CPAP (n=34) | TC, LDL-C and HDL-C within normal limits | 49±8 | 31 (range 24-41) | 39 (range 15-104) | | 3 months | TC | No difference before and after CPAP treatment |
| Lattimore et al. 2006 | 10 | No | | 8.6±1 | | | | 4 months | TC | No difference before and after CPAP treatment |
| Kitahara et al. 2006 | 17 | No | | | | | | | | |
| West et al. 2007 | 42 | Therapeutic CPAP (n=20) Placebo CPAP (n=22) | Mild dyslipidaemia, borderline high levels of LDL-C | 58 (range 29-74) | 36.6 (range 26.2-49.2) | 14.7±3.5 | 36.8 (range 29.2-47.1) | 13.6±3.5 | 3 months | TC, triglycerides, HDL-C | No difference before and after CPAP treatment |
| Drager et al. 2007 | 24 | Yes | | | | | | 4 months | TC, triglycerides, HDL-C, LDL-C | No difference before and after CPAP treatment |
| Coughlin et al. 2007 | 34 | Crossover trial: Sham CPAP 2.6 (0-7.5) Therapeutic CPAP 3.9 (0-7.4) | | 49±8.3 | 36.1±7.6 | 13.8±4.9 | | 6 weeks | TC, triglycerides, HDL-C, LDL-C | No difference before and after CPAP treatment |
| Li et al. 2009 | 20 | No | | | | | | 90 days | TC, triglycerides, HDL-C, LDL-C | No difference before and after CPAP treatment |
treatment of OSA with CPAP for 4 months did not significantly improve the lipid profile. Patients and control subjects presented with high borderline levels of LDL-C.

In a randomised blinded crossover trial in 34 obese Caucasians with symptoms indicating OSA, Coughlin et al. [46] compared the effect of 6-week therapeutic vs sham CPAP on serum lipids. No change was observed in terms of lipids in either group. Specifically, in the therapeutic CPAP group, there was no significant change in cholesterol (p=0.07), TGs (p=0.33), HDL-C (p=0.15) and LDL-C (p=0.66) after CPAP treatment.

Li et al. [47] examined the effect of 90 days of CPAP treatment in 20 patients, with moderate to severe OSA, who received nasal CPAP treatment, lasting 6 to 8 h per night. Comparison before and after CPAP treatment showed no significant changes in blood cholesterol, LDL-C, HDL-C and TGs.

6. EFFECT OF CPAP ON SERUM LIPIDS: CURRENT SITUATION AND IMPLICATIONS FOR FURTHER RESEARCH

Current evidence suggests that CPAP may exert a beneficial effect on lipid profile in patients with OSA. However, this effect is not definitely proven. Indeed, there is data indicating that CPAP may fail to produce favourable changes. Based on the positive studies, CPAP may reduce cholesterol levels in both patients with normal initial [34] and abnormal lipid levels [20]. In the former, CPAP may improve total cholesterol, TGs, HDL-C and reduce LDL-C levels. Importantly, favourable changes are observed only in OSA patients with sufficient adherence to CPAP treatment [6, 35]. Nonetheless, negative studies question the aforementioned beneficial effects [39-47]. It must not escape our notice, though, that some of the studies may be criticised for their small sample size [39, 42] and their relatively short follow-up period [34, 46]. Other limitations of the studies are the poor patient adherence to treatment [29] and the missing information regarding diet or physical activity [20].

Clearly, the role of CPAP in the amelioration of serum lipids has not been defined. Results are rather conflicting, which may be explained by the differences in recruited populations, as well as in study design and endpoints. Indeed, some works have included dyslipidaemic patients with OSA, under medication or not, while others patients with normal baseline lipids levels. The differences in recruited population and baseline characteristics between the subjects of the studies may produce contrasting results. Endpoints have also been variable, including various parameters of lipid profile. Of greater significance, follow-up periods varied significantly, ranging from 1 month [34] to 1 year [37].

Hence, further research is necessary to re-examine the effect of CPAP on lipids and especially to compare the beneficial effect of CPAP with that of statin administration. The latter class of agents is the established treatment for hyperlipidaemia, while it is also increasingly being appreciated for its valuable pleiotropic effects [48]. It is important to define which patients stand to benefit and how long the duration of treatment is, in order to produce favourable changes. Moreover, the magnitude of the effect needs to be re-evaluated in comparison with the initial health status of patients, their adherence, lipid lowering medication, diet and the weight changes. Two further issues to address would be the potential favourable effect of CPAP on weight and insulin sensitivity. Regrettably, these two parameters have largely been ignored in the studies included, and so it is difficult to determine whether any of the observed effects on lipids could be, at least partly, attributed to or mediated by such changes.

All aforementioned issues should be addressed by large-scale randomised controlled trials. It is the authors’ opinion that the accumulating evidence for a positive effect of CPAP on lipids is very promising and warrants careful investigation.

7. CONCLUSIONS

The potential benefit of CPAP treatment on the lipid profile is of interest. However, it still remains under debate, due to the largely inconsistent results from numerous studies, recruiting populations with different characteristics. This disparity is, partly at least, explained by the differences in studied populations, study design and primary outcomes. Such disparity frequently renders meaningful conclusions difficult, as already shown for the effect of CPAP on glucose metabolism [49]. Indeed, the major limitations of most studies on the effect on lipids are the short follow-up period and the fact that adherence to CPAP treatment, a newly recognised factor affecting CPAP efficacy [50, 51], was overlooked. Furthermore, the small size of the study samples, the diversity of the patients, the validity of the placebo treatment and the missing information about the physical activities of the subjects may weaken study results. Hence, there is a clear need for large-scale randomised, controlled studies with better adherence to therapy and long-term follow-up, which will allow a definite conclusion regarding the effect of CPAP on the lipid profile. Such interventional studies will provide an answer to the question whether OSA is responsible for alterations in the lipid profile. In the meantime, CPAP still remains the established treatment for OSA and future research may, possibly, confirm an additional beneficial effect on lipids.

DECLARATION OF INTEREST

None

ABBREVIATIONS

AHI = Apnoea-hypopnoea index
Apo = Apolipoprotein
BMI = Body mass index
CPAP = Continuous positive airway pressure
EDS = Excessive daytime sleepiness
ESS = Epworth sleepiness scale
HDL-C = High-density lipoprotein cholesterol
IH = Intermittent hypoxia
LDL-C = Low-density lipoprotein cholesterol
MetS = Metabolic syndrome
OSA = Obstructive sleep apnoea
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