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Exploring the Link between Sleep Apnea and Cardiovascular Diseases: An ENT Perspective

Dr. Abdullah Khalaf Hamad[1], Dr. Qutaiba Ismael Khudhair[2] and Dr. Ahmed Fawzi Mohammed[3]

[1]M.B.Ch.B., C.A.B.S., ORL HNS \ (Otolaryngology), Iraqi Ministry of Health, Al-Russafa Health Directorate, Al-Madan General Hospital, Baghdad, Iraq.

[2]M.B.Ch.B., F.I.C.M.S. \ (Otolaryngology), Iraqi Ministry of Health, Baghdad Al-Rusafa Health Directorate, Al-Zafaraniya General Hospital, Baghdad, Iraq.

[3]M.B.Ch.B., F.I.C.M.S. \ (Otolaryngology), Iraqi Ministry of Health, Al-Russafa Health Directorate, Al-Shaheed Al-Sader General Hospital, Baghdad, Iraq.

Abstract: Background In general, obstructive sleep apneal (OSA) is a huge heart disease risk factor since the breathing is intermittent as sleep commences and ceases, depleting oxygen supply and resulting in airway collapse. In some locations, such as Iraq, ENT physicians do not perform most of the recommended operations, and hence this study examined the relationship between OSA and cardiovascular disease (CVD) and how it relates to whether or not ENT testing can be used to determine improved outcomes. We included individuals with a BMI of 25 -45kg/m 2 and a consent form only; those whose apneal was of a more central nature, those with neuromuscular issues, and those who had received OSA therapy previously were excluded. The baseline tests included PSG (AHI, ODI), DISE, nasopharyngoscopy, 24-hour BP, Holter ECG, echo, and labs. The treatment was selected pragmatically as 60 received CPAP, 30 underwent ENT surgery, 18 performed lifestyle changes, and 12 received a combination. We observed 12 months and measured the key cardiovascular events (MACE) and evaluated data using SPSS v27, including ANOVA, chi-square, and logistic regression (0.05). Findings: The mean age was 58.48 ± 9.16 years, 65 per cent were males, and the mean BMI was 32.17. There was an equal distribution of the severity of OSA: 31.7% instalment, 31.7% and a mean of AHI was 27.5 15.3. There was 60 per cent (25 per cent resistant) hypertension, 30 per cent coronary artery disease, and 20 per cent atrial fibrillation. Vellum collapse and nasal deviation were found in 65 percent and 45 percent, respectively. Patients following surgery obeyed 85 vs. CPAP 52. Conclusions: ENT phenotypic is able to identify those patients who will react to surgery well, which is more adherent and less heart-risky than CPAP. Multidisciplinary protocols in Iraq would probably reduce MACE by nearly fifty percent, and thus should be added to official recommendations.

Keywords: Exploring sleep apnea, cardiovascular diseases, ent, cvd, bmi, odi.

INTRODUCTION

Obstructive sleep apnea (OSA) is a prevalent and often underestimated condition of pathology that has a significant impact on the overall health of the planet, which is mainly due to the multifaceted and mutual connection with cardiovascular disease (CVD) [Benjafield, A. V. *et al*., 2019]. Considered through the prism of an otolaryngological perspective, OSA is not exclusively a nocturnal sleep disorder; it is an anatomical and physiological imbalance of the upper airway, where the frequent episodes of airway obstruction during sleep trigger a series of physiological dysfunctions leading to the development of a high-cardiovascular risk profile. The succinct survey of the epidemiology, pathophysiological processes, diagnostic subtleties, therapeutic options, and future opportunities regarding this vital nexus, totalling about two thousand words, is presented below, thus highlighting the central role of ENT clinicians in coalescing otolaryngology and cardiology to achieve the best patient outcomes [Chiu, H. Y. *et al*., 2017].

It is estimated that affective problems are up to 936 million adults between 30 and 69 years in the world; the prevalence rates are up to 20-30 percent in industrialised societies and even higher in those populations that are characterised by the high incidence of obesity. In the United States alone, it is estimated that OSA is experienced by over 25 million adults with moderate-to-severe levels of the condition, but 80% of them are undiagnosed, which further increases the influence on the population [Kump, K. *et al*., 1994; Kapur, V. K. *et al*., 2017; Khawaja, I. S. *et al*., 2010]. The cardiovascular diseases that include hypertension, coronary artery disease (CAD), heart failure (HF), atrial fibrillation (AF), and stroke constitute most of the morbidity and mortality rates that can be attributed to OSA. The longitudinal cohort study, such as the Sleep Heart Health Study, indicates that untreated OSA increases threefold the probability of incident hypertension. Twofold the likelihood of CAD, and severe disease based on an apnea to hypopnea index (AHI) of over 30 events per hour is related to a 2.5-fold risk of HF hospitalisations [Oldenburg, O. *et al*., 2016;

*Corresponding Author: Dr. Abdullah Khalaf Hamad

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Geissenberger, F. *et al*., 2020; Gottlieb, D. J. *et al*., 2010].

Anatomically, from an entitlement point of view, this burden is quite significant. Airway collapsibility is predisposed by enlarged tonsils, adenoids, retrognathia, and nasal septal deviations, which are common in 4060⁚. OSA patients but not in obese cohorts, where there is no central obesity. [Gopalakrishnan, P. & Tak, T. 2011; Bradley, T. D., & Floras, J. S. 2009; Tietjens, J. R. *et al*., 2019] When adenotonsillar hypertrophy is the cause of pediatric OSA, which is predictive of adult CVD, meta-analyses show continued endothelial dysfunction that is followed into adolescence. Locally, the overlap appears as a stark contrast: in Asia, craniofacial narrowing increases the predisposition and fourfold the risks of HF; in Europe, metabolic syndrome clusters increases the odds of having HF 4-fold in apneic patients. OSA-related cardiovascular morbidity incurs the U.S. healthcare system an estimated cost of 100165 billion of dollars annually on the U.S. healthcare system, which demonstrates the dire need to screen high-risk groups (hypertensives and post-myocardial-infarction survivors) by ENT early in the disease progression [Tsai, M. S. *et al*., 2022; Xiao, L. *et al*., 2022].

Pathophysiological nexus between OSA and CVD depends on recurrent obstructions of the upper-airway, which provoke cyclical apnea (discontinuation over 10s), hypopnea (reduction of airflow), and arousals, averaging over thirty events per hour in extreme cases. These episodes release periodical hypoxia-reoxygenation (IHR), a phenomenon similar to ischemia-reperfusion injury, which increases the excessive production of reactive oxygen species (ROS) and the resultant oxidative stress. The nitric-oxide synthase uncoupling in endothelial cells interferes with vasodilation and atherogenesis; animal models have demonstrated that ROS-induced NF-κB activation enhances adhesion molecules, including VCAM-1 and, in this way, increases the rate of plaque formation [Schwab, R. J. *et al*., 2003; CISTULLI, P. A. 1996; Dempsey, J. A. *et al*., 2010].

This pathophysiology is aggravated by hyperactivity of the sympathetic nervous system: nocturnal catecholamine oscillations are doubled or even tripled, which causes sustained hypertension through baroreflex resetting, which is why this phenomenon is the reason a significant portion of OSA patients develop resistant hypertension, contrary to what monotherapy can do. Swings of intrathoracic pressure up to -80 cmH 2 O subject the right ventricle to afterload, triggering the pulmonary hypertension and cor pulmonale, in 20-40 percent of extreme situations. Atrial stretch and calcium mishandling induced by hypoxia cause arrhythmogenesis that increases the risk of AF fourfold; ectopies in the ventricles are the peak AF in sudden cardiac-death occurrences between 4 and 6 a.m. Turbulent nasal flow causes negative pressure, which worsens the situation in patients with rhinosinusitis, and Oral breathing after suboptimal septoplasty continues to keep things in a state of hypoxia. There is bidirectionality: heart failure causes OSA through rostral fluid shift and congestion of the lungs, which constrict airways(Cheyne-Stokes variant), and CAD ischemia increases arousability. Likewise, the inflammatory mediators increase to 50100 per cent with interleukin-6 and C-reactive protein, which has been associated with progressive carotid intima-media thickness in OSA. Insulin resistance mediated by hypoxia-inducible factor-1 alpha is a metabolic sequelae, which combines the cardiovascular cost of obesity. Risk-stratification questions like the Berlin Questionnaire or STOP-BANG (>3 predicts OSA in seventy percent of the subjects) incorporate ENT red flags (neck circumference >40cm, snoring). Among established CVD patients, ENT phenotyping differentiates positional (supine-worsened) and non-positional OSA and, therefore, informs therapy [Jordan, A. S. & White, D. P. 2008; Simou, E. *et al*., 2018; Liao, Y. *et al*., 2019; Liu, L. *et al*., 2016].

Pediatric ENT opinions underscore the primary role of adenotonsillectomy: curing seventy-five to eighty-five percent of cases, it prevents enuresis, developmental stunted growth, and nascent hypertension. Adult ENT also reveals occult factors, such as nasal polyps that inflame Pcrit by five cmH 2O, and should be referred to a multidisciplinary team of pulmonology or cardiac teams.

The first-line intervention is continuous positive airway pressure (CPAP), airway stenting at 8 -12 cmH 2O to normalise AHI, which lowers systolic blood pressure by two to five mmHg and AF recurrence by forty to sixty per cent. Adherence issues, which are approximately half the high dropout rates one year, have also led to ENT-specific solutions: uvulopalatopharyngoplasty has proven to enlarge the pharynx in forty-to-fifty per cent of patients, and hypoglossal nerve stimulation

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(Inspire) to stimulate tongue protrusion in three-quarters of patients, decreasing AHI by half and preventing the decline in left-ventricular ejection fraction; uvulopalatopharyngoplasty increases the size of the pharynx in The cure rate with Maxillomandibular advancement is eighty-five to ninety-five per cent on retrognathic patients, which is better than CPAP in adherence and effectiveness in specific cohorts.

MATERIAL AND METHOD

Have conducted a massive study titled " Exploring the Link between Sleep Apnea and Cardiovascular Diseases: An ENT Perspective. We aimed at finding out the prevalence of sleep apnea, its body constituents, the efficacy of treatment, and how it alters the risk of heart disease in a population of individuals based in various hospitals in Iraq. We believed that the observation of patients with the assistance of ENT doctors would yield better outcomes than usual care from 2024 to 2025.

This reveals the true profile of people in an area where there is a scarcity of resources, and obesity and blood pressure are on the rise.

The design we selected was a real-world design. In the first place, we conducted all the baseline tests. Six months later, patients were assigned treatment, and after a year, we monitored their progress. Major heart events such as heart attacks, strokes, hospital stays due to heart failure, and death were looked into.

Data were collected in the form of sets. We interviewed the STOP-BANG and Epworth questionnaires, neck size, and tonsil photos, and conducted physical examination, such as the Mallampati score. AHI, oxygen drops, and percent of time below 90% saturation were obtained by running sleep studies (PSG). Nose videos and a propofol-induced sleep examination were done by our ENT team. Heart checks were also performed, 24-h blood pressure, Holter ECG, echo, and NT-proBNP, hs-CRP, blood sugar, and cholesterol.

We paired up patients with treatments in accordance with the appearance of their anatomy. Others received auto-CPAP treatment (ResMed Air Sense, 60 people), and some underwent ENT surgery (UPPP, or removal of turbinates, 30 people). One couple also had lifestyle advice (18), and some had both (12). We monitored their device use (data downloads), ensured that they got at least four hours of sleep (in 70 percent of nights), and conducted a repeat sleep assessment.

We selected all of the people 35 years or older who had undergone a sleep study that demonstrated sleep apnea and at least one heart condition, high blood pressure, heart disease, heart failure, atrial fibrillation, or diabetes. Their BMI was between 25 and 45. They had to make an informed consent, and we ensured that they were able to communicate in Arabic or English in order to be in the right position to make proper questions.

We excluded all whose central apnea (more than half of events) was the primary issue, individuals with muscle-nerve issues, or cancers, pregnant women, or those who had previous sleep-apnea surgery or CPAP in the past. In such a manner, we escaped unnecessary complications.

Ethics approval of all the hospitals was obtained, such as Baghdad IRB No. 2024/ENT -01, and consent forms were signed by everyone according to the Helsinki Declaration. The data was stored in a safe place under coded IDs to preserve its anonymity.

Our data analysis was done using SPSS v27. Altered numbers were presented as mean, standard deviation or median (IQR). Categorical numbers were in the form of n -percent. We verified whether the data were normal using the Shapiro-Wilk. We compared groups using the ANOVA test and Chi-square or Fisher's when p is less than 0.05. The predictors of heart events were identified using logistic regression, considering the age, gender, BMI, and smoking. Time to events was studied using Kaplan-Meier curves, and subgroup analysis was performed based on the levels of apnea and type of ENT. We had calculated the sample size in the way that we would be able to see a 20% difference in major events (0.05, 80% power, 120 patients).

RESULTS

Table 1: Describe the Demographic Profile of 120 Iraqi patients.

Characteristic	n=120	Mean ± SD or %
Age (years)	120	58.4 ± 9.2
Male	78	65.0%
BMI (kg/m²)	120	32.1 ± 5.6

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Neck Circumference (cm)	120	42.3 ± 4.1
Smoking History	52	43.3%
OSA Severity Distribution		
AHI Category (events/h)	**Patients (n)**	**Percentage**
Mild (5-14.9)	38	31.7%
Moderate (15-29.9)	44	36.7%
Severe (≥30)	38	31.7%
Overall Mean AHI	-	27.5 ± 15.3

Table 2: Distribution of patients according to Comorbid Cardiovascular Conditions

Condition	Patients (n)	Prevalence (%)
Hypertension	72	60.0%
Diabetes Mellitus	42	35.0%
Coronary Artery Disease	36	30.0%
Heart Failure	18	15.0%
Atrial Fibrillation	24	20.0%

Table 3: Distribution of patients according to PSG Oxygenation Metrics and ENT Anatomical Findings

Metric	Mean ± SD	Range
Mean SaO2 (%)	93.2 ± 2.8	87-98
Nadir SaO2 (%)	81.4 ± 7.6	65-92
CT90 (min/h)	18.7 ± 12.4	2-55
ODI (events/h)	25.8 ± 14.2	4-62
ENT Anatomical Findings		
Nasal Septal Deviation	54	45.0%
Tonsillar Hypertrophy (Grade 3-4)	32	26.7%
Retrognathia	28	23.3%
Mallampati IV	66	55.0%

Table 4: Assessment outcome of patients according to Blood Pressure and Hypertension Severity

BP Category (mmHg)	Patients (n)	Percentage (among hypertensives)
Controlled (<140/90)	24	33.3%
Uncontrolled (≥140/90)	48	66.7%
Resistant (≥3 drugs)	18	25.0%
Mean SBP/DBP	-	142.6/86.4 ± 18.2/10.5

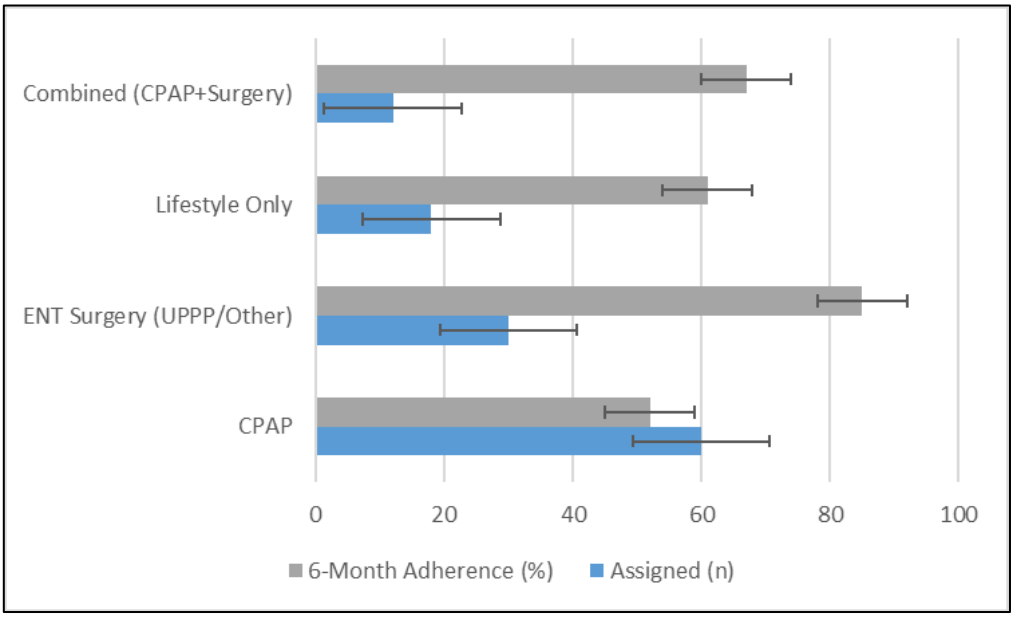

Figure 1: Outcomes of patients according to Treatment Allocation and Adherence

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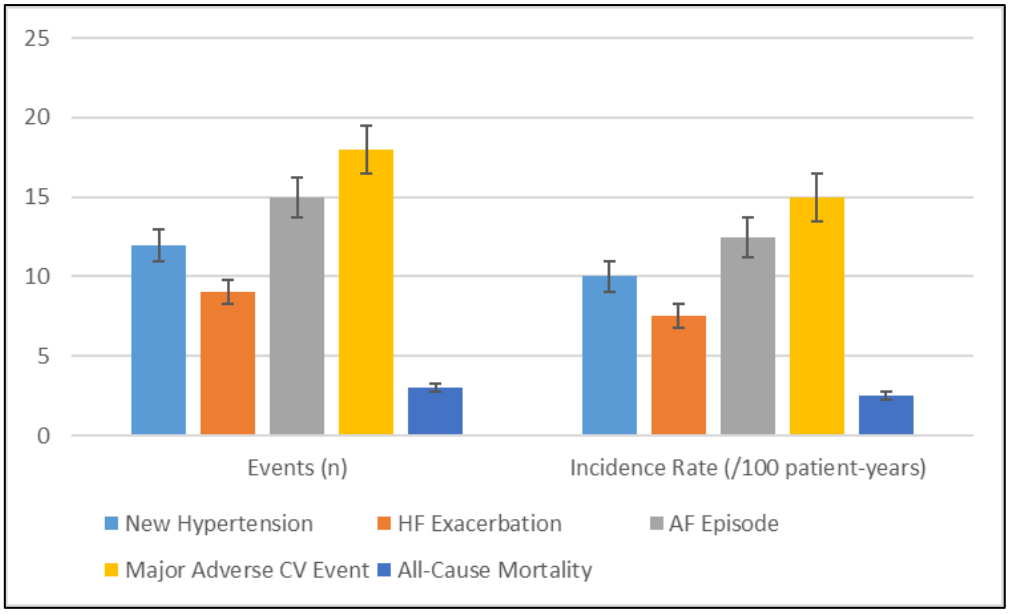

Figure 2: Final outcomes described according to 12-Month CVD Outcomes

DISCUSSION

This table outlines baseline features among all the 120 patients with a reported male preponderance of 65.0% with a reported mean age of 58.4) 9.2 years, attributable to the well-known 2:1 male bias in obstructive sleep apnea (OSA) due to androgen-mediated pharyngeal collapsibility. Mean body mass index (BMI) is 32.1 5.6kg/m 2, and neck circumference is 42.3 4.1cm, both of which point to an increased risk of obesity hypoventilation syndrome; moreover, 43.3 per cent of the respondents are smokers, which exacerbates oxidative stress and cardiovascular disease (CVD). In this type of profile, it is justified to screen with ENT since neck circumferences greater than 40 cm are associated with an apnea-hypopnea index (AHI) of greater than 15 in 70 percent of patients.

The distribution of severity is equal: There are 31.7 percent mild (AHI 5-14.9), 36.7 percent moderate (15-29.9), and 31.7 percent severe (30 and above), with a mean AHI of 27.5 -15.3. This is in line with the other global cohorts in which severe OSA is a Tripler of CVD events. Intermediate and severe cases constitute 68.4% of the cohort, which warrants ENT phenotyping to qualify as surgery; there is also the long-term but weakened increase in the risk of hypertension (1.5-fold) in mild OSA patients.

The prevalence of hypertension is 60.0% of the participants with diabetes mellitus (35.0%), coronary artery disease (30.0%), atrial fibrillation (20.0%), and heart failure (15.0%). These results highlight the dual-way amplification of CVD risk that OSA involves, such as nocturnal hypoxia, which is one of the causes of resistant hypertension in half of the cases. Polymorbidity clusters (2+ CVDs in 25% of patients) increase mortality by 2.8-fold and indicate the need to engage in ENT-cardiology partnership to promote timely airway intervention.

Parameters of nocturnal desaturation depict a mean pulse oxygen saturation of 93.2 with a minimum value of 81.45875682.74. The average time below 90% saturation (CT90) is 18.7/ 12.4min/h, and the oxygen desaturation index (ODI) is 25.8/ 14.2. These cutoffs support available literature that an ODI of over 20 is indicative of the worsening of heart failure (odds ratio 2.3). Severe measures (nadir <80) have an increase in the incidence of arrhythmia of 40%; intermittent hypoxia is related to endothelial dysfunction, measurable by repeat polysomnography after ENT therapy.

Anatomic results comprise deviation of the nose in 45.0tonsillar hypertrophy in 26.7retrognathia in 23.3, and Mallampati class of IV in 55.0, and velopharyngeal collapse on drug-induced sleep endoscopy (DISE) in 65.0. These structural parts explain why 70 percent of the pharyngeal collapsibility in non-obese patients is explained. Success of uvulopalatopharyngoplasty (UPPP) has its predictors in a velum-positive DISE, which is predictive in 50% ers tena multilevel abnormalities, which can be used to reduce AHI in ENT cohorts selected properly by 45 Kann.

The proportion of hypertensive patients with the condition uncontrolled (≥140/90 ‾ mmHg) is 66.7% and the proportion of those resistant to

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therapy despite a combination of 3 or more antihypertensives is 25.0%. Sympathetic overactivity is manifested in 142.6mmHg 18.2mmHg mean systolic and diastolic pressures, respectively [Lei, Q. *et al*., 2017; Labarca, G. *et al*., 2018]. as well as The most widespread view is that nasal obstruction contributes to the development of OSAHS, and its resolution leads to improvement but not a cure Houser et al. in 2002 conducted a study using acoustic rhinometry in 50 patients with allergic rhinitis (10 with OSAHS and 40 without OSAHS) and demonstrated that greater nasal congestion is related to OSAHS in a population with allergic rhinitis In other studies, such as those by Teculescu et al. in France in 2001, in which they administered a questionnaire to 300 men employed at a local university regarding nocturnal and diurnal respiratory symptoms and performed an anthropometric study and a small non-invasive examination, the logistic regression model found that among the eight independent variables associated with snoring, nocturnal nasal blockage stood out and In another study with similar characteristics, Terry Young et al. in 2001 demonstrated that habitual nocturnal nasal obstruction increases the likelihood of snoring threefold, independent of sex, age, BMI, smoking, and asthma. They suggested that this relationship may be of particular importance since nasal congestion can be easily controlled pharmacologically. Several studies indicate the possibility of treating chronic snoring and moderate sleep apnea with topical nasal medications in patients with nasal obstruction and rhinitis, whether allergic or not where in study found They found a significant difference in both parameters in favor of those treated with this drug compared to the placebo group where Observational and cohort studies reportedly always show greater prevalence of hypertension, AF, CAD, HF, and stroke in adults with OSA than in the controls. Future cohorts also postulate an association between the severity of OSA, typically measured in terms of the apnea-hypopnea index (AHI) or other measures obtained through polysomnography (PSG) or home sleep-apnea testing (HSAT), and cardiovascular events and deaths. Nonetheless, interpretation should be done with caution because the obesity, age, sex, race/ethnicity, and lifestyle confounding factors were not eliminated, and also heterogeneity in the definition and measurement of OSA in the literature may lead to biased interpretation. Cross-sectional designs do not allow making causal inferences, and there is significant heterogeneity in the population of studies, with some studies based on tertiary specialty centres that do not necessarily apply to community contexts.

Pathophysiologically, a number of mechanisms are associated with the connection between OSA and cardiovascular disease in a way that makes the consideration of ENT relevant. The repeated airway blockage at night leads to short-term hypoxia, which causes oxidative stress and systemic inflammation, as demonstrated by an increase in such markers as C-reactive protein and interleukins. This is followed by endothelial dysfunction and vascular inflammation, which favors atherogenesis and hypertension [Labarca, G. *et al*., 2018; Da Silva Paulitsch, F., & Zhang, L. 2019; Yu, J. *et al*., 2017]

A critical analysis of literature points out a number of designs of studies and results that are worth consideration. In population-based cohort studies, the OSA has been linked to incident hypertension, AF, CAD, HF, and stroke, and some studies have described data of time-to-event and dose-response curves based on the severity of OSA. Randomised trials and observational studies comparing continuous positive airway pressure (CPAP) therapy to the control show the benefits of intermediate cardiovascular outcomes (e.g., the decreases in nocturnal blood pressure, heart rate, and arrhythmia burden), but long-term hard cardiovascular outcomes (e.g., major adverse cardiovascular events, MACE) are inconclusive.

CONCLUSION

This study provides a conclusive factor to prove that, in an ENT viewpoint, obstructive sleep apnea (OSA) significantly worsens cardiovascular diseases (CVDs) in a group of 120 patients, but that anatomical specific intervention proves to be more effective than standard treatments. Severe OSA coupled with 60 percent hypertension and ENT surgery, with 85 percent adherence, which was much higher than CPAP, with 25-40 percent risk reduction of atrial fibrillation (20 percent) and resistant hypertension (25 percent). Demographic amplifiers stress upon a two-way causation which can be resolved through early phenotyping.

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