

## Does The Etiology of The Heart Failure Influence The Presence of The Sleep Apnoea Syndrome?

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### Introduction

Many previous studies have shown the intercurrent association between the heart failure and the sleep apnea syndrome. (SAS) the SAS prevalence in the heart failure was estimated between 47% and 76 %. This disturbing rate is explained by several factors.

The aim of our study is to determine the possible association between the etiology of the heart failure and the SAS's presence.

### Materials and Methods

We conducted a retrospective study including 35 patients followed in our hospital, between January 2010 and December 2013 diagnosed with heart failure and SAS, which was confirmed by the polysomnography.

### Results

The average age of our population is  $63 \pm 5.3$  years old with extremes going from 32 years to 83 years. The population is essentially constituted of men (69 %) with a sex ratio of 2.2. The diabetes and hypertension were respectively found in 57 % (n=20) and 48 % (n=17) of the cases.

Other cardiovascular risk factors are in decreasing order: dyslipidemia found in 43 % (n=15), smoking in 35 % (n=12) of the patients.

The ischemic etiology of the HF( Heart failure ) was found in 52 % of the cases, hypertensive cardiomyopathy in 14 % of the cases, valvular in 9 % of the cases, 3 % of toxic origin

(alcoholic), cardiomyopathy of the pérupartum in 3%, and 19 % of indefinite origin.

The SAS is found in 73 % (n=25) of the cases of IC.

On the other hand, a prevalence of the central syndrome of apnea was found in 63 % (n=16) VS 37 % (n=8) for the obstructive sleep.

The aim of this study was to determine the possible association between the etiology of heart failure and the presence of the obstructive sleep apnea. Recent research has shown a correlation between the two. It is estimated that 30% to 40% of hypertensive patients have OSA (Obstructive sleep apnea) and 50% to 56% of OSA patients have hypertension.

### Discussion

SAS is caused by the collapse of the pharyngeal airway interrupting sleep. It's characterized by a repetitive interruption of ventilation during night's sleep. SAS occurs during respiratory effort, resulting in intermittent hypoxia and arousals, leading to a cascade of hemodynamic, inflammatory, autonomic and metabolic effects, which is responsible for its adverse cardiovascular effect [1-5].

The apnea-hyperpnoea index (AHI) is used to indicate the severity of SAS. This is the number of apnea's and hypopneas per hour of sleep: it (pauses in breathing) must last for at least 10 seconds and be associated with a decrease in blood oxygenation. AHI score is between 0- 30. A diagnosis of SAS is accepted when the index (AHI) is above 5 with symptoms such as daytime sleepiness, fatigue and impaired cognition. A diagnosis is also accepted, without other symptoms, when

the AHI is equal to or above 15.

This condition affects the general population, regardless of race or ethnicity. It affects 5-10 % of the general population [6], with a higher prevalence in males. In literature, sex ratio male-to female is about 3.3:1 (invest), our study has shown sex ratio of 2.2, which seems to be an important risk factor of sleep apnea.

Congestive heart failure (CHF) is a serious medical condition in which the heart cannot pump enough blood to meet the body's needs. Hypertension, coronary and valvular Heart diseases are often the causes of heart failure.

It is important to distinguish between OSA (obstructive sleep apnea) and central sleep apnea in patients with heart failure due to the difference of their pathophysiologies (central apneas are not caused by pharyngeal occlusion, but occur when the partial pressure of carbon dioxide falls below a threshold level needed to stimulate breathing) and treatments [13].

SAS was found to be associated with increased cardiac morbidity. [7] Obstructive sleep apnea was found to be an independent risk factor for cardiovascular disease with a high prevalence. In fact, up to 69% amongst patients present an acute coronary syndrome [1].

In 2014, a study looked at traditional risk factors and a physiologic biomarker of cardiovascular risk in comorbid insomnia and sleep apnea.

This study included 795 participants, which were chosen with symptoms of insomnia sleep apnea risk, obesity, smoking, sedentary lifestyles, hypertension, dyslipidemia, and diabetes. Its authors conclude that sleep apnea is a major contributor to cardiovascular risk [3].

A number of risk factors have been identified for the development of SAS in HF (heart failure), such as male sex, New York Heart Association functional class (III-IV), low ejection fraction, hypocapnia, higher prevalence of atrial fibrillation, high BNP levels, and frequent nocturnal ventricular arrhythmias [10-12].

In our study we looked at similar cardiovascular risk factors amongst 35 patients. These included diabetes and hypertension, which were respectively found at 57 % (n=20) and 48 % (n=17). Dyslipidemia and smoking were found in 43 % (n=15). Many studies have demonstrated the association between sleep apnea and heart failure, with the aim to prove that the treatment of the SAS improves the symptoms and decreases the mortality of the heart failure with reduced or preserved ejection fraction.

T Douglas Bradley [13], as many other authors in epidemiological studies have shown significant independent associations between hypertension, coronary artery disease, arrhythmias, heart failure, stroke and OSA, considering all etiologies of heart failure as an item of risk factor regardless to different causes of heart failure.

Shahar [15] in 2001, in a Cross-sectional study showed a 2.38-times increased likelihood of having heart failure in association with OSA independent of other risk factor, the presence of OSA was associated with impaired recovery of left-ventricular systolic function. However, in our knowledge there are no prospective data concerning SAS and the risk of developing either left-ventricular hypertrophy or failure. In patients with heart failure, the prevalence of OSA with an AHI exceeding 10-15 is high at 11-53%. Because the prevalence of central sleep apnea with an AHI of 15 or more in patients with heart failure is 15-37% but less than 1% in the general population, the overall prevalence of these sleep-related breathing disorders in patients with heart failure is about 50%. The finding that patients with heart failure have a lower body-mass index for a given AHI than the general population suggests that factors unrelated to obesity might contribute more to the pathogenesis of OSA in patients with heart failure. In addition to its chronic effects on systemic blood pressure, OSA could, through several mechanisms independent of blood pressure, promote left-ventricular hypertrophy, diastolic and systolic dysfunction, and overt heart failure. These mechanisms include the mechanical and trophic consequences of repetitive increases in left-ventricular wall stress.

Several studies reported increased left-ventricular thickness or mass in association with OSA, but these relations were not significant after adjustment for body weight [10,11].

Noda A, [14] in adults with non-ischaemic dilated cardiomyopathy left ventricular thickening was more prevalent in patients with OSA than in those without

Reduced ejection fraction patients with OSA have higher daytime sympathetic-nerve traffic and hypertension than those with heart failure alone, associated with a worse prognosis, hypertrophic cardiomyopathy,  $\beta$ -adrenoceptor desensitisation, and cardiac arrhythmias. In a prospective observational study involving 164 patients with heart failure who were followed up over 7.3 years, 10 mortality was significantly greater in the 37 patients with untreated OSA (AHI $\geq$ 15) than in 113 patients without sleep apnea [13].

In contrast with other studies, we have focused on the different causes of heart failure and their prevalence in association with the sleep apnea syndrome.

## Conclusion

In conclusion we found there was no influence of the etiology of heart failure on the presence of OSA in our patient's data. However, as previously mentioned, other studies have found links between the two, thus indicating further need for research in this area.

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