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The prevalence of allergic rhinitis and atopic markers in obstructive sleep apnea

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Abstract Allergic rhinitis (AR) related inflammation might worsen the severity of obstructive sleep apnea (OSA), however, the relationship between the two disorders remains controversial. Our aim was to determine the prevalence of AR and atopic markers in OSA. This cross-sectional study recruited participants with sleep-related complaints referred to a sleep center from February 2013 to June 2014. The diagnosis of OSA was based on the Berlin questionnaire (BQ) followed by confirmatory polysomnography (PSG). The diagnosis of AR was made via focused history and clinical examination and was confirmed by measuring atopic markers. OSA was diagnosed in 97 out of 157 adults attending the sleep clinic (61.8%). There was a high prevalence of AR (52.6%) among OSA individuals. This was not significantly different from the frequency in the non-OSA individuals ($p = 0.5$). Elevated total immunoglobulin E (IgE; >100 K/μL), eosinophil count, and positive Phadiatop tests were found in individuals with OSA to be 37.1%, 11.3%, and 41.2%, respectively. Individuals without OSA have shown similar percentages. In our cohort, there was no

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significant difference in frequency of AR and atopy among participants with OSA compared to those without OSA.

1. Introduction

Obstructive sleep apnea (OSA) refers to repeated episodes of halted respiration during sleep, despite a continuous effort to breathe [1]. Clinically, OSA is characterized by excessive daytime sleepiness, disruptive snoring, and nocturnal hypoxemia. This condition is a common sleep-breathing disorder affecting approximately 4.0% of males and 2.0% of females of middle age in the developed world [1]. Many studies have shown that OSA is a significant source of morbidity and mortality; it is associated with serious health consequences, mostly afflicting the cardiovascular and cerebrovascular systems [1]. In addition to excessive daytime sleepiness, people with OSA have subsequent decreased functional ability, which could lead to motor vehicle accidents [1].

The mechanism of OSA is not well understood, but is mostly attributed to mechanical upper airway disruption (airway narrowing or collapse), leading to increased airflow resistance. Accordingly, several reports have hypothesized that allergic rhinitis (AR) may influence the occurrence of OSA through inflammatory elements that induce nasal congestion and subsequently nasal obstruction [2]. In addition, the high nasal resistance secondary to nasal obstruction leads to a more negative intraluminal pressure in the lower airways that may promote partial or intermittent collapse of the pharynx, thus increasing the risk of OSA. This hypothesis is strengthened by the beneficial effect of intranasal corticosteroid in OSA and rhinitis [6].

Several studies have proposed a link between AR and OSA. Recently, a Turkish study reported that 23.0% of OSA patients had AR [3], whereas several European studies have revealed variable rates [4]. We also recently reported that approximately half of OSA patients in a pilot study had AR, regardless of the severity of sleep apnea [5]. Furthermore, several clinical trials investigating the effect of treating AR in OSA patients found very promising outcomes. In particular, by reducing levels of inflammatory mediators, AR treatment may improve the severity of OSA, the quality of sleep, and daytime somnolence [6]. These findings illustrate a possible link between OSA and AR, and also suggest a role for allergic inflammatory mediators (e.g., eosinophils) in the development and progression of sleep-disordered breathing. Additionally, other atopic diseases, such as bronchial asthma (BA) and atopic dermatitis, have been implicated in sleep-disordered breathing, mostly among children [7]. This significant finding sheds light on the potential role of allergic sensitization (atopy) in the pathophysiology of OSA.

Individuals with atopy are usually identified based on the presence of allergic diseases such as asthma, AR, allergic conjunctivitis, food allergy, and eczema [8]. Allergic sensitization to allergens is classically confirmed by an *in vivo* skin-prick test (SPT) or an *in vitro* assay for specific serum immunoglobulin E (IgE) antibodies [8]. Total IgE and peripheral eosinophils have also been established in the literature as nonspecific markers of atopy; however, their diagnostic value is limited [9].

In addition to a lack of local data, the relationship between AR and OSA remains controversial in the literature. Furthermore, the association between atopy and OSA is not well established in the adult population. Therefore, the present study aimed to determine the prevalence of atopy in general and of AR in particular among adult OSA patients referred to a university-based sleep center. This is a continuation of a previously small pilot study addressing the relation between AR and OSA (5), as it is felt that the subject has not been fully evaluated. Therefore a relatively larger study is needed to address the possible association and hence this study.

2. Materials and methods

In this cross-sectional study, all participants with sleep disorders who were referred to the sleep clinic at King Abdulaziz University Hospital in Jeddah, Saudi Arabia from February 2013 to June 2014 were recruited. Hospital ethics committee approval was obtained, and written informed consent was requested from all participants. The study end points were estimates of the prevalence of atopy and AR among OSA patients.

Individuals who refused to participate or to complete the requirements of the study were excluded. The study was conducted in two stages. In Stage 1, each participant was interviewed and