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DOI: 10.2478/rjr-2019-0002

Romanian Journal of Rhinology, Volume 9, No. 33, January-March 2019

LITERATURE REVIEW

The united airway disease

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ABSTRACT

OBJECTIVES. The aim of this paper is to review the united airway concept.

MATERIAL AND METHODS. We searched Pubmed, Google, Google Scholar and Proquest Central database of Kırıkkale University.

RESULTS. Upper and lower airways are thought as a morphological and functional unit. There is a link between rhinitis and asthma. Over 80% of asthmatics have rhinitis and 10-40% of rhinitis patients have asthma. Rhinosinusitis is related to asthma in 34-50% of the patients. The relationship between rhinosinusitis and asthma may include "naso-bronchial reflex, pharyngobronchial reflex, inhalation of dry, cold air and environmental pollutants inhalation".

CONCLUSION. The united airway concept suggests that upper and lower airways are thought as a morphological and functional unit. It has been commonly accepted in recent years. Allergic rhinitis (AR) is a risk factor for asthma; and Allergic Rhinitis and Asthma (ARIA) suggest bronchial involvement in AR patients.

KEYWORDS: united airway, upper airway, lower airway, asthma, allergic rhinitis.

INTRODUCTION

Upper and lower airways are thought as a morphological and functional unit and the relationship between them has been observed for a long time[1,2]. The nose is located in the passage of the airway and the underlying airway approaches as a strong ventilation to protect it from the destructive effects of the air. The nose warms, filters, humidifies and saturates the air, so at 37°C the natural air is sent to the lungs with full steam. During nasal breathing the collected particles with an aerodynamic equivalent diameter (AED) higher than 15 μm accumulate in the upper respiratory tract. Particles with AEDs > 2.5 μm are basically collected in the trachea and bronchus, while those with low AED enter the gas trade area of the lungs[3]. The nose and the bronchial mucosa simulations are shown, and the strikingness among the most important aspects of nasal-lung communication is a practical complement to the lungs that provides nasal safety[4]. However, the upper respiratory tract elements and their communication with the lower respiratory tract are more extensive than air conditioning[5].

Asthma and rhinitis patients tend to have a more serious disease with higher treatment costs, and therefore the "airway disease" has a considerable effect, including the treatment and prophylaxis of these air distributions[6,7]. Allergic rhinitis (AR) is a risk factor for asthma; Allergic Rhinitis and Asthma (ARIA) suggest bronchial involvement in AR patients[8,9].

In this paper, we review the united airway concept. We searched Pubmed, Google, Google Scholar and Proquest Central database of Kırıkkale University.

EPIDEMIOLOGY

AR is the most widely accepted phenomenon of all atopic diseases and although most patients may be of any age, most patients report the onset of symptoms before the age of 30, which is the most well-known conception in children[4]. AR can be seen as a significant general medical issue due to the patient's personal satisfaction, work / school performance and profitability being dominated by monetary weight and influence[4,5].

AR is thought to be a risk factor for asthma[4]. Asthma is portrayed by the historical backdrop of episodic respiratory symptoms, for example, "wheezing, shortness of breath and chest torment", and is related

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Received for publication: August 31, 2018 / **Accepted:** September 7, 2018

with variable expiratory flow limitations[5,10]. Non-AR patients (NAR) are at expanded risk of creating non-allergic asthma. NAR appears further in life than AR and it is not a solitary issue; however, it is made out of a heterogeneous gathering of diseases[5,11].

The link between rhinitis and asthma has been the subject of some epidemiological studies that reliably demonstrate an important implication of the two diseases[12]. Despite asymptomatic asthma in approximately 19-38% of patients with AR and the fact that 30-80% of asthmatics had AR, late studies found that 98.9% of rhinitis was present in allergic asthmatics and 78.4% in non-allergic asthmatics[13]. The vast majority of AR patients have bronchial hyperreactivity (BHR)[10].

Over 80% of asthmatics have rhinitis and 10-40% of rhinitis patients have asthma to suggest "one airway, one disease" [4]. Rhinitis findings are found in 98.9% of allergic asthmatics and 78.4% of non-allergic asthmatics. In addition, approximately 30% of patients with AR alone, without asthma, have either methacholine or histamine hypersensitivity[2,4,5,14].

Although allergic rhinitis is seen as a risk factor for the formation of asthma, it can be assumed that this term cannot be changed exactly; this can be explained by an initial time at which the upper airway disease (UAD) can go as far as asthma[8].

PATHOPHYSIOLOGICAL EVIDENCE

The upper and the lower airways provide a continuum that allows air to enter and exit the lungs and share many anatomic and histological features[2]. The ciliary epithelium, basement membrane, lamina propria, glands and goblet cells contain regular structures that shape the so-called united airway[15]. Then again, there are contrasts between the upper and the lower airways. The nasal mucosa, enriched with vessels, attached to the bone; the bronchial mucosa, enriched with smooth muscle cells, attached to the cartilage[5,16].

The upper respiratory tract is the main focus of allergens, physical and compound natural stimuli; thus, they have a tendency to be the first influenced by the allergic airway disease, and if the disease intensity is low, the upper respiratory tract might be the fundamental part of the influenced respiratory tract. Be that as it may, when the whole respiratory tract is included, rhinosinusitis and asthma begin after a parallel highway[5,17].

The allergic respiratory disease is caused by hypersensitivity or IgE-mediated responses when the inhaled allergen reacts with "mast cells and basophiles, the major effector cells, bearing IgE antibodies". Concomitant cross-linking of allergen-specific IgE molecules to cells by allergen particles allows granule-associated mediators (e.g., "histamine, tryptase"), "membrane lipid-derived mediators (i.e., leukotrienes)", and cytokines to come[18,19].

AR and atopic asthma arise from an allergic response to an IgE response associated with variable strength airway aggravation[4]. Since the Ig displayed on the surface of B cells is IgM, it is of vital importance to convert IgM to IgE with the goal of creating allergic aggravation. IgE converting isotypes require antigen entry and two different signals[20]. The first markers, IL-4R and IL-13R, are mediated by IL-13 as well as interleukin (IL) -4, which mediates STAT6 translating IgE isotypic interpretation. The flags are given by CD40 binding in CD40L B-cells in T-cells that mainly initiate DNA-key recombination[20]. The IgE interfering safe reaction is initiated when allergens are taken up by cells presenting antigen through the cell surface Ig receptor. Subsequently, the T helper (TH) cells, which recognize and trigger the allergen – major histocompatibility complex II compound, are introduced into the major histocompatibility complex class II. The allergen-specific TH2 cells form IL-4 and IL-13 and exchange CD154 and require IgE class change[5,20,21].

When IgE is produced by the B cells, Ig binds to the high affinity receptor FcεR1 on mast cells and basophiles[20]. In the future, contacts with polyvalent sensitizing allergens will result in FcεR1, initiating a quick-tip varnish response that is the focus of ARC and the pathogenesis of allergic asthma[20]. The reaction has gone to a quick pace, triggered by the development of pre-built up and quickly incorporated mediators from mast cells and basophiles, activating occasions, for example, erythema, edema and skin itching in the upper respiratory tract, nasal obstruction in the upper respiratory tract, and cough, edema and mucus secretion in the lower respiratory tract[20]. A late stage, which is intervened by cytokines and chemokines and described by edema and leukocytic union, can happen 6 to 24 hours after the coveted stage. Eosinophils, which are mainly produced by the IL-5 delivered by the TH2 cells, emerge and depend on the chronic inflammatory process and tissue harm[5,20].

Several components have been recommended to clarify the interaction between upper and lower airways[5,22]. Most likely, inflammatory changes in the upper and lower airways lead to a basic reaction, involving bone marrow, which then leads to the arrival of the progenitor cells, which are recorded at tissue locations. The provocative discharges can be spread from the upper airway to the underlying area by postnasal diving and basic dissemination[20]. An important confirmation of UAD interaction is the proximity of thickening of the epithelial basement membrane, which is a typical hallmark of the lower

respiratory tract remodelling in asthmatic atopic patients as well as in asthmatic patients. For each situation, the reestablishment has all the earmarks of being less extensive in the nasal mucosa than in the bronchial mucosa. The reason for this qualification might be cleared up by the production of particular cytokines of smooth muscle cells and their vicinity to embryological separation qualities in the lungs or by their diverse re-expression in asthma and rhinitis[19,23].

RISK FACTORS

Allergen exposure

Allergenic input is seen as a real risk factor for the recovery of UAD[19,24]. Rhinitis and asthma are characterized by high sensitization to the usual allergens in the group, i.e. aeroallergens. When considering various air / breathing allergens, the most important qualities are the sufficiency between open air allergens (i.e., dust and forms), closed area allergens (e.g., cat, dog, puppies, parasites) and words related operators[19,25].

Hereditary Factors

The rate of allergic diseases in families is extraordinary: 35% to 95% for asthma, and 33% to 91% for AR[26]. Focuses on the prevalence of allergic manifestations in relationship with family history indicate expanding increments with the risk of creating asthma or AR, with maybe a couple allergic diseases, and more than three allergic diseases have turned out to be more conspicuous if the allergic disease happens[26]. A positive family history is one of the strictest devices for anticipating the allergic disease[19,27].

The relationship of asthma and AR with different chromosomal domains was found recently. Surely, there are roughly 161 diverse potential biomarkers engaged with respiratory irritation[27]. For instance, Liu et al. have demonstrated that single nucleotide polymorphisms in TNFSF4 and FAM167A-BLK genes might be related with asthma and AR[28] and Zhao et al. reported the PBX2 gene in the 6p21.3 asthma susceptibility locus with an expanded risk for both AR and asthma[19,29].

REACTION TRIGGERS

There might be a few factors that trigger reflex airway responses. Infections and microbes are in charge of infectious rhinitis, rhinosinusitis, bronchiolitis and pneumonia. Chronic Obstructive Pulmonary Disease (COPD) and asthma exacerbations can happen related with them[30-32]. Tobacco smoke and climatic or professional pollutants can likewise cause chronic aggravation of upper and lower respiratory tracts, for example, chronic bronchitis, and can cause metaplasia and carcinogenic lesions. Tobacco smoke is the most vital hazard factor for COPD[33], laryngeal cancer, bronchial carcinoma, and rhinitis[34]. The presentation of cadmium smoke to word related exposure may bring about the result of emphysema[35], while the exposure to asbestos may cause cancer[36].

Allergens are solid stimulants of allergic rhinitis and asthma. Indoor allergens, such as dust mites or animal proteins, are highly associated with asthma[37]. Outdoor air allergens, e.g. pollens, are generally associated with rhinitis[38]. Pollen particles are usually too large to enter the lower airways, but pollen grains are found in bronchial secretions and in the lung parenchyma[38]. Asthma and rhinitis hypersensitivity reactions can be activated by beta blockers or aspirin, exposure to cold weather and physical exercise[36,39,40].

Neural reflexes

The presence of a naso-bronchial reflex starting from the sensory nerve endings, through the trigeminal nerve, goes to the central nervous system and follows an efferent pathway in the vagus nerve to deliver airway smooth muscle contraction[5,17].

Several studies in healthy subjects and in asthmatics have shown that airway resistance increases after cold and dry air is inhaled[39,41]. Another important study showed an expansion of the AHR after nasal allergen induction in asthmatics, showing that asthma symptoms deteriorated after seasonal rhinitis exacerbation[42]. Remember that the traditional naso-bronchial reflex is part of the dive reflex[43]. The cold water submersion of the head causes rapid respiration suppression (apnea), laryngospasm and bronchoconstriction to suppress the lower respiratory tract from the dive[43]. Contingent upon the relaxation of the inspiratory muscles, the respiration of dust, contaminants, and aggravations through the nose may bring about prompt bronchoconstriction, while the expiratory airway is breathing at the stage[43]. Consequently, in people with the allergic respiratory disease, this reflex may cause an expansion in asthma manifestations after nasal damage[5].

THE LINK BETWEEN ALLERGIC RHINITIS AND ASTHMA

Allergic rhinitis is the most recognized agent of all atopic diseases, although it normally occurs before school age in patients who exhibited atopic dermatitis during the first years, and may occur at any age. Allergic rhinitis may be irregular or unstable with conditional or gentle or direct / extreme conditional side effects, leading to the severity of side effects experienced by the patient[44]. Apart from traditional indications such as sniffing, nasal congestion, AR is

shown to be weakness and possibly restlessness in the personal satisfaction of patients, enthusiastic subjects, disability in exercises and social services[44-46].

Asthma is defined as recurrent airway obstruction disease and it is analyzed using lung studies and bronchial hyperreactivity measurements. Primer findings include dry cough, expiratory wheezing, chest relaxation and dyspnea, intermittently activated with the exacerbation of allergies and diseases of the airways[47]. Small airway pathology characterized by reduced forced expiratory flow (FEF) at 25-75% of the lung volume and abnormal spirometry has been suggested to be a sign of early allergic or inflammatory involvement of small airways with allergic diseases without asthma[45,48].

Allergic rhinitis is considered a risk factor for asthma, but in these cases it is thought that these terms cannot be fully resolved[48], as the united airway diseases that may lead to asthma may speak at the onset of the disease. Approximately 19% to 38% of allergic rhinitis patients have active asthma and 30-80% of asthmatics have allergic rhinitis. Nevertheless, this information is probably considered to be surprisingly low since rhinitis is present in 98.9% of the asthmatics and in 78.4% of non-allergic asthmatics[13,45].

Allergic rhinitis (AR) is now associated with asthma and regularly prevents bronchial hyperreactivity[48]. Most patients with AR (up to 80% of cases) show bronchial hyperreactivity (BHR), no loss of lung function and no clinical signs of asthma[10,49], and this finding may be a prognostic factor for the progression of asthma[36,50].

THE LINK BETWEEN RHINOSINUSITIS AND ASTHMA

Rhinosinusitis and asthma are two unique expressions of a common pathological process that is typically not influenced by allergies, in which eosinophils and airway epithelium assume a part[51]. Late movement, in the consciousness airway pathology, has distinguished inflammation as a method for understanding these infections. Taken together, different diverse parts that link airway fragments from "the upper (nose, sinuses, larynx, pharynx, and trachea) and fundamental (bronchus and lungs)" can likewise be incorporated[36,52].

In children, the paranasal sinus diseases are thought to be a critical hazard factor for the treatment of lower respiratory tract illnesses, and rhinosinusitis and asthma seem to have framed two unique expressions in a common pathological process[53]. The late advances in understanding the investigation of science of the airway diseases explained the role of inflammation in these two pathologies. There can be a few unique systems that connect the "upper (nose, sinus, larynx, pharynx, and trachea) and lower (bronchus and lungs)" airway sections[52].

Precipitation of asthma, as a rule, stimulates sinusitis, and then the connection between exacerbation of sinusitis and asthma can be an epiphenomenon[53]. Rhinosinusitis is associated with asthma in 34% to half of patients[54]. In patients with asthma, the occurrence of associated rhinosinusitis increases to 84%, especially with exacerbations of asthma[45,55].

It is vague whether rhinosinusitis is a quick trigger for asthma or whether the two conditions are demonstrative of a run of a typical underlying process. The conceivable clarifications of the relationship of rhinosinusitis and asthma may include the "naso-bronchial reflex, pharyngobronchial reflex, inhalation of dry, cold air and environmental pollutants inhalation"[54]. To be sure, even bone marrow can give this connection between the upper and lower airways while making a run of the typical disease: blood eosinophils count is frequently connected with asthma development and asthma seriousness, though IL-5 might be a critical cytokine to facilitate the systemic interaction[36,54,55].

Now it is confirmed that rhinosinusitis without polyposis or eosinophilic inflammation is a quick stimulant for asthma, but rhinosinusitis with polyps and eosinophilic exacerbation suggests the main mechanisms of asthma[47]. In essence, it is clear that the markers of bronchial irritation, commonly found in asthmatics, correspond to the severity of sinusitis[36,56].

CONCLUSIONS

The united airway concept suggests that upper and lower airways are thought as a morphological and functional unit. It has been commonly accepted in recent years. Allergic rhinitis (AR) is a risk factor for asthma; Allergic Rhinitis and Asthma (ARIA) suggest bronchial involvement in AR patients.

Ethics approval: There was no need to take Ethical Committee Approval.

Financial discloser: There is no financial disclosure of the author.

Conflict of interest: The author reports no conflicts of interest.

Contribution of authors:

Nuray Bayar Muluk: Planning, designing, literature survey, writing.

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