

Editorial

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Allergic Rhinitis and Asthma: The United Airways Disease

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Interactions between the upper and lower airways have been well investigated in the past 30 years. The nasal and bronchial mucosa share similarities, in addition to their functional interaction. At least 80% of asthmatics have rhinitis and up to 40% of patients with rhinitis have asthma proposing the concept of 'one airway one disease' although there is still some differences between rhinitis and asthma.¹

Recently, many clinical and as well as experimental studies suggested uniting the upper and lower airway diseases in a single term (allergic rhinobronchitis or united airways disease (UAD) as was proposed by Passalacqua et al.² This new link was founded by clinical epidemiological and immunological studies in addition to therapeutic outcomes. The recent understanding of the underlying pathogenetic mechanisms, including the cells, mediators and cytokines involved in the allergic inflammation in the respiratory tract has added more proof to the functional links between the nose and bronchi.³

According to the latest guidelines on the treatment and control of allergic rhinitis: The Allergic rhinitis and its impact on asthma (ARIA) workshop report; bronchial asthma and allergic rhinitis are distinct manifestations of a single airway and of the same disease.⁴ Among the underlying evidence of the link between rhinitis and asthma is the common co-existence of rhinitis and asthma as patients can present with symptoms of allergic rhinitis then later develop asthma or the opposite can be the presenting scenario. Since, upper respiratory tract infections are among the very important causes of asthma exacerbation and also rhinitis has been found to be an important risk factor for developing asthma through postnasal drip into the lower airways or through mediators that directly alter airway reactivity or cause lower airway inflammation.⁵

Moreover, bronchial hyperreactivity (BHR) which is a characteristic of bronchial asthma, is also present in patients with allergic rhinitis who have no clinical evidence of asthma.⁶ Many patients with rhinitis do not report the classical bronchial asthma symptoms and have no proof of airway obstruction on spirometry, but they present mainly with bronchial hyperreactivity which could be due to the presence of subclinical inflammation of the lower respiratory airway. Many studies were done on patients with seasonal allergic rhinitis, where bronchoalveolar lavage (BAL), bronchial biopsy and sputum samples have provided evidence of lower airway inflammation including eosinophils. This supports the presence of subclinical inflammation within the lower airways of patients with allergic rhinitis, which might be the underlying cause of the BHR in these patients.⁷

Therefore, there is a category of patients with persistent allergic rhinitis who present with symptoms of cough and/or chest tightness and show no evidence of airway obstruction on spirometry, their symptoms could be due to bronchial hyperreactivity. In addition to adequate rhinitis treatment, these patients usually benefit from asthma medications such as inhaled corticosteroids as well as leukotriene modifiers which can be of value in controlling upper as well as lower airway allergy. From practical experience it's believed that the early recognition and treatment of those patients might prevent their subsequent development of full blown asthma.

Moreover, in patients with asthma, rhinitis should be appropriately evaluated and

treated to ensure good control of their asthma symptoms. Hence, a combined therapeutic approach should ideally be used to manage the upper and lower airway diseases, benefiting from the concept of united airways disease (UAD).

In conclusion, allergy is a systemic disorder and should not be considered as an organ disease, thus patients with respiratory allergy can present with rhinitis, bronchial hyperreactivity and/or asthma.

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