

## How do we treat chronic rhinosinusitis in asthmatic patients?: In aspects of medical treatment

Department of Pulmonology, Allergy and Critical Care Medicine, CHA Bundang Medical Center, CHA University

Mi-Ae Kim

The nose and lung are both part of the respiratory tract. Numerous studies have highlighted the fact that the respiratory system is a single entity and the concept of “united airway disease” has become important. When treating rhinitis, it is often necessary to assess the presence of asthma. Patients with sinusitis should be evaluated for a possible concomitant asthma. Conversely, patients with asthma should always be evaluated for possible nasal disease. The medications that treat nasal diseases appear to be useful in improving control of asthma and in reducing bronchial hyperresponsiveness as well.<sup>1)</sup>

The natural progression of respiratory allergy commonly starts from the upper respiratory tract and later spreads to the lower tract.<sup>2)</sup> Allergic inflammation involves the whole respiratory tract, affecting the nose, the paranasal sinuses and the bronchi. Nasal allergic inflammation results in a systemic inflammation by soluble mediators that involve the bone marrow and, as a result, production of eosinophils.<sup>3-5)</sup> In addition, bronchial challenge may induce nasal inflammation as well as pulmonary allergic inflammation after nasal allergen challenge.<sup>6-8)</sup>

Allergic rhinitis (AR) is frequently associated with asthma and it often precedes bronchial hyper-reactivity. Approximately 19-38% of patients with AR have concomitant asthma and 30-80% of asthmatics have AR.<sup>9,10)</sup> A large proportion of patients with AR (up to 80% of cases) show bronchial hyperreactivity (BHR), even though they do not present any clinical sign of lung function impairment nor of asthma.<sup>11,12)</sup> Small airway disease (SAD), defined as a reduction in forced expiratory flow (FEF) at 25%-75% of the pulmonary volume and a normal spirometry, is suggested to be a marker of early inflammatory involvement of the small airways in subjects with allergic diseases and no asthma.<sup>9)</sup> FEF<sub>25-75</sub> seems to be significantly associated with BHR, and it has been proposed as an early marker of bronchial involvement in patients with AR who perceive only nasal symptoms.<sup>13)</sup>

Rhinosinusitis is a common disease that may influence the clinical course of asthma by a variety of mechanisms.<sup>14)</sup> Symptoms include nasal congestion, nasal discharge, nasal purulence, postnasal drip, facial

pressure, hyposmia, cough, fever, halitosis, dental pain, ear fullness, and headache. The diagnosis and management of sinusitis are often challenging, but generally unsatisfactory.<sup>15)</sup> It is not clear whether rhinosinusitis is a direct trigger for asthma or if the two conditions are just manifestations of a common underlying process. Possible explanations for the observed association of rhinosinusitis and asthma may include the nasobronchial reflex, pharyngobronchial reflex, postnasal drainage of inflammatory mediators from the upper to lower airway, inhalation of dry, cold air and environmental pollutants, and the “shared pathogenesis” of rhinosinusitis and asthma.<sup>16)</sup> Current evidence suggests that rhinosinusitis without either polyps or eosinophilic inflammation is a direct trigger for asthma, whereas rhinosinusitis with both polyps and eosinophilic inflammation shares underlying mechanisms with asthma.<sup>17)</sup>

Intranasal steroids are widely used to control AR and chronic rhinosinusitis, especially when combined with inhaled steroids. There are some evidences that nasal inhalation of a corticosteroid results in both upper and lower airways deposition and effective asthma control.<sup>18,19)</sup> Antibiotics treatment can be helpful in chronic rhinosinusitis in addition to nasal saline irrigation or decongestant. Antihistamines, leukotriene receptor antagonists, and allergen-specific immunotherapy are used in controlling symptoms of AR.

## References

1. Ciprandi G, Caimmi D, Miraglia Del Giudice M, La Rosa M, Salpietro C, Marseglia GL. Recent developments in United airways disease. *Allergy Asthma Immunol Res* 2012;4:171-7.
2. Pedersen PA, Weeke ER. Asthma and allergic rhinitis in the same patients. *Allergy* 1983;38:25-9.
3. Denburg J. The nose, the lung and the bone marrow in allergic inflammation. *Allergy* 1999;54:73-80.
4. Sehmi R, Wood LJ, Watson R, Foley R, Hamid Q, O'byrne PM, et al. Allergen-induced increases in IL-5 receptor alpha-subunit expression on bone marrow-derived CD34+ cells from asthmatic subjects. A novel marker of progenitor cell commitment towards eosinophilic differentiation. *J Clin Invest* 1997;100:2466-75.
5. Gaspar Elsas MIC, Joseph D, Xavier Elsas P, Boris Vargaftig B. Rapid increase in bone-marrow eosinophil production and responses to eosinopoietic interleukins triggered by intranasal allergen challenge. *Am J Respir Cell Mol Biol* 1997;17:404-13.
6. Blaiss MS. Rhinitis-asthma connection: epidemiologic and pathophysiologic basis. *Allergy Asthma Proc* 2005;26:35-40.
7. Braunstahl G-J, Kleinjan A, Overbeek SE, Prins J-B, Hoogsteden HC, Fokkens WJ. Segmental bronchial provocation induces nasal inflammation in allergic rhinitis patients. *Am J Respir Crit Care Med* 2000;161:2051-7.
8. Braunstahl G-J, Overbeek SE, KleinJan A, Prins J-B, Hoogsteden HC, Fokkens WJ. Nasal allergen provocation induces adhesion molecule expression and tissue eosinophilia in upper and lower airways. *J Allergy Clin Immunol* 2001;107:469-76.
9. Compalati E, Ridolo E, Passalacqua G, Braido F, Villa E, Canonica GW. The link between allergic rhinitis and asthma: the united airways disease. *Expert Rev Clin Immunol* 2010;6:413-23.
10. Simons FER. Allergic rhinobronchitis: the asthma-allergic rhinitis link. *J Allergy Clin Immunol* 1999;104:534-40.
11. Braman SS, Barrows AA, DeCotiis BA, Settignano GA, Corrao WM. Airway hyperresponsiveness in allergic rhinitis: a risk factor for asthma. *Chest* 1987;91:671-4.
12. Riccio MM, Proud D. Evidence that enhanced nasal reactivity to bradykinin in patients with symptomatic

- allergy is mediated by neural reflexes. *J Allergy Clin Immunol* 1996;97:1252-63.
13. Ciprandi G, Cirillo I, Klersy C, Marseglia GL, Vizzaccaro A, Pallesstrini E, et al. Role of FEF<sub>25-75</sub> as an early marker of bronchial impairment in patients with seasonal allergic rhinitis. *Am J Rhinol* 2006;20:641-7.
  14. Meltzer EO, Szwarcberg J, Pill MW. Allergic rhinitis, asthma, and rhinosinusitis: diseases of the integrated airway. *J Manag Care Pharm* 2004;10:310-7.
  15. Jani AL, Hamilos DL. Current thinking on the relationship between rhinosinusitis and asthma. *J Asthma* 2005;42:1-7.
  16. Barbi E, Longo G. Chronic and recurrent cough, sinusitis and asthma. Much ado about nothing. *Pediatr Allergy Immunol* 2007;18:22-4.
  17. Tosca M, Riccio A, Marseglia G, Caligo G, Pallesstrini E, Ameli F, et al. Nasal endoscopy in asthmatic children: assessment of rhinosinusitis and adenoiditis incidence, correlations with cytology and microbiology. *Clin Exp Allergy* 2001;31:609-15.
  18. Stelmach R, Maria do Patrocínio TN, Ribeiro M, Cukier A. Effect of treating allergic rhinitis with corticosteroids in patients with mild-to-moderate persistent asthma. *Chest* 2005;128:3140-7.
  19. Mygind N, Bisgaard H, Dahl R. Simultaneous treatment of rhinitis and asthma by nasal inhalation of corticosteroid from a spacer. *Allergy* 1999;54:132-5.