“The Expert Panel recommends that clinicians evaluate patients who have asthma regarding the presence of rhinitis/sinusitis diagnosis or symptoms….. it is important for clinicians to appreciate the connection between upper and lower airway conditions and the part the connection plays in asthma management”.1

The concept of a united airways disease has been on the cards for the past few years and recent understanding and literature has only strengthened its association. This understanding started with the association of the increasing incidence of allergic rhinitis and asthma.2 This has clear clinical, epidemiological, functional and immunological components that have been confirmed at every level and appeared on documents of scientific and regulatory bodies.

Allergic rhinitis has been identified as an independent risk factor for asthma and hence there has evolved multiple propositions that aim at the prevention of asthma by early recognition and control of allergic rhinitis. The role of upper airway infections in exacerbating existing asthma has also been well documented.3 The production of common mediators of inflammation and their subsequent actions at both levels of the respiratory tract is a pointer to the fact that this is a disorder not of a single organ but the whole respiratory tract itself. These observations have under mined the concept and terminology of UAD – United Airways Disease.4
The association of the above two diseases have an economic impact in the sense that they have to be treated with more medication and probably for a longer duration of time there by impacting on the economics of the disease profile. The formation of ARIA—allergic rhinitis and its impact on asthma by the WHO clearly focuses on the role of rhinitis as a risk factor for asthma.2

Epidemiological data suggest that rhinitis occurs in 98.9 percent of allergic asthmatics and in around 78.4% of non—allergic asthmatics. Generally rhinitis will precede the asthma and patients with rhinitis have at least a three fold greater tendency to develop asthma than controls.3 There is also interesting data on childhood rhinitis. The6 Tasmanian asthma study demonstrated a two to seven fold increased asthma risk in children who develop childhood rhinitis. It appears that the above observations have firmly established the clear associations between one airway and one disease.

The LARA study—Swiss link allergic rhinitis in asthma survey reported that seventy six percent of patients with asthma had rhinitis and only twenty four did not. Patients with rhinitis were younger in age and hence had an increased tendency to develop asthma in future.7

THE NASO BRONCHIEL CROSS TALK

The respiratory tract can be considered as a single morphologic entity with inflammation as the core pathologic process that affects the whole system. Lymphoid tissue in the form of MALT8 – mucosal associated lymphoid tissue plays a key role in the inflammatory cascade that characterizes rhinitis and asthma is generously distributed in both the nose and bronchial tree. The major difference between the nose and bronchi is the presence of a high degree of vascularity in the nose and the characteristic bronchial smooth muscle in the lung. This probably explains the diversity of symptomatology produced by similar inflammatory cytokines. One should also be conscious of the fact that the nose plays an important role as a physical barrier, heater and humidifier of inhaled air and this is a key function that could have implications in the lower respiratory fact.9

The Sinobronchial reflex is a possible pathogenetic mechanism that could induce nasal irritation and cause broncho-constriction and initiate systemic release of inflammatory mediators and promote bronchial hyper reactivity. The phenomenon of MINIMAL PERSISTANT INFLAMMATION involves a week inflammatory response which is chronic in nature and has low level symptoms that may be missed on clinical examination.

Other possible mechanism involves neurogenic inflammation with neuropeptides such as substance P and neurochimine A. The role of the small airways – SAD Small Airways Disease. (reduced FAF at 25-75 ) with normal FEC and FEV1 / FVC ratio has been proposed to be a marker of patients with allergic disease but no clear symptoms of asthma. These patients obviously have a greater tendency to develop asthma in future.10

There is a clear association between the presence of rhinosinusitis in asthma not withstanding the presence or absence of polyps and eosinophilic inflammation. Thus there is an overwhelming flood of information regarding the link between rhinitis and asthma and unifying the airway as a UNITED AIRWAYS DISEASE.
It is obvious that inflammation being the key pathologic entity anti – inflammatory therapy is the backbone of treatment for rhinitis. Anti histamines and decongestants have a clearly defined but narrow therapeutic scope in therapy and have been found to be inferior to inhaled steroids. A metaanalysis of nine randomized trial clearly flavoured intra nasal steroids over anti histamines. Antileukotrienes like montelukast and zileuton do reduce nasal symptoms and may reduce the need for rescue medication in asthma. Unfortunately they are not effective in all patients and there are a clear set of non responders. Hence it appears unlikely that they will replace the inhaled steroids. Omalizumab a monoclonal anti IGE anti body has demonstrated a dose depended clinical benefit in patients with rhinitis and asthma and is approved at step 5 of GINA but may be a therapeutic option in patients with severe rhinitis and asthma in the future. All intra nasal steroids – beclomethasone, flunisolide, budesonide, fluticasone, and mometasone, are highly effective with a very minimal side effect profile. Immunotherapy has a narrow and limited role although there has been a recent increased interest in sublingual immunotherapy.

The only drug that appears to have a specific action on every single nasal symptom is the intranasal steroid.

Asthma has been well established as a chronic eosinophilic inflammatory disease with striking similarities to rhinitis at all levels – anatomic physiologic and epidemiological. The therapy for asthma has been well documented and established by the GINA GUIDELINES. It is obvious that the ARIA and GINA GUIDELINES form the foundation of the treatment of united airways disease. A casual look at both of them will underscore the basic inflammatory nature of the disease and also of the therapeutic superiority of the inhaled steroid. There is enough evidence to demonstrate this superiority.

Combination inhalation therapy has revolutionized therapy
for asthma over the past decade and asthma control as defined by GINA GUIDELINES has been a goal not only achievable but also within practical reach. This therapy which is based on a combination of ICS LABA is thus the foundation of asthma therapy today. The evolution of trials like the AMD adjustable maintenance dosing and the SMART approach have also refocused on the need for additional doses of ICS thus further demonstrating the significance of ICS therapy in the over all picture of asthma control. There appears to be a very marginal difference in the efficacy of the various inhaled steroids and all of them appear equally effective. Salmeterol and formoterol are the two LABAS commonly used and formoterol appears to have a better therapeutic profile with regards to onset and duration of therapy and also a dose responsive profile. Thus combination of formoterol and ICS appear to be the drugs of choice in asthma today.

This emphasis on inhaled steroid and the combination with the LABA is the backbone of GINA and various other guidelines that dictate asthma therapy today. One look at the guidelines will tell us that the inhaled steroid has the most effective and versatile profile at every single step of therapy. The introduction of levels of control have further simplified the GINA guidelines and made them more practical and effective.

The concept of one airway one disease appears to be the corner stone of the ARIA document and has very relevant implications for the successful therapy of not only rhinitis but asthma as well. Intra nasal and inhaled steroids appear to be the foundation of therapy today and establish the concept of one airway one disease one drug.

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