Concept of unified airway

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Abstract:

According to European rhinological society guidelines chronic rhinosinusitis with nasal polypi and chronic rhinosinusitis without nasal polypi are two different entities. This article attempts to review published literature which attempts to study link between nasal polyposis and lower airway disorders. The concept of unified airway attempts precisely to explain this linkage. Developmentally and functionally it makes sense to combine both upper and lower airways in studying pathophysiology of various airway disorders.

Introduction:

An attempt to define the following terminologies will not be out of place.

Rhinosinusitis / Nasal polyposis:

Number of authors have attempted to define this condition, majority of these definitions were based on symptomatology and duration of the disease. Till date there is no universally accepted definition of this condition [1]. European rhinological society has stepped in to define rhinosinusitis in unambiguous terms.

Rhinosinusitis / Nasal polyposis is defined as:

Inflammation of mucosal lining of nose and paranasal sinuses characterised by two or more symptoms, one of which should be either nasal block / nasal obstruction / nasal discharge (anterior / posterior discharge):

Presence or absence of facial pain / tenderness

Reduction / Loss of smell.

And / either:

Endoscopic signs of :

Polypi

Mucopurulent discharge / oedematous mucosa over middle meatus

And / either

CT scan changes showing mucosal oedema / OMC obstruction

The severity of the disease can be classified using visual analogue scale:

1. Mild – Visual analogue score 0-3

2. Moderate – Visual analogue score 3-7

3. Severe – Visual analogue score – 7-10

Visual analogue score of more than 5 affects quality of life of the patient.

Duration of the disease:

Acute : < 12 weeks with complete resolution of symptoms

Chronic: > 12 weeks without complete resolution of symptoms. Chronic rhinosinusitis can show periods of acute exacerbations.

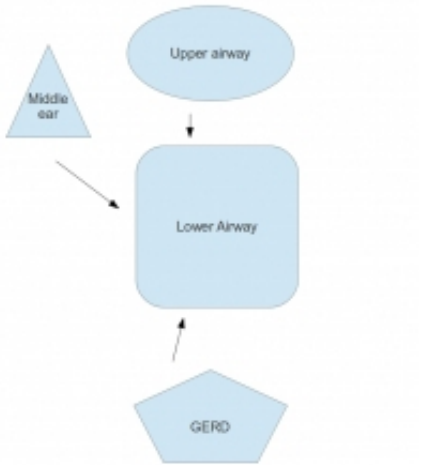
Studies attempt to divide chronic rhinosinusitis with / without nasal polyposis based on inflammatory markers [2], [3]. Why nasal mucosa balloons in to polypi in certain patients with chronic sinusitis is still an enigma. In these patients polypi tend to recur even after apparantly complete surgical removal. Some studies have demonstrated pronounced eosinophilia and IL – 5 expression in patients with nasal polyposis than in those with chronic sinusitis without nasal polypi. Samter in his classic description states the presence of a triad in some of these patients. This triad goes after his name “Samter’s Triad”. Patients with Samter’s triad have asthma, aspirin hypersensitivity and nasal polyposis.

Majority of patients with chronic rhinosinusitis with nasal polypolis manifest with asthma and bronchial hyper-responsiveness 3. The role of medico-surgical management of nasal polyposis in managing patients with associated lower airway symptomatology is still not clear. Some studies even go to the extent of suggesting that surgical removal of nasal polypi in these patients actually worsens bronchial asthma in these patients [4]. Vleming [5]in his study has reported that nasal polypectomy actually helped in alleviating symptoms due to bronchial asthma in these patients.

Theoretically larynx is considered to be the boundary between the upper and lower airway system. Functionally speaking the major function of the upper air way is to conduct air and also make it fit for gas exchange in the lower airway by adding moisture to inspired air. The lower air way plays a vital role in gas exchange. Currently the term unified airway encompasses the following structures: middle ear mucosa, nasal cavity and paranasal sinus mucosa, and the entire tracheo bronchial tree. Studies have demonstrated that pathologies affecting one portion of this unified airway ultimately progresses to involve the other areas as well.

It was Keller in 1920 [6]who noticed that commonly patients with lower respiratory diseases also had complaints pertaining to the upper airway as well. Keller also hypothesised that decreased ability of upper air way to humidify inspired air leads to worsening of bronchial asthma.

Concept of unified airway



Current status is that whenever a diagnosis of rhinitis or asthma is considered then the entire airway should be

evaluated [7]. Even Gastro oesophageal reflux [8]disease has also been implicated with airway disorders. Hence no investigation for asthma / upper airway disorder is complete without upper GI endoscopy.

Criteria supporting the concept of unified airway:

1. Patients with upper airway diseases like rhinitis and rhinosinusitis have increased prevalence of lower respiratory disorders like bronchial asthma

2. Patients with lower airway disorders like bronchial asthma have increased prevalence of rhinosinusitis

3. Pathophysiological mechanisms causing both upper and lower airway disorders are more or less similar

4. Treating one component of unified airway disorder should have beneficial effect on the other components as well.

Corren's[9] role in strengthening the concept of unified airway:

Corrent in his classical paper clearly demonstrated the coexistance of rhinitis in patients with bronchial asthma. He also observed a temporal relationship between bronchial asthma and rhinitis. He stated that attacks of bronchial asthma was preceded by an attack of rhinitis.

According to Guerra et al [10]sufferers of allergic rhinitis are three times more likely to develop asthma when compared to controls in their study.

Starting from 1991 when Natioal Institute of Health [11]classified asthma as inflammatory disease, thereby shifting the focus from bronchospasm to inflammation. This caused a major shift in the treatment stratergy. Inflammation is the common denominator defining both upper and lower airway disorders. It should also be stressed at this point that the lining epithelium of both upper and lower airway i.e ciliated columnar epithelium functionally are similar. In response to chronic inflammation the mucosal lining of both upper and lower airway demonstrate similar histopathological patterns i.e. Inflammatory cellular infiltrate and eventual thickening of basement membrane. Eosinophils have been

identified as the common inflammatory cell causing problems in disorders involving both upper and lower airway. Other inflammatory cells that have been implicated in unified airway disorders include CD4 T-Lymphocytes and mononuclear cells.

The most compelling proof of the concept of unified airway comes from the fact that the inflammatory mediators released starting the cascade of inflammation in the upper and lower airway are virtually the same. Inflammatory triggers from one portion of airway also affects other portions as well.

Hence by viewing the entire airway as a single unit patient reaps the benefit of unified treatment protocol. It is always prudent to control chronic sinusitis in addition to managing asthma by bronchodilators. Successful management of chronic rhinosinusitis results in decreased asthma medication[12] in these patients with tell tale improvement in pulmonary function. An additional benefit being reduced number of exacerbations.

Mechanisms responsible for unified airway disease:

1. Nasobronchial reflex

2. Loss of nasal protection to lower airway

3. Shared inflammation through out the respiratory tract

Nasobronchial reflex:

This concept was first proposed by Sluder[13] who stated that nasal irritation can cause bronchial irritation leading on to bronchospasm. Kaufmann[14] and Wright added further proof to this concept. In their study they applied silica particles to the nasal mucosa of individuals without bronchial asthma and demonstrated there is increased lower airway resistance following this application. Even in individuals in whom this reflex is not readily demonstrable delayed changes have been demonstrated in the lungs starting from 30 mins – 4 hours following antigenic challenge of nasal mucosa. This observation suggests that other mechanisms in addition to direct reflex arc[15] could be responsible for interaction between upper and lower airway structures.

Loss of nasal protection to nasal airway:

This concept was first proposed by Shturman – Ellestein in 1978 [16]. In their classic experiments performed on volunteers they were able to demonstrate that mouth breathing worsened bronchospasm, while nasal breathing reduced lower airway resistance. This phenomenon can be explained when the primary function of the nosal airway (airconditioning) is taken into consideration. Lower air way responds to unconditioned inspired air by increasing their resistance to the inspired air. This can infact be considered to be a protective mechanism.

Shared inflammation:

This concept is also catching up as one of the explanations for common pathological basis of unified airway disorders. This concept has found its root from the observation that inflammatory disorders of various portions of the unified airway are caused by release of same immune / inflammatory mediators. Braunstahl et al [17]observed that stimulation of one portion of the airway mucosa with anigen results in a system wide inflammatory changes within hours.

Conclusion:

The concept of unified airway disorders has infact shifted the focus from individually managing various disorders affecting the components of airway to that of unified management modality. This calls for

multidisciplinary approach in managing these patients in an optimal manner. Specialities involved in designing management protocol for unified airway disorders include Thoracic medicine and otolaryngologists.

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