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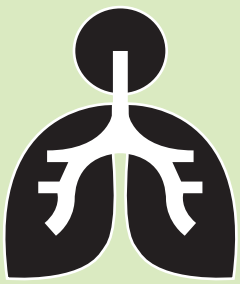
Asthma Focus

The Asthma-COPD Overlap Syndrome



Asthma and Chronic Obstructive Pulmonary Disease (COPD) are pulmonary disorders characterized by various degrees of airflow limitation, inflammation and tissue remodeling. Bronchial asthma, an allergic disease that develops in childhood, is physiologically characterized by reversible airflow obstruction. It has an episodic course and a generally favorable prognosis, as it responds well to anti-inflammatory treatment. In contrast, pure COPD is caused by tobacco smoke, develops in mid-life or later and is characterized by incompletely reversible airflow limitation that results in a progressive decline in lung function and leads to premature death.

These definitions describe the physiological and anatomic extremes of asthma and COPD and allow them to be recognized as distinct disease entities. However, in clinical practice, many older patients have patho-biological and symptomatic features of both diseases, necessitating a reevaluation of the concept of COPD and asthma as separate conditions. Asthma and COPD are both chronic inflammatory lung diseases. In both conditions, inflammation is associated with structural alterations at large and small airway levels. This can result in a transient phenotypic overlap or a combined syndrome with characteristics of both diseases.

The accepted definitions for the distinct obstructive airway diseases and their component syndromes are described in table 1. When a patient exhibits features of more than one condition, then they have an overlap syndrome. This article focuses on the overlap between asthma and COPD. The current descriptions of asthma and COPD have been simplified into patterns of abnormal airway physiology (fig 1) which, in conjunction with symptoms, can be used to facilitate clinical recognition. There is a need to broaden these descriptions to accommodate the common clinical reality of people who fit the criteria for more than one condition. Guideline development in COPD has placed more emphasis on recognizing COPD as a disease where incomplete reversibility of airflow obstruction is the defining characteristic. This allows easier recognition of obstructive airway diseases in general, and COPD in particular. However, the potential for overlap of the individual obstructive airway disease syndromes has received less attention.

A recent study of the overlap in obstructive airway diseases using data from large population studies found that 17% and 19%, respectively, of patients had more than one condition present. With increasing age, there was a greater increase in the proportion of patients with obstructive lung disease who had overlapping diagnoses (fig 2). In older patients, a combined syndrome of asthma and COPD was the most common situation, as now confirmed using objective testing. Fewer than 20% of older patients have the classical phenotypes of emphysema alone or chronic bronchitis alone.

Asthma versus COPD

In theory, asthma and COPD are different diseases each with a unique natural history and pathophysiology. Asthma and COPD are distinct diseases that develop by unique mechanisms. It is widely accepted that asthma generally manifests as intermittent and reversible airway obstruction, where as COPD is progressive and irreversible.

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Table 1: Definition of obstructive airway syndromes	
Syndrome	**Definition**
Asthma	Episodic respiratory symptoms Variable airflow obstruction occurring spontaneously, with treatment or after provocation
COPD	Incompletely reversible airflow obstruction
Overlap syndrome	Asthma and COPD-that is, symptoms of increased variability of airflow and incompletely reversible airflow obstruction
Chronic bronchitis	Symptomatic mucus hyper secretion with cough and sputum daily for at least 3 months over 2 years
Emphysema	Abnormal airspace enlargement
Variable airflow obstruction	Increased diurnal variability of peak flow: maximum-minimum/average _10% Increased response to bronchodilator: _200 ml FEV1 and _12% baseline Increased airway responsiveness: provocation dose or concentration _normal
Incompletely reversible airflow obstruction	Post bronchodilator FEV_1 _80% predicted and FEV1 /FVC _70%
Bronchodilator responsiveness	Improvement in FEV_1 _15% and 400 ml after a therapeutic dose of inhaled rapid acting b2-agonist
Airway hyper-responsiveness	Significant fall in FEV_1 from a stable baseline after inhalation of bronchial provocation stimulus occurring at a stimulus dose less than required to induce a significant change in FEV_1 in healthy controls.

COPD-Chronic Obstructive Pulmonary Disease; FEV_1- Forced Expiratory Volume in <1st second; FVC- Forced Vital Capacity

According to current guidelines, the post bronchodilator response in asthma shows complete reversibility of airway obstruction. In COPD, there is either no reversibility (i.e., fixed obstruction) or thereis partial reversibility of airway obstruction following bronchodilator, described as "COPD with partial reversibility". In "COPD with partial reversibility", one can demonstrate reversibility as an improvement in lung function, but the patient remains obstructed on spirometric measurements (hence, the designation of "COPD" rather than "asthma"). Preserved carbon monoxide diffusion capacity (DLCO) on PFT and a higher ratio of airway-to-lung parenchymal abnormalities (on lungimaging by high-resolution chest tomography) may also distinguish asthma from COPD.

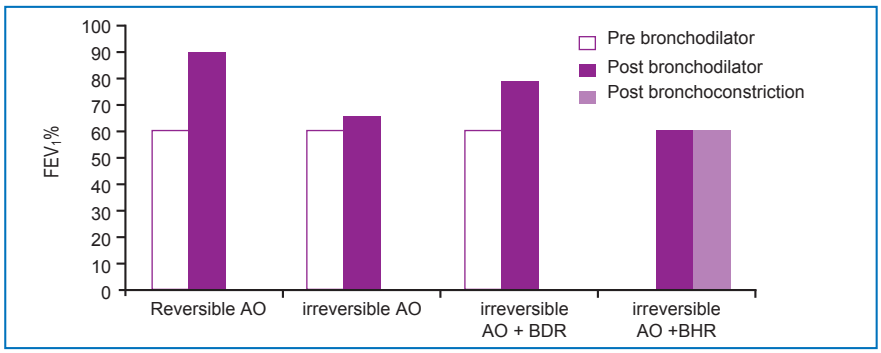

Figure 1: Physiological patterns of airflow obstruction, expressed as forced expiratory volume <1st second in (FEV_1), percentage predicted. Reversible AO- reversible airflow obstruction with improvement in FEV_1 <1st second after bronchodilator; irreversible AO-incompletely reversible airflow obstruction, post-bronchodilator FEV_1 is <80%; irreversible AO+BDR- incompletely reversible airflow obstruction with significant bronchodilator responsiveness (BDR); irreversible AO+BHR- incompletely reversible airflow obstruction with significant bronchial hyper-responsiveness with fall in FEV_1 after bronchoconstrictor.

- The label 'asthma' can be applied to reversible AO, irreversible AO+BDR and irreversible AO+BHR.
- Chronic Obstructive Pulmonary Disease (COPD) can be applied to each of the conditions with irreversible AO+BHR.

Clinically, the distinction between asthma and COPD ismost apparent at the extremes of age, where younger patients tend to have more asthma symptoms and older patients (age > 60) tend to have COPD symptoms. A history of cigarette smoking and evidence of emphysema in an older patient with spirometric airflow obstruction would favor COPD. A nonsmoking younger patient with a history of childhood asthma or wheezing and atopy with reversible air-flow obstruction would favor asthma. Though symptoms can often overlap, Beeh and colleagues developed a questionnaire to differentiate asthma and COPD. On a scale of 1 to 15, the questionnaire performed best at a cut off of 7 with a sensitivity of 87.6% for COPD, though 20% of patients had overlap features (scores 6-8). Practically speaking for the clinician, the distinction between asthma and COPD is largely based on clinical findings.

Major differences exist in the structural and inflammatory signatures of asthma and COPD when studied in isolated, well-defined populations. These include elevated IgE, induction of T_h2 cells, eosinophilic infiltration, reticular basement membrane thickening and smooth muscle hyperplasia in asthma. In contrast, increased neutrophils, induction of T_h1 and T_h17 cells, TGF β-induced small airway fibrosis, goblet cell hyperplasia, and MMP elastic tissuedestruction are typically found in COPD. Though there are major histologic differences between asthma and COPD, endobronchial biopsy is not widely used in the workup and management of obstructive lung disease as the risk-benefit profile is often unfavorable. Existing treatment options for asthma and COPD highlight some important differences. For example, COPD with reduced DLCO may qualify for supplemental oxygen therapy and referral to a pulmonary rehabilitation program. Severe allergic asthma with an appropriate high level of serum IgE may qualify for treatment with omalizumab and in those with allergic asthma not well controlled on ICS, leukotriene receptor antagonists or 5-lipoxygenase inhibitors may improve symptoms.

Table 2. Characteristic Similarities and Differences between Asthma, Chronic Obstructive Pulmonary Disease(COPD) and the Overlap Syndrome

	Asthma	COPD	Overlap Syndrome
Pathology	Chronic airway inflammation, typically eosinophilic and driven by CD4 cells. Neutrophilic inflammation has been observed in theairways of some asthmaticsand is associated with increased steroid resistance.	Chronic airway inflammation, typically neutrophilic and driven by CD8 cells. Eosinophilic inflammation has been observed in the airways of some patients with COPD and is associated with greater steroid sensitivity.	Pathologic overlap in the inflammatory profiles of both asthma and COPD, particularly among the elderly
Pathophysiology	Reversible airway obstruction; progressive deterioration over time is uncommon.	Partially reversible airway obstruction; progressive deterioration overtime is typical.	Functional overlap between asthma and COPD, particularly among the elderly.
Treatment of acute exacerbations			
Systemic corticosteroids &inhaled bronchodilators	Improve symptoms and lung function and decrease the length of hospital stay.	Improve symptoms and lung function and decrease the length of hospital stay.	No data available.
Maintenance treatment of stable disease			
ICSs	The mainstay of treatment in patients with persistent asthma.	Less effective response. ICSs are recommended for patients with more severe COPD FEV_1 <50% of predicted) whose symptoms are not optimally controlled with inhaled bronchodilators. ICS mono therapy is not recommended.	No data available.
Inhaled bronchodilators	Inhaled short-acting β_2-agonists are the mainstay of treatment for intermittent asthma. Inhaled long-acting β_2-agonists mono therapy is not recommended	The mainstay of treatment in patients with COPD; inhaled anticholinergics may be more effective than inhaled β_2-agonists as mono therapy in COPD.	No data available.

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ASTHMA SCOOP

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Many clinicians recognize that asthma and COPD can appearmore similar than dissimilar clinically. Typical symptoms of dyspnea, wheezing or cough do not have the sensitivity orspecificity to distinguish these two disorders among older adults after excluding comorbid conditions such as heart failure, diastolic dysfunction, aspiration, GERD or vocal corddysfunction. According to Soriano et al., asthma and COPD are diffcult to differentiate because (1) the conditions are viewed as part of a disease continuum; (2) they have strong overlapping features; (3) there is no incentive to differentiate whether their treatment and prognosis are the same; (4) there is a lack of clear guidelines as to how the distinction can be made in clinical practice; (5) uncertain criteria are used byphysicians to classify patients as having asthma or COPD. Despite years of research and the prolific expansion of guidelines from both European and American respiratory societies, distinguishing these two common diseases remains adaunting challenge.

Recent reviews under score the pitfalls in diagnosis, management and treatment of overlap syndrome, asthma, and COPD. The Dutch Hypothesis maintains that asthma and AHR predispose patients to develop COPD later in life and that asthma and COPD are different expressions of asingle disease (based on the timing of environmental and epigenetic influences amidst a common genetic background). Some authorities argue that obstructive lung disease is aprogressive disease that begins in early childhood, where COPD is the final manifestation. Recent epidemiologic findings, from a long-term cohort study in the United States, point to asthma as a significant risk factor for the future development of COPD. Unless there are clear exposures, such as a prolonged smoking history in a person with severe emphysema, clinicians recognize that considerable phenotypic heterogeneity makes clear distinctions between obstructive lung diseases problematic.

Not all comparisons between asthma and COPD pathology show unique structural differences. In 100 select patients with clinically determined asthma and COPD who underwent endobronchial biopsies, there were no statistically significant differences in key pathologic features. Thoug heosinophilic infiltration and basement membrane thickeningwere associated with asthma, the overall differences in these features, metaplasia, and epithelial inflammation did not allow for pathologic differentiation. Airway remodeling and the lung's specific repair responses may account for some of the pathological similarities reported in asthma and COPD. These structural similarities in small airways may contribute to the observed clinical overlap. Up to 50% of COPD patients can have AHR due to the narrowing of the irdistal airways and predisposition to bronchospasm.

Patients with more physical activity requires fewer hospitalizations from lung condition

Taking daily walks of at least two miles can reduce hospitalizations from severe episodes of a life-threatening breathing disorder, new research suggests. The study published in the journal “Respirology” reported that, Chronic Obstructive Pulmonary Disease (COPD) patients without regular walking regimens had about twice the rate of hospitalizations triggered by the condition compared to those who maintained the highest levels ofphysical activity. This was defined as walking between roughly two and four miles each day. The third-leading cause of death in the United States, COPD claims about 134,000 lives annually, according to the American Lung Association. COPD describes a group of progressive respiratory conditions that include emphysema and chronic bronchitis. Smoking is the most common risk factor for the condition, but others include family history and inhaling pollutants such as fumes, chemicals and dust, according to the COPD Foundation.For the new study, Esteban and his team recruited about 550 COPD patients from five Spanish respiratory clinics. The researchers calculated participants' exercise totals based on self-reported tallies of the distance they walked during the course of a week. The data was compared to hospitalization records and patients were tracked for at least two years.Patients who maintained moderate or high level of exercise -- which in most cases equated to a walking regimen- were hospitalized only 53 percent as often as those who didn't walk regularly, Esteban said. Dr. Venessa Holland, a pulmonologist at Houston Methodist Hospital in Texas, said the study "helps solidify what we've been doing with patients with any obstructive lung disease." But Holland, who wasn't involved in the research, said a "major flaw" in the study was the self-reported patient data. "We know people self-report by pure error," she said. "There's no real documentation [their mileage totals] happened." "Physical activity is important, as with all diseases, to improving health and quality of life," she said. "People used to think that if you have COPD, you shouldn't be physically active. This reinforces that you should." Study author Esteban recommended that COPD patients - who may have difficulty trekking long distances due to breathlessness - walk routinely with others or even with a dog to keep motivation levels high. Holland agreed, saying the support of family members and health care providers is crucial to help these patients stick with a walking regimen. "If they're motivated, they'll continue," Holland said. "I can't tell you how many patients I have with severe lung disease who I haven't had to hospitalize, but they're proactive about physical activity."

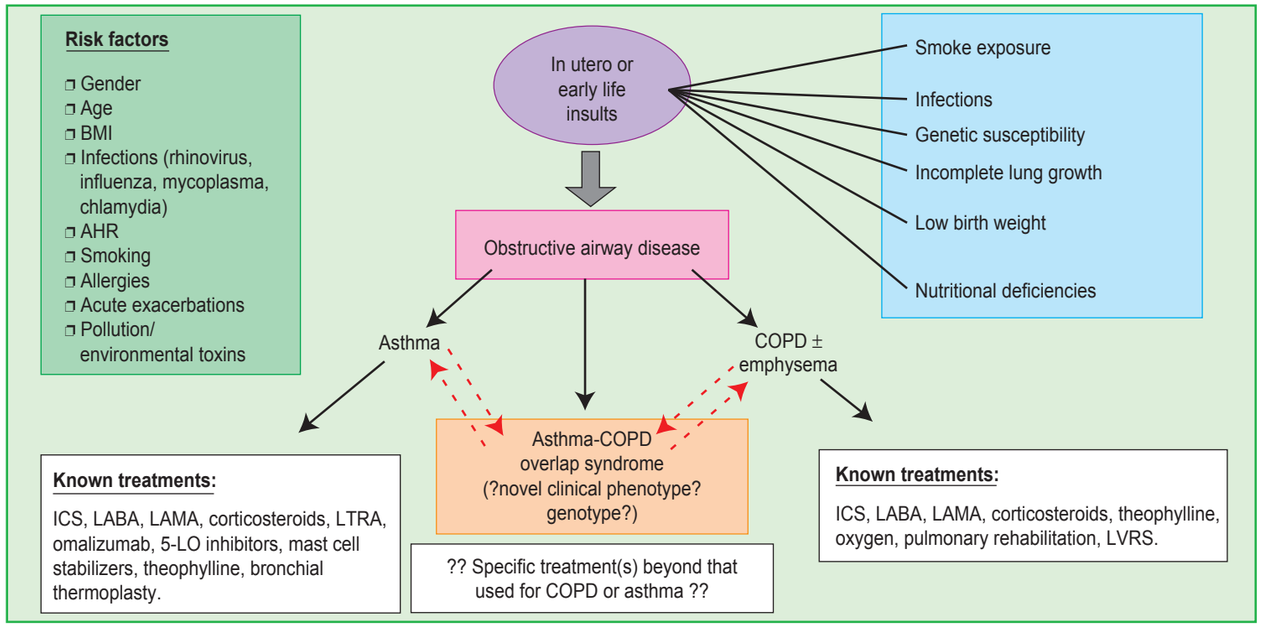

Figure 2: Common risk factors for the development of obstructive airways disease. Although asthma, COPD, and asthma-COPD overlapsyndromelikely share many of the same risk factors, it is unknown whether the overlap syndrome is a unique genotype and pathophysiologically unique clinical phenotype. The progression from early life insults to pediatric disease and finally chronic obstructive airway disease inadulthood involves complex genetic, epigenetic and environmental interactions. Because the underlying pathogenic mechanisms that lead tothe overlap syndrome have not be elucidated, we have no known disease-specific therapies other than those extrapolated from clinical trialsdone in asthma- or COPD-only subjects. In this lies an opportunity for further research focused on the overlap syndrome as the “third arm”of the most common obstructive airway diseases. Given that asthma-COPD overlap syndrome prevalence increases with age, knowledgeabout this syndrome will have great clinical and economic implications for our aging population.

Abbreviations: inhaled corticosteroid (ICS), long-acting beta-agonist (LABA), long-acting muscarinic antagonist(LAMA), leukotriene receptor antagonist (LTRA), 5-lipoxygenase (5-LO), body mass index (BMI), airway hyperreactivity (AHR), and lungvolume reduction surgery (LVRS).

However, despite a similar degree of fixed airway obstruction, asthmatic and COPD airway inflammation and structural changes do not appear the same. But as imaging and airflow resistance assessment techniques improve, the small airway changes associated with asthmaand COPD which appear dissimilar may result in a phenotypically similar outcome. Limitations inherent to the different patient cohorts studied and research methodologies used may confound and limit final conclusions regarding aclear histopathologic-physiologic link.

Asthma and COPD can overlap in their inflammatory sputum profiles and lung function. Severe bronchial asthma with fixed obstruction has an increased number of neutrophils similar to COPD. Eosinophilic inflammation in COPD may play a substantial role and be associated with greater postbronchodilator reversibility. Chronic bronchitis or emphysema patients with airway eosinophilia demonstrate increased airway reversibility and respond more readily to corticosteroid therapy. In smokers with severe obstructive bronchitis, sputum eosinophilia predictsa beneficial response to prednisone treatment. Evidence of bronchodilator response (i.e., reversibility) or AHR may be an important measure not only for diagnosis but also for prognosis with respect to the rate of lung function decline and asthma mortality. However, not all studies agree that bronchodilator responses correlate with the subsequentrate of lung function decline. Some authors found that the prognosis in terms of all-cause mortality is strongly correlated with age, smoking, and the best attainable FEV_1 regardless of reversibility. Given that overlap features become prevalent with increasing age and a smoking history, there are important implications for prognosis regarding this syndrome.

Asthma-COPD overlap may have origins in childhood, for example, infections, atopy and tobacco-smoking exposure but escapes earlier diagnosis because of current definitions and guideline-based approaches to chronicobstructive lung diseases. This could result in a heuristic biaspotentially overlooking patients in their 40s and 50s with overlap syndrome by labeling them as lone “COPD” or partially reversible “asthma.” This becomes even more relevantin the elderly population (age ≥ 65 years) where the overlap syndrome is increasingly recognized because of variablere sponse to guideline drug therapy, more frequent health carere source utilization and multiple comorbid conditions.

Asthma-COPD Overlap Syndrome

The asthma-COPD overlap syndrome is not clearly defined. It is a syndrome in which older adults with a significa smoking history have asthmatic features to their chronic obstructive airway disease.

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Individuals may have a history of childhood asthma or asthma as young adults. There are many cases of pathologic and functional overlap between asthma and COPD, yet authorities debate whether this overlap syndrome represents the coexistence of two common airway diseases or whether there are common underlying pathogenic mechanisms leading to this common phenotype. Overlap syndrome appears to share many of the same disease risk factors as that of asthma and COPD (Figure 2). Thisis a major challenge to our understanding of pathogenesis; however, this observation can guide future basic studies andthe development of novel therapies.

The exact definition of this syndrome is evolving. For example, Gibson and Simpson defined overlap syndrome asasthma and COPD, that is, “symptoms of increased variability of airflow and incompletely reversible airflow obstruction,” but this is rather limited when based solely on FEV_1 and bronchodilator response. For example, patientswith COPD can demonstrate a variable and significant degree of reversibility of airflow obstruction following bronchodilator challenge. Nearly 66% of COPD patients in the UPLIFT trial improved their FEV_1 by more than 15% after receiving 80 g ipratropium and 400 g of salbutamol, yetthey were not considered to have overlap syndrome or concomitant asthma. Thus, a clear definition remains elusive regarding overlap syndrome since there is no consensusin the literature. Moreover, the presence of exercise intolerance and static or dynamic hyperinflation and indices of pulmonary emphysema in the aging patient may be important factors to include when considering overlap syndrome-a complex question needing further research.

Few studies have specifically investigated the asthma-COPD overlap syndrome. Epidemiologic studies report an estimated prevalence of 20%. Patients with coexisting asthma and COPD present similarly to pure asthma or COPD, manifesting signs and symptoms of obstructivelung physiology. However, patients with overlap syndrome have worse lung function, more respiratory symptoms, anda lower health-related quality of life than either disease alone. They also consume more medical resources compared to asthma or COPD alone, as much as 2 to 6 times higher. From a cost perspective alone (besides the importance of proper diagnosis and treatment), there is amplereason to increase our research efforts in this area.

Cigarette smoking interacts with the inflammation andremodeling that occur in asthma and COPD. The diseases are most different when nonsmoking asthmatics with AHR and smokers with COPD but no AHR are compared. Smoking influences the pattern of inflammation and steroid responsiveness. Asthmatics who smoke have more neutrophils in their airways, rather than eosinophils, resembling COPD. Smoking promotes neutrophilic inflammation in both asthma and COPD which results in increased steroid resistance. Disease severity increases as thepatterns of inflammation become more similar and steroidresistance increases. Similarly, mucosal eosinophils increase in acute exacerbations of mild COPD, a feature normally seen in asthma. This similarity in inflammatory responses may be one pathophysiologic linkto the clinical phenotype of asthma-COPD overlap. Despite published definitions of asthma and COPD by international respiratory societies such as ATS/ERS and global initiatives such as GINA and GOLD, there remains considerable clinical and pathologic overlap between these two disorders which defies such limited definitions.

Overlap syndrome is more prevalent in the aging population. In general, lung function tends to deteriorate naturallywith increased age. Elderly patients with asthma display more features of fixed obstruction than their younger counter-parts, and they tend to have more severe symptoms. Their asthma may manifest as chronic persistent airflow obstruction mimicking COPD. AHR also increases with age where it is three times higher in the elderly compared to nonelderly adults. Age is a very important variable when assessing obstructive lung diseases given the known changesin lung function that occur with increased age and the possible role of aging genes especially in COPD. Because of this age effect, appropriate comparisons between asthmaand COPD should be made in patient cohorts with the sameor similar age, inhaled exposures and disease severity. Indeed, increasing age may be a powerful factor “blurring theline” that separates asthma from COPD, thus, contributing to the manifestation of overlap syndrome

Treatment of Asthma-COPD Overlap Syndrome

Clinical recognition of the overlap between asthma and COPD is based on inflammatory features. Inflammation in asthma is associated with increased airway hyper-responsiveness, which leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night or in early morning.This inflammation is present even in those with very mild asthma and is unique in that the airway wall is infiltrated by lymphocytes, eosinophils, macrophages/monocytes and mast cells. In contrast, the pathological hallmarks of COPD are destruction of the lung parenchyma (pulmonary emphysema) and inflammation of the peripheral airways (respiratory bronchiolitis) and central airways, along with parenchymal inflammation. There is a marked increase in macrophages and neutrophils in the bronchoalveolar lavage fluid and induced sputum.

Given that asthma and COPD are both pulmonary disorders characterized by various degrees of inflammation and tissue remodeling, they present common therapeutic targets.

Althoughspecific interventions vary by disease, the treatmentgoals of asthma and COPD are similar and drivenprimarily by patient-centered outcomes such ascontrolling symptoms, optimizing health status andquality of life and preventing exacerbations (i.e., reducing their frequency and severity). In general, therapies for COPD have a muchmore limited effect compared with those for asthma. While inhaled corticosteroids (ICSs) arethe cornerstone of the pharmacologic management of patients with persistent asthma, inhaled bronchodilators (β_2-agonists and anti cholinergics) are the therapeutic mainstay for patients with COPD. There are no disease-modifying medications currently available that can alter the progression of AO in either asthma or COPD. Smoking cessation, however, is an essential component of the successful management of any obstructive airway disease.

At present there is no randomized clinical trial data to help guide therapeutic interventions inasthma-COPD overlap syndrome. In fact, patients with overlapping asthma and COPD are frequently excluded from treatment trials for either condition, which limits the generalizability of these trials in this neglected patient population. However, practical treatment principles are similar to those for asthma or COPD and involve a comprehensive therapy directed toward airway inflammation, AO and AHR.

In general, the principles of workup and treatment are similar for asthma, COPD and the overlap syndrome. Becausethis syndrome is seen more commonly in older populations, there may be a higher probability of adverse reactions to thevarious classes of inhaled agents or systemic corticosteroids. Cognitive deficiencies leading to lower medication compliance may be an issue and general under-diagnosis andunder-treatment may occur. Martinez et al. recommend treatment with a ICS/LABA combination, with orwithout a long-acting anticholinergic agent (i.e., LAMA). Smoking cessation, oxygen supplementation, pulmonary rehabilitation and vaccines are all reasonable interventions. At present, there are no randomized clinical trial datato help guide therapeutic interventions in overlap syndrome. Based on clinical experience, a symptom-targeted approach was recommended. For dynamic obstruction and/or hyperinflation, bronchodilators may provide the greatest benefit. Whether LAMAs alone or in combination with LABAs are appropriate in overlap syndrome remains to be elucidated. For patients in whom bronchospasm is described and/or demonstrated, bronchodilators and ICSs are reasonable options. Adjunctive treatments such as leukotriene receptor antagonists, 5-lipoxygenase inhibitors, methylxanthines, oromalizumab deserve further study and should be administered by pulmonary or allergy subspecialists. As the prevalence of overlap syndrome increases with age, targeting non-respiratory age-related changes which may influence respiratory disease is paramount. This includes targeting nasal obstruction symptoms (due to non-allergic or allergic rhinitis, mucosal dryness or vasomotor symptoms) with nasal irrigation, nasal steroids, and/or nasal anticholinergic. Additionally, treating co-morbidities such has heart failure orchronic aspiration is important, especially GERD or VCD which can be subclinical. These recommendations arebased on our clinical experiences with the overlap populationat UCDMC, where optimal treatment strategies still require customization and additional study.

Ref : Zeki, A. A., Schivo, M., Chan, A., Albertson, T. E., & Louie, S. (2011). The Asthma-COPD Overlap Syndrome: A Common Clinical Problem in the Elderly.Journal of Allergy , 2011, 1-10.

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Editorial Note

Dear Doctor,

We are happy to present you the 2nd issue of "Asthma Focus" Newsletter, 2014. In this issue we have concentrated on The Asthma-COPD Overlap Syndrome. We hope you will enjoy reading the publication!

We appreciate your comments and queries.

Please participate in Quiz competition & win prizes.

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