



TO ASSESS THE ASTHMA COPD OVERLAP SYNDROME AMONG PATIENTS OF ASTHMA AND COPD.

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ABSTRACT

Background and Objective: Asthma-COPD Overlap Syndrome (ACOS) represents a complex interaction between asthma and Chronic Obstructive Pulmonary Disease (COPD), exhibiting characteristics of both conditions. This overlap presents significant challenges in clinical practice, often resulting in increased disease activity, higher rates of exacerbations, hospitalizations, and mortality. Despite its considerable impact on health, ACOS is frequently under diagnosed and inadequately treated, mainly due to the similarity of its symptoms and the lack of standardized diagnostic criteria. This study aims to evaluate the prevalence and clinical features of ACOS in patients with asthma and COPD, with the goal of enhancing diagnostic accuracy to improve treatment outcomes.

Materials & Methods: A cross-sectional observational study was conducted at the National Institute of Medical Sciences and Research, Jaipur. Patients diagnosed with asthma or COPD attending the respiratory medicine outpatient department were screened for ACOS using Global Initiative for Asthma (GINA) and Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines. Data on demographics, clinical presentation, spirometry, and inflammatory biomarkers were collected and analyzed to differentiate between asthma, COPD, and ACOS.

Result: Out of 330 patients screened, 20% met the criteria for ACOS. Of 66 patients, 33 (50.0%) were COPD and 33(50.0%) were diagnosed as asthma . Of 66 patients ,30 were male and 36 were female . ACOS in COPD group had significantly higher number of respiratory rate ,pulse rate and lower SpO₂ and lower spirometric variables (P <.001) compared to ACOS in asthma group . Sputum eosinophil and serum IgE count is significantly higher in ACOS in asthma group (P <.001).

Conclusion: The prevalence of ACOS was 20% with higher proportion of females. ACOS in COPD group had higher respiratory rate , pulse rate and low saturation(SpO₂) as compared to asthma group . This indicates ACOS in COPD group has more work of breathing, hypoxemia as compare to asthma group which can leads to more exacerbation. The positive link between the low spirometry variables (pre and post bronchodilator FEV₁,FVC, FEV₁/FVC) in ACOS- COPD group linked to more disease severity as compared to asthma group. So, these patients can more frequently hospitalised and should be observed more closely at follow-up. So, identification of ACOS patients in COPD may be an important goal for the patient management.

Keywords: Asthma-COPD Overlap Syndrome, Chronic Obstructive Pulmonary Disease, Asthma, Spirometry, Inhaled Corticosteroids and Exacerbations.

INTRODUCTION

Asthma and Chronic Obstructive Pulmonary Disease (COPD) are two of the most common respiratory conditions that significantly impact public health worldwide.

Asthma is characterized by chronic airway inflammation, leading to variable but reversible airflow obstruction and hyper responsiveness to allergens and irritants. The condition typically presents in childhood but can affect individuals of all ages. The hallmark symptoms include wheezing, shortness of breath, chest tightness, and cough often worse at night or early morning and may be productive or dry, which are often triggered by exposure to allergens, exercise, or cold air. These symptoms are typically reversible with bronchodilators and anti-inflammatory treatment [1].

On the other hand, COPD is primarily a disease of older adults, most commonly caused by long-term exposure to noxious particles or gases, with cigarette smoking being the predominant risk factor. COPD encompasses two main pathological conditions: chronic bronchitis and emphysema. The disease leads to persistent airflow limitation, which is not fully reversible and tends to progressively worsen over time. Symptoms such as chronic cough, sputum production, fatigue and breathlessness (persistent dyspnea) are common, and COPD is often complicated by frequent exacerbations and significant comorbidities. Unlike asthma, the airflow limitation in COPD is not fully reversible, and symptoms tend to worsen progressively, especially in individuals with continued exposure to risk factors such as smoking or environmental pollutants [2].

Asthma-COPD Overlap Syndrome (ACOS) has emerged as a critical area of focus due to its unique clinical characteristics. ACOS refers to the condition in which individuals exhibit features of both asthma and COPD.

Patients with ACOS typically show a combination of persistent airflow limitation, a hallmark of COPD, along with the reversible airflow obstruction and heightened bronchial responsiveness characteristic of asthma. Symptoms include dyspnea, chronic cough and sputum production, frequent exacerbations compared to COPD.[3]

ACOS often involves both eosinophil inflammations seen in asthma, and neutrophilic inflammation, which is characteristic of COPD [4].Using bronchodilators alone may not sufficiently control asthma symptoms in ACOS, while exclusive reliance on corticosteroids may not adequately address the neutrophilic inflammation seen in COPD [5].

This overlap complicates the clinical management of the condition, as these patients experience more frequent exacerbations, worse health outcomes compared to those with asthma or COPD alone [6].ACOS requires a tailored management approach, which includes the use of inhaled corticosteroids, bronchodilators, and other pharmacological agents, depending on the dominant features of the disease. ACOS is more common in older adults, particularly in smokers with a history of asthma.

The exact mechanisms underlying ACOS are not fully understood, but it is thought that in patients with ACOS, both Th2-mediated eosinophilic inflammation (as seen in asthma) and neutrophilic inflammation (as seen in COPD) are often present, leading to both reversible and irreversible airflow obstruction ,persistent airway inflammation, airflow limitation, and frequent exacerbations [10,9].

MATERIAL AND METHODS

The present study is a comparative cross sectional study, involving 66 patients with asthma and COPD (33 each) admitted to Department of Respiratory Medicine at the National Institute of Medical Science and Research, Jaipur, Rajasthan from 1st May 2023 to 30th November 2024.

Inclusion Criteria

- Age \geq 40 years, irrespective of sex.
- Persistent respiratory symptoms, including chronic cough, sputum production, dyspnea, or wheezing.
- Diagnosed cases of COPD with a post-bronchodilator FEV1/FVC ratio $<$ 0.7.
- Exposure to risk factors, including:
 - Smoking history ($>$ 10 pack-years).
 - Indoor or outdoor pollution exposure.
- Diagnosed cases of Bronchial Asthma.
- History of atopy or allergies.

Exclusion Criteria

- Age $<$ 40years.
- Active pulmonary infections, including pulmonary tuberculosis.
- Other pulmonary morbidities such as bronchiectasis, lung malignancy, or interstitial lung disease.
- Patients diagnosed within 6 months of study commencement.
- Patients experiencing exacerbation of asthma or COPD at the time of evaluation.
- Patients on recent oral or injectable steroid therapy.
- Patients unwilling to provide consent.

Diagnostic Criteria for ACOS

Major Criteria:

1. Post-bronchodilator increases in FEV1 \geq 12% and \geq 400 mL.
2. Sputum eosinophilia $>$ 5%.
3. History of asthma.

Minor Criteria (at least one):

1. High total IgE $>$ 100 IU/mL.
2. History of atopy.
3. Post-bronchodilator increases in FEV1 \geq 12% and \geq 200 mL on \geq 2 occasions.

A diagnosis of ACOS was required to fulfill at least two major criteria or one major criterion and two minor criteria.

Data Collection

Participants undergo a thorough evaluation that includes:

1. Clinical Assessment: Detailed history taking, including symptoms, smoking history, environmental exposures, and history of atopy or allergies, and Comprehensive physical examination.
2. Spirometry:
 - o Pre- and post-bronchodilator testing to measure FEV1, FVC, and FEV1/FVC ratio.
 - o Assessment of bronchodilator reversibility (\geq 12% and \geq 200 mL increase in FEV1).
3. Radiological Evaluation: Chest X-rays or high-resolution computed tomography (HRCT) to assess lung parenchyma and airways.
4. Laboratory Investigations:
 - o Sputum analysis for eosinophilia ($>$ 5%).
 - o Total serum IgE levels ($>$ 100 IU/mL).
5. Questionnaire and Risk Factor Assessment: Structured questionnaires to evaluate environmental exposures, smoking habits, and quality of life.

Statistical Analysis

Data was analyzed using SPSS software (version 23) and Microsoft Excel. Statistical methods include:

1. Descriptive Statistics: Mean, standard deviation, frequency, and percentage to summarize data.
2. Comparative Analysis: Chi-square test for categorical variables, Student's t-test or ANOVA for continuous variables.
3. Correlation Analysis: To identify relationships between clinical, radiological, and spirometry features.

A **p-value $<$ 0.05** was considered statistically significant.

RESULTS

The study enrolled a total of 66 patients, evenly distributed between the asthma group and the COPD group, with each comprising 33 patients (50%).

The mean age of the participants was 53.0 ± 9.63 years. The sex distribution showed a slightly higher proportion of females (36; 54.5%) compared to males (30; 45.5%).

The distribution of occupations indicated that the majority of participants were labourers (20; 30.3%), followed by teachers (16; 24.2%), farmers (12; 18.2%), business persons (10; 15.2%), and engineers (8; 12.1%).

Regarding their area of living, most patients were from urban areas (30; 45.5%), followed by semi-urban (24; 36.4%) and rural areas (12; 18.2%). Smoking status revealed that 17 (25.75%) participants were current smokers, 25 (37.87%) were ex-smokers, and 24 (36.36%) were non-smokers.

The body mass index (BMI) classification showed that the majority of patients had normal weight (28; 42.42%), followed by underweight (25; 37.87%), overweight (11; 16.66%), and obesity (2; 3%).

The general examination of the study participants revealed a distribution of smoking status. Among the participants, 17 were current smokers, with 7 (41%) in the asthma group and 10 (59%) in the COPD group. Ex-smokers comprised 25 participants including 13 (52%) in the asthma group and 12 (48%) in the COPD group. Non-smokers accounted for 24 participants, with 13 (53%) in the asthma group and 11 (47%) in the COPD group.

Pallor was observed in 31 participants with 16 (51.6%) from the asthma group and 15 (48.4%) from the COPD group. Icterus was present in 33 participants, with 16 (48%) from the asthma group and 17 (52%) from the COPD group.

Pedal edema was reported in 31 participants with a higher prevalence in the COPD group, 19(61.3%), compared to the asthma group 12 (38.7%).The variance for pedal edema approached statistical significance with a p-value of 0.52.

All participants tested negative for HBsAg, HIV, and HCV, with an equal distribution between the asthma (33; 50%) and COPD (33; 50%) groups. The variance is considered non-significant with a p-value <0.05.

The mean values and standard deviations for various parameters were as follows: The Pulse Rate (PR) was 80.0 ± 11.4 in the Asthma and COPD group was 94.0 ± 8.4 , showing no significant difference between them.

The respiratory rate (RR) was significantly higher in the COPD group (26.2 ± 2.30) compared to the Asthma group (21.2 ± 2.20), with a p-value of <0.001, indicating a statistically significant difference. Pulse Oximetry at room air showed no significant difference between the two groups, with the asthma group at 97.1 ± 1.92 and the COPD group at 93.4 ± 4.41 (p-value = 0.52). The data indicate that the variance in respiratory rate is significant, with a p-value of <0.05, highlighting a notable difference between the groups in this parameter. Other vitals, including pulse oximetry did not show significant differences between the asthma and COPD groups.

In the laboratory parameters of both the asthma and COPD groups, several tests were analyzed to compare the findings. The random blood sugar (RBS) levels were also similar, with the asthma group at 127 ± 25.1 and the COPD group at 131 ± 29.3 (p-value = 0.55) showing no significant difference.

Complete blood count (CBC) parameters, hemoglobin (Hb) levels were similar between the two groups, with the asthma group at 14.1 ± 2.46 and the COPD group at 13.9 ± 2.40 , showing no significant difference (p-value = 0.81). Total leukocyte count (TLC) was slightly higher in the asthma group (7.66 ± 2.06) compared to the COPD group (6.91 ± 2.06), but this difference was not statistically significant (p-value = 0.14).

Regarding the liver function test, total bilirubin levels showed no significant difference, with the asthma group at 0.73 ± 0.28 and the COPD group at 0.58 ± 0.28 (p-value = 0.73). Similarly, the Alkaline Phosphate levels were similar between the groups (p-value = 0.89). However, the serum glutamic oxaloacetic transaminase (SGOT) levels showed no statistically significant difference between the two groups, with the asthma group having a mean value of 23.6 ± 9.33 and the COPD group at 24.3 ± 8.96 (p-value = 0.52).

In the renal function test, blood urea levels were not significantly different between the two groups (p-value = 0.89). However, serum creatinine levels showed a marginal difference, with the asthma group at 1.0 ± 0.25 and the COPD group at 1.12 ± 0.25 , yielding a p-value of 0.05 suggesting a borderline significance.

The eosinophil count in sputum was significantly higher in the asthma group (589 ± 86.1) compared to the COPD group (113 ± 14.4), with a p-value of <0.001 , indicating a statistically significant difference. Serum immunoglobulin E (IgE) levels were also significantly higher in the asthma group (346 ± 35.3) than in the COPD group (161.8 ± 7.58), with a p-value of <0.001 .

For the FEV1/FVC ratio, the overall asthma group had a significantly higher ratio compared to the COPD group, with a p-value of <0.001 , reflecting a substantial difference. The spirometry tests before and after bronchodilator administration showed notable differences between the two groups. Pre-Bronchodilator FEV1 (L) was higher in the asthma group (2.44 ± 0.12) compared to the COPD group (1.25 ± 0.241), with a p-value of <0.001 . Similarly, pre-bronchodilator FVC (L) was significantly higher in the asthma group (3.93 ± 0.15) than in the COPD group (2.47 ± 0.29), with a p-value of <0.001 .

Post-Bronchodilator measurements also demonstrated significant differences. Post-Bronchodilator FEV1 (L) was significantly higher in the asthma group (2.96 ± 0.14) compared to the COPD group (1.46 ± 0.29), with a p-value of <0.001 . Similarly, post-Bronchodilator FVC (L) was higher in the Asthma group (4.04 ± 0.16) compared to the COPD group (2.57 ± 0.29), with a p-value of <0.001 . All diagnostic parameters presented significant variances with p-values less than 0.05, particularly for sputum eosinophil count, serum IgE levels, FEV1/FVC ratio, and spirometry measurements, which demonstrate a clear distinction between the asthma and COPD groups.

Table 1. Demographic details	n (%)
Total Patients	66 (100%)
Group	

To assess asthma COPD overlap syndrome among patients of asthma and COPD.

Asthma /COPD	33 (50%) /33 (50%)
Age(mean \pm SD)	53.06 \pm 09.63
Sex	
Female/ Male	36(54.5%) /30 (45.5%)
Occupation Category	
Business/Engineer/ Farmer/ Labourer Teacher	10 (15.2%)/ 8 (12.1%)/ 12 (18.2%) 20 (30.3%)/ 16 (24.2)
Area of Living	
Rural /Semi-Urban/ Urban	12 (18.2%)/ 24 (36.4%)/ 30 (45.5%)
Smoking Status	
Current Smoker Ex-smoker /Non-smoker	17 (25.75%) 25 (37.87%)/ 24 (36.36%)
BMI Category	
Under Weight /Normal Weight/ Over Weight Obesity	25 (37.87%)/ 28 (42.42%)/ 11 (16.66%) 2 (3.03%)
<i>All the data is represented in frequency, n and percent (%) with their mean \pm standard deviation.</i>	

Table 2. General Examination	Asthma(33)	COPD(33)	Total(66)	p-value
Pallor				
Yes	16 (48.4%)	15 (45.4%)	31 (46.9%)	0.4
No	17 (51.5%)	18 (54.5%)	35 (53.0%)	
Icterus				
Yes	15 (45.45%)	18 (54.54%)	33 (50%)	0.2
No	18 (54.54%)	15 (45.45%)	33 (50%)	
Pedal Edema				
Yes	12 (36.3%)	19 (28.7%)	31 (47%)	0.08
No	21 (65.7%)	14 (71.3%)	35 (53%)	
<i>All the data is represented in Frequency, n and Percent (%). The variance is significant with p-value <0.05.</i>				

Table 3. Vitals Mean \pm SD	Asthma	COPD	Total	p-value
PR	80.0 \pm 11.4	94.0 \pm 8.4	87.0 \pm 9.9	<0.001
RR	21.2 \pm 2.20	26.2 \pm 2.30	23.7 \pm 3.37	<0.001
Pulse Oximetry	97.1 \pm 1.92	93.4 \pm 4.41	95.2 \pm 3.16	<0.001
<i>All the data is represented in their Mean \pm Standard Deviation. The variance is significant with p-value <0.05.</i>				

Table 4. Laboratory Parameters	Asthma	COPD	Total	p-value
RBS	127 \pm 25.1	131 \pm 29.3	129 \pm 27.1	0.55
CBC				
HB	14.1 \pm 2.46	13.9 \pm 2.40	14.0 \pm 2.41	0.81

TLC	7.66 ± 2.06	6.91 ± 2.06	7.28 ± 2.08	0.14
DLC	54.3 ± 9.54	54.2 ± 9.27	54.2 ± 9.33	0.99
Polymorphs	59.1 ± 7.78	58.1 ± 9.93	58.6 ± 8.86	0.65
ESR	25.8 ± 9.53	27.4 ± 8.29	26.6 ± 8.90	0.45
Liver Function Test, Mean ± SD				
Total Bilirubin	0.73 ± 0.28	0.58 ± 0.28	0.65 ± 0.29	0.73
SGOT	23.6 ± 9.33	24.3 ± 8.96	24.0 ± 9.09	0.52
Alkaline Phosphate	98.0 ± 32.4	99.0 ± 28.9	98.5 ± 30.5	0.89
Renal Function Test, Mean ± SD				
Blood Urea	28.8 ± 7.21	28.5 ± 7.79	28.7 ± 7.45	0.89
Serum Creatinine	1.0 ± 0.25	1.12 ± 0.25	1.06 ± 0.26	0.05
<i>All the data is represented in their Mean ± Standard Deviation. The variance is significant with p-value <0.05.</i>				

Table 5. Investigations	Asthma	COPD	Total	p-value
Sputum Test				
Sputum Eosinophil Count	589 ± 86.1	113 ± 14.4	351 ± 247	<0.001
Serum IgE	346 ± 35.3	161.8 ± 7.6	204 ± 145	<0.001
Spirometry Pre-Bronchodilator				
Pre-Bronchodilator FEV ₁ (L)	2.44 ± 0.12	1.25 ± 0.241	1.85 ± 0.62	<0.001
Pre-Bronchodilator FVC (L)	3.93 ± 0.15	2.47 ± 0.29	3.20 ± 0.77	<0.001
FEV ₁ /FVC Ratio	0.65 ± 0.02	0.43 ± 0.02	0.54 ± 0.11	<0.001
Spirometry Post-Bronchodilator				
Post-Bronchodilator FEV ₁ (L)	2.96 ± 0.14	1.46 ± 0.29	2.21 ± 0.78	<0.001
Post-Bronchodilator FVC (L)	4.04 ± 0.16	2.57 ± 0.29	3.30 ± 0.77	<0.001
FEV ₁ /FVC Ratio	0.78 ± 0.02	0.41 ± 0.03	0.59 ± 0.05	<0.001
<i>All the data is represented in their Mean ± Standard Deviation. The variance is significant with p-value <0.05.</i>				

DISCUSSION

The present study aimed to compare the demographic, clinical, and laboratory parameters of patients with asthma and COPD, focusing on the overlap syndrome and its clinical significance. The findings provide valuable insights into the similarities and differences between these two groups, highlighting areas of potential clinical relevance.

The demographic distribution of the study participants demonstrated a balanced representation of asthma and COPD patients. The mean age of the participants were 29.0 ± 5.63 years, which is relatively younger compared to other studies focusing on these conditions [25]. This could be attributed to the specific population targeted in this study. Sex distribution revealed a slightly higher proportion of females (54.5%) compared to males (45.5%), consistent with prior reports indicating a higher prevalence of asthma among females due to hormonal and genetic factors [26].

The occupational distribution was varied, with labourers (30.3%) being the most common, followed by teachers (24.2%), farmers (18.2%), businesspersons (15.2%), and engineers (12.1%). This diversity in occupation indicates that the study encompassed individuals from different socioeconomic strata. Such variation provides a broader understanding of the demographic profile of patients with asthma and COPD, which is essential for assessing the overlap syndrome [27].

Area of living revealed an urban predominance (45.5%), followed by semi-urban (36.4%) and rural areas (18.2%). This pattern aligns with the known urban-rural disparity in access to healthcare and the higher prevalence of COPD in urban settings due to increased exposure to pollutants [28].

A significant finding in my study was the distribution of smoking status among participants. Of the total 66 patients, 17 (25.75%) were current smokers, 25 (37.87%) were ex-smokers, and 24 (36.36%) were non-smokers. Within the asthma group, 7 participants (41%) were current smokers, 13 (52%) were ex-smokers, and 13 (53%) were non-smokers. In the COPD group, 10 participants (58.8%) were current smokers, 12 (48%) were ex-smokers, and 11 (47%) were non-smokers. This distribution highlights that a significant portion of patients had a history of smoking, particularly in the COPD group. The presence of a substantial number of non-smokers suggests that factors beyond smoking, such as environmental exposures and genetic predispositions, may also play a role in the development of asthma-COPD overlap syndrome (ACOS) [29]. The BMI distribution showed that a significant proportion of participants were in the normal weight category (42.4%), followed by overweight (39.4%) and obesity (13.6%), underscoring the role of nutritional and lifestyle factors in these diseases [30].

The general examination revealed that pedal edema was more prevalent in COPD patients (28.8%) compared to asthma patients (18.2%), approaching statistical significance (p -value = 0.08). This finding aligns with the path physiology of COPD, which often involves systemic inflammation and associated fluid retention [31]. The presence of pallor, icterus, and other clinical signs did not differ significantly between the groups, reflecting overlapping clinical features [32].

Respiratory Rate (RR) was significantly higher in COPD patients (26.2 ± 2.30) compared to asthma patients (21.2 ± 2.20), with a p -value < 0.001 [33]. Pulse Rate (PR) was significantly higher in COPD patients (94.0 ± 8.4) compared to asthma patients (80.0 ± 11.4) with a p -value 0.048. These findings are consistent with the chronic airway obstruction characteristic of COPD, which results in compensatory tachypnoea & tachycardia. Oxygen saturation (SpO₂) levels showed significant differences, suggesting lower oxygenation status in ACOS-COPD group [34].

The laboratory findings revealed several key insights. Random Blood Sugar (RBS) levels and Complete Blood Count (CBC) parameters, including haemoglobin (Hb), total leukocyte count (TLC), differential leukocyte count (DLC), and erythrocyte sedimentation rate (ESR), were comparable between the two groups. The similarity in these parameters underscores the systemic nature of both asthma and COPD, with overlapping inflammatory pathways [35].

Liver Function Test (LFT), the SGOT levels were marginally higher in COPD patients (24.3 ± 8.96) compared to asthma patients (23.6 ± 9.33), with a p -value of 0.52, with no statistical significance [36]. This may reflect subclinical hepatic stress or inflammation associated with COPD. Renal Function Test (RFT) results showed no significant differences in blood urea

levels; however, serum creatinine levels were borderline significant (p-value = 0.05), with asthma patients exhibiting slightly higher values [37].

The differences observed, particularly in respiratory rate and SGOT levels, emphasize the need for tailored management strategies for each condition [38].

Strength and Limitations

The study's strengths include its balanced representation of asthma and COPD patients and the comprehensive analysis of clinical and laboratory parameters. However, the study has a few minor limitations. It was conducted at a single center, which may limit the diversity of patient backgrounds and environmental factors. Some patient data, such as lifestyle habits and family history, were based on self-reporting, which may introduce slight recall bias. Additionally, while the study followed established diagnostic guidelines, minor variations in clinical practice may affect the consistency of diagnosis. Lastly, although the study used advanced diagnostic tools like spirometry, certain emerging biomarkers were not included, which could provide additional insights in future research. Despite the study's limitations, it lays the groundwork for future research to refine diagnostic criteria and develop targeted interventions for patients with ACOS.

CONCLUSION

The prevalence of ACOS was 20% with higher proportion of females. ACOS in COPD group had higher respiratory rate, pulse rate and low saturation (SpO₂) as compared to asthma group. This indicates ACOS in COPD group has more work of breathing, hypoxemia as compared to asthma group which can lead to more exacerbation. The positive link between the low spirometry variables (pre and post bronchodilator FEV₁, FVC, FEV₁/FVC) in ACOS- COPD group linked to more disease severity as compared to asthma group. So, these patients can more frequently be hospitalised and should be observed more closely at follow-up. So, identification of ACOS patients in COPD may be an important goal for the patient management.

REFERENCES

- [1] Global Initiative for Asthma (GINA). (2023). Global Strategy for Asthma Management and Prevention.
- [2] Global Initiative for Chronic Obstructive Lung Disease (GOLD). (2023). Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease.
- [3] To, T., Stanojevic, S., Moores, G., Gershon, A. S., Bateman, E. D., Cruz, A. A., & Boulet, L. P. (2017). Global asthma prevalence in adults: Findings from the cross-sectional world health survey. *BMC Public Health*, 12(1), 204.
- [4] Gibson, P. G., & McDonald, V. M. (2015). Asthma-COPD overlap 2015: Now we are six. *Thorax*, 70(7), 683-691.
- [5] Hardin, M., Silverman, E. K., Barr, R. G., Hansel, N. N., Schroeder, J. D., Make, B. J., & Crapo, J. D. (2011). The clinical features of the overlap between COPD and asthma. *Respiratory Research*, 12(1), 127.
- [6] Fahy, J. V. (2015). Type 2 inflammation in asthma—present in most, absent in many. *Nature Reviews Immunology*, 15(1), 57-65.
- [7] Pavord, I. D., Beasley, R., Agusti, A., et al. (2018). After asthma: redefining airways diseases. *The Lancet*, 391(10118), 350-400.
- [8] Barnes, P. J. (2016). Inflammatory mechanisms in patients with chronic obstructive pulmonary disease. *Journal of Allergy and Clinical Immunology*, 138(1), 16-27.
- [9] Rabe, K. F., Watz, H. (2017). Chronic obstructive pulmonary disease. *The Lancet*, 389(10082), 1931-1940.
- [10] Postma, D. S., & Rabe, K. F. (2015). The asthma-COPD overlap syndrome. *New England Journal of Medicine*, 373(13), 1241-1249.
- [11] Miravittles, M., Soriano, J. B., Ancochea, J., et al. (2013). Characteristics of the overlap COPD-asthma phenotype: focus on physical activity and health status. *Respiratory Medicine*, 107(7), 1053-1060.
- [12] Soriano, J. B., Ancochea, J., Miravittles, M., et al. (2012). Recent trends in COPD prevalence in Spain: A repeated cross-sectional survey 1997-2007. *European Respiratory Journal*, 39(4), 807-814.

- [13] Lee, J. H., Kim, E. K., Lee, J. H., et al. (2015). Prevalence and impact of asthma-COPD overlap syndrome in Korea—a nationwide population-based study. *Asia Pacific Allergy*, 5(4), 210-215.
- [14] Papi, A., Bellettato, C. M., Braccioni, F., et al. (2018). Infections and airway inflammation in chronic obstructive pulmonary disease severe exacerbations. *American Journal of Respiratory and Critical Care Medicine*, 173(10), 1114-1121.
- [15] Kauppi, P., Kupiainen, H., Lindqvist, A., et al. (2011). Overlap syndrome of asthma and COPD predicts low quality of life. *Journal of Asthma*, 48(3), 279-285.
- [16] Christenson, S. A., Smith, B. M., Bafadhel, M., et al. (2015). Asthma-COPD overlap: clinical relevance of genomic signatures of type 2 inflammation in chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*, 191(7), 758-766.
- [17] Han, M. K., Agusti, A., Calverley, P. M., et al. (2020). Chronic obstructive pulmonary disease phenotypes: the future of COPD. *American Journal of Respiratory and Critical Care Medicine*, 195(2), 139-144.
- [18] Kaplan, A., Thomas, M., & Goldstein, M. (2017). Triple therapy in the management of COPD: where does it fit? *American Journal of Respiratory and Critical Care Medicine*, 196(1), 25-37.
- [19] Roca, M., Hernández, C., & Roig, M. (2015). Pulmonary rehabilitation in chronic obstructive pulmonary disease. *Current Opinion in Pulmonary Medicine*, 21(1), 86-92.
- [20] Watz, H., Pitta, F., Rochester, C. L., et al. (2014). An official European Respiratory Society statement on physical activity in COPD. *European Respiratory Journal*, 44(6), 1521-1537.
- [21] Tashkin, D. P., & Murray, R. P. (2009). Smoking cessation in chronic obstructive pulmonary disease. *Respiratory Medicine*, 103(6), 963-974.
- [22] Vestbo, J., Hurd, S. S., Agustí, A. G., et al. (2013). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *American Journal of Respiratory and Critical Care Medicine*, 187(4), 347-365.
- [23] Wechsler, M. E., Laviolette, M., Rubin, A. S., et al. (2013). Bronchial thermoplasty: Long-term safety and effectiveness in patients with severe persistent asthma. *Journal of Allergy and Clinical Immunology*, 132(6), 1295-1302.
- [24] McCarthy, J. R., Miller, M. R., & Hurst, J. R. (2021). The impact of asthma-COPD overlaps on health-related quality of life. *BMC Pulmonary Medicine*, 21(1), 24.
- [25] **Global Initiative for Asthma (GINA) 2023 Report.**
- [26] Vink NM, et al. "Gender differences in asthma prevalence: A review of studies." *Allergy*, 2018.
- [27] Gupta N, et al. "Socioeconomic factors influencing asthma management." *Journal of Asthma*, 2019.
- [28] Thakur JS, et al. "Urban versus rural health disparities in chronic diseases." *Indian Journal of Public Health*, 2020.
- [29] Centers for Disease Control and Prevention (CDC). "Impact of smoking on respiratory health." *CDC Reports*, 2021.
- [30] Ng M, et al. "Global trends in BMI and obesity-related respiratory diseases." *Lancet*, 2019.
- [31] Vogelmeier CF, et al. "Global strategy for the diagnosis, management, and prevention of COPD." *GOLD 2022 Report*.
- [32] **Barnes PJ. "Inflammatory mechanisms in COPD." *European Respiratory Journal*, 2021.**
- [33] **Mahler DA, et al. "Respiratory rate and clinical outcomes in COPD." *Chest*, 2020.**

- [34] Ferguson GT, et al. "Oxygenation strategies in COPD patients." *Pulmonology*, 2019.
- [35] Rabe KF, et al. "Overlap syndrome: Asthma and COPD combined." *American Journal of Respiratory Medicine*, 2022.
- [36] **Gibson PG, et al. "Liver dysfunction in chronic airway diseases." *Respiratory Research*, 2020.**
- [37] Minai OA, et al. "Renal function and chronic pulmonary conditions." *Kidney International Reports*, 2019.
- [38] Lipworth BJ, et al. "Management of overlap syndrome in clinical practice." *Thorax*, 2020.