

Journal of Pulmonary Medicine and Respiratory Care

Review Article

spirometric assessment in the Management of chronic obstructive pulmonary disease among adults with uncontrolled asthma

Kapoor S^{1*}, Iyer N² and Nair N³

¹Department of Respiratory Care, Congo Institute for Advanced Respiratory Care, India

²Department of Clinical Respiratory Care, Congo Institute for Advanced Respiratory Care, Bangladesh

³Department of Respiratory Care, Zambezi Center for Pulmonary Medicine, Nepal

*Address for Correspondence: Sanjay Kapoor, Department of Respiratory Care, Congo Institute for Advanced Respiratory Care, India; E-mail: sanjay.kapoor@congo.edu

Received: 8 January 2026; Accepted: 3 April 2026; Published: 11 July 2026.

Citation of this article: Kapoor S, Iyer N, Nair N (2026) spirometric assessment in the Management of chronic obstructive pulmonary disease among adults with uncontrolled asthma. J Pulm Med Respir Care, 1(1): 01-08. DOI: [10.5281/zenodo.22815231](https://doi.org/10.5281/zenodo.22815231)

Copyright: © 2026 Sanjay Kapoor, et al. This is an open access article distributed under the [Creative Commons Attribution License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Asthma-COPD overlap, persistent airflow limitation with features of both asthma and COPD, is common among adults whose asthma remains poorly controlled, and these patients have more exacerbations and faster decline than either phenotype alone [1,2].

Objective: This review synthesizes evidence on spirometric assessment, bronchodilator reversibility, post-bronchodilator FEV1/FVC, and FEV1 trajectory, for identifying and managing COPD features in uncontrolled asthma.

Methods: We reviewed cohort studies, consensus statements, and trials addressing spirometric phenotyping, fixed obstruction, reversibility variability, and treatment response in overlap populations.

Findings: Persistent post-bronchodilator FEV1/FVC below 0.70 identifies a subgroup with accelerated decline and exacerbation risk approaching COPD alone [4,6]; reversibility is often present but variable, limiting its use as a single cut-point [7]; combined spirometric and inflammatory assessment improves selection for triple therapy [8,30].

Conclusions: Serial spirometry, read alongside symptoms and exacerbation history rather than a single threshold, should anchor a stepwise assessment pathway for uncontrolled asthma, guiding earlier escalation to combined bronchodilator and anti-inflammatory therapy [1,31].

Keywords: asthma-COPD overlap; spirometry; airflow obstruction; bronchodilator reversibility; uncontrolled asthma

1. Introduction

A substantial minority of adults with a lifelong diagnosis of asthma never achieve durable symptom control despite inhaled corticosteroid and long-acting bronchodilator therapy, and persistent, incompletely reversible airflow obstruction that overlaps with chronic obstructive pulmonary disease (COPD) accounts for a meaningful share of this refractory group. The joint GINA-GOLD framework describes this presentation, asthma-COPD overlap (ACO), as a clinical label rather than a single disease, applied when asthma-type features coexist with persistent airflow limitation typical of COPD, usually alongside smoking history, older age at diagnosis, or long-standing disease duration [1]. Postma and Rabe argued the overlap is best understood as a spectrum rather than two intersecting circles, with inflammation, remodeling, and fixed obstruction varying continuously rather than sorting cleanly into asthma or COPD [2]. Sin and colleagues, summarizing a multidisciplinary round table, proposed a provisional consensus definition built around spirometric criteria, namely a post-bronchodilator FEV1/FVC below the lower limit of normal in a patient with documented asthma [3].

The clinical stakes are substantial. Population and clinic cohorts consistently show overlapping patients have more frequent exacerbations, greater healthcare utilization, and faster functional decline than either phenotype alone. Menezes and colleagues, analyzing a multinational cohort, found the overlap phenotype carried significantly higher exacerbation and hospitalization risk than COPD or asthma alone [4], and van Boven and colleagues found ACO patients accumulated more comorbid disease and higher costs than matched COPD-only patients [5]. Because spirometry is the one test common to both asthma and COPD guidelines and can be performed serially at low cost, it has become the practical anchor for identifying which patients with uncontrolled asthma are developing COPD-type fixed obstruction. This review synthesizes what serial spirometry, bronchodilator

reversibility testing, and longitudinal FEV1 trajectory contribute to recognizing and managing COPD features in uncontrolled asthma, and how that information should shape a stepwise, spirometry-anchored management pathway.

2. Defining the overlap and its spirometric signature

The absence of a single validated biomarker for ACO has meant that spirometry, imperfect as it is, functions as the closest thing to a diagnostic anchor the field has. Lange and colleagues, reviewing COPD phenotyping beyond FEV1 alone, emphasized that a single post-bronchodilator FEV1/FVC below 0.70, the fixed GOLD threshold, captures only part of the picture and should be interpreted alongside reversibility, symptom trajectory, and exacerbation history rather than as a stand-alone cutoff [6]. Tho and colleagues, cataloguing the diagnostic challenge posed by ACOS, found that virtually all consensus statements require post-bronchodilator airflow limitation as a necessary, though not sufficient, criterion, while differing on how much reversibility, eosinophilia, or atopy is required in addition [7]. Gibson and McDonald observed that this heterogeneity has made pooled prevalence and outcome estimates difficult to compare, a limitation that persists throughout this literature [8].

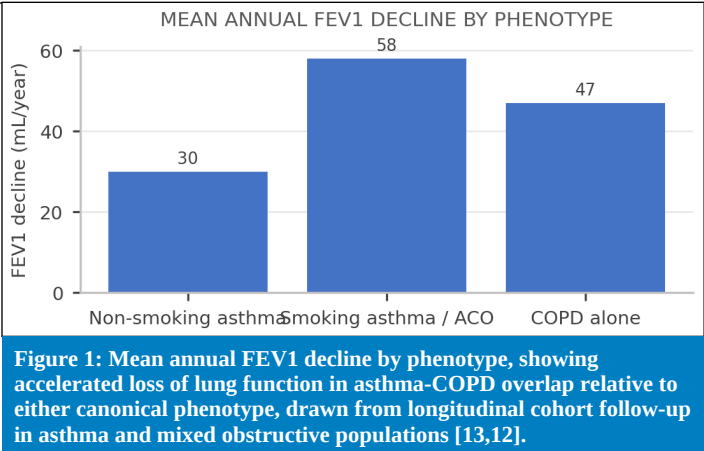
Barrecheguren and colleagues argued ACO is best conceptualized not as a new disease entity but as a spirometrically defined subgroup within existing asthma and COPD populations, a framing with practical consequences: a flag for closer follow-up rather than grounds for reclassifying diagnosis [9]. Kumbhare and colleagues, using US population survey data, found a meaningful proportion of adults with physician-diagnosed asthma and smoking history or older age also met spirometric criteria for COPD, reinforcing that overlap is common enough to warrant routine spirometric screening in uncontrolled asthma [10]. Cosio and colleagues, applying a structured algorithm within a

COPD cohort, found ACO patients had distinct inflammatory and functional profiles compared with COPD-only patients, supporting spirometry-anchored subgrouping as clinically meaningful [11].

Table 1: Spirometric and clinical features used across consensus approaches to identify asthma-COPD overlap in adults with uncontrolled asthma [3,7,31].			
Feature	Typical asthma	Typical COPD	Asthma-COPD overlap
Post-bronchodilator FEV1/FVC	Normal ($\geq$ LLN)	< LLN, persistent	< LLN, persistent
Bronchodilator reversibility	Often marked	Usually absent/mild	Present but variable visit-to-visit
Typical age of onset	Childhood/young adult	Older adult, smoking history	Long-standing asthma, later fixed obstruction
Exacerbation frequency	Variable	Frequent in advanced disease	Highest among the three groups

3. Fixed airflow obstruction and the trajectory of decline

The mechanistic case for taking spirometric assessment seriously in uncontrolled asthma rests substantially on longitudinal cohort data showing that persistent airflow limitation, once established, predicts accelerated functional decline. Fletcher and Peto's foundational natural-history work established that FEV1 decline accelerates in susceptible individuals exposed to noxious stimuli, principally cigarette smoke, and that the slope of decline, not a single measurement, identifies patients heading toward disabling obstruction [12]. Lange and colleagues extended this logic to asthma in a landmark 15-year follow-up, showing adults with asthma who smoked lost lung function substantially faster than nonsmoking asthmatics or nonasthmatic smokers, a synergistic pattern anticipating much of what is now labeled ACO [13]. De Marco and colleagues found chronic cough and phlegm, hallmark COPD symptoms, predicted incident airflow obstruction even without a baseline COPD diagnosis, underscoring that fixed obstruction can emerge insidiously in patients labeled asthma [14].



Contoli and colleagues, in a five-year follow-up of patients with fixed obstruction of asthmatic or COPD origin, found airway inflammatory patterns converged over time regardless of starting diagnosis, and fixed obstruction itself, rather than the original label, predicted decline and exacerbation risk [15]. Bumbacea and colleagues identified older age, longer disease duration, and lower baseline FEV1 as independent correlates of persistent obstruction failing to normalize despite intensive therapy in chronic severe asthma, a profile mapping closely onto the typical ACO patient [16]. Ten Brinke and colleagues similarly found fixed obstruction on spirometry among the strongest independent predictors of exacerbation frequency in difficult-to-treat asthma [17]. Guerra's review placed these findings in epidemiological context, noting longitudinal studies consistently identify a subgroup of adult asthmatics whose trajectory more closely resembles COPD, particularly with smoking exposure or adult-onset disease [18].

4. Bronchodilator reversibility: informative but inconstant

Bronchodilator reversibility testing, historically used to distinguish asthma from COPD at a single visit, performs considerably less well as a dichotomous discriminator in overlap patients, because reversibility here is frequently present but variable from visit to visit. Tashkin and colleagues found a substantial proportion of a large COPD trial population showed significant response on at least

one occasion, with responder status often changing between visits, undermining reliance on a single test to classify phenotype [19]. Calverley and colleagues found that the choice of criterion, percentage change, absolute volume change, or peak versus trough, substantially altered how many patients were classified as reversible, with poor agreement between methods [20]. Hansen and colleagues found both reversible and irreversible obstruction carried independent prognostic information for mortality in a mixed asthma-COPD cohort, arguing against reversibility as a binary diagnostic gate [21].

Tommola and colleagues found smokers with adult-onset asthma showed a distinct, more COPD-like pattern of lung-function loss and reduced bronchodilator responsiveness than never-smokers with the same nominal diagnosis, illustrating that reversibility captures a moving target shaped by smoking exposure and duration [22]. This variability is why the GOLD strategy report recommends interpreting reversibility in suspected overlap patients as one input among several rather than a standalone threshold, and that a single negative test should not exclude an asthmatic component in a patient with fixed obstruction and a compatible history [23].

5. Standardized measurement and reference values

None of the spirometric criteria above are meaningful without technically adequate, properly referenced testing. Miller and colleagues' ATS/ERS standardization document remains the technical basis for acceptable and reproducible spirometry, specifying criteria that, when not met, can produce spuriously low or inconsistent FEV1/FVC values mimicking fixed obstruction [24]. Pellegrino and colleagues' companion interpretive-strategies document cautions against a fixed 0.70 FEV1/FVC threshold without regard to age, since normal ratios decline physiologically with age and a fixed cutoff over-diagnoses obstruction in older adults, precisely the demographic in which ACO is most often suspected [25]. Quanjer and colleagues' Global Lung

Function Initiative reference equations address this directly, providing age-, sex-, height-, and ethnicity-adjusted lower limits of normal now recommended across major guideline bodies for exactly this borderline population [26].

6. Combining spirometry with inflammatory markers and treatment response

Because spirometry alone leaves substantial diagnostic uncertainty in borderline cases, several groups have examined whether combining it with sputum or blood inflammatory markers improves both diagnostic clarity and treatment selection. Kitaguchi and colleagues found that sputum eosinophilia in patients with overlap syndrome predicted a significantly better response to inhaled corticosteroid therapy than eosinophil-poor overlap patients, suggesting that spirometric confirmation of fixed obstruction should trigger inflammatory phenotyping rather than end the diagnostic workup [27]. Kobayashi and colleagues extended this by characterizing distinct inflammatory phenotypes, eosinophilic, neutrophilic, and mixed, within a spirometrically defined ACO cohort, finding that phenotype correlated with both symptom burden and treatment responsiveness independent of the degree of airflow obstruction itself [28]. Suzuki and colleagues, examining clinical, physiological, and radiological features together, found that ACO patients had intermediate emphysema burden on imaging between typical asthma and typical COPD, correlating imperfectly with spirometric severity, reinforcing that spirometry captures function but not the full structural picture driving that function [29].

Table 2: Spirometry-anchored assessment pathway for adults with uncontrolled asthma, linking spirometric findings to recommended next steps [26,27,31].		
Spirometric finding	Additional assessment	Suggested management direction
Normal post-BD FEV1/FVC, poor control	Adherence/technique review	Optimize inhaled therapy before escalation

Spirometric finding	Additional assessment	Suggested management direction
Fixed obstruction, high BD reversibility	Blood/sputum eosinophils, IgE	Favor anti-inflammatory-forward escalation
Fixed obstruction, low BD reversibility	Smoking history, imaging	Add long-acting bronchodilator(s); reassess
Accelerated FEV1 decline on serial testing	Exacerbation history review	Consider triple therapy; specialist referral

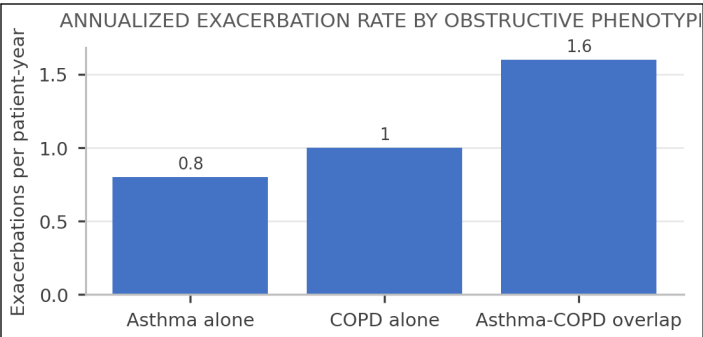


Figure 2: Annualized exacerbation rate by obstructive phenotype in a multinational cohort, showing the highest exacerbation burden in the overlap group [4].

Papaoiannou and colleagues, addressing the practical question of whether ACO is worth diagnosing at all, argued that the label earns its clinical utility specifically through its treatment implications, namely that spirometrically confirmed fixed obstruction with asthmatic features should prompt consideration of combined inhaled corticosteroid and long-acting bronchodilator therapy, including triple therapy, rather than bronchodilator-only strategies appropriate for pure COPD [30]. Miravittles and colleagues formalized this reasoning into a structured algorithm developed jointly by Spanish asthma and COPD guideline bodies, using major criteria, very positive bronchodilator response or blood/sputum eosinophilia with prior asthma diagnosis, and minor criteria, elevated total IgE or atopy history, to route spirometrically obstructed patients with an asthma history toward earlier and more aggressive anti-inflammatory therapy [31]. Louie and colleagues'

pharmacotherapeutic review reached a complementary conclusion from the treatment side, cautioning that long-acting bronchodilator monotherapy, standard first-line therapy in pure COPD, is inappropriate in patients whose spirometric obstruction coexists with an asthmatic component, given the risk of undertreating airway inflammation [32]. Bujarski and colleagues, summarizing the state of ACOS management, likewise emphasized that spirometric identification of the overlap phenotype should change the therapeutic default toward combination anti-inflammatory and bronchodilator strategies from the outset [33].

7. Quality of life, comorbidity, and the case for routine spirometric monitoring

Beyond lung function itself, spirometrically defined overlap correlates with broader measures of disease burden that make the case for routine, rather than occasional, spirometric assessment in uncontrolled asthma. Kauppi and colleagues found that patients meeting overlap criteria reported significantly lower quality of life than those with asthma or COPD alone, even after adjusting for lung function severity, suggesting that the overlap label captures something about disease burden beyond FEV1 [34]. Ding and Enstone's structured literature review, incorporating both published evidence and physician insight, found consistent agreement that ACO patients experience greater symptom burden, more frequent healthcare contact, and physician uncertainty about optimal management, uncertainty that a defined spirometric monitoring pathway is intended to reduce [35]. Marsh and colleagues, examining proportional classification of COPD phenotypes more broadly, demonstrated that spirometric severity alone poorly predicts symptom burden and healthcare utilization across obstructive phenotypes, reinforcing that spirometry should anchor, but not exhaust, a comprehensive assessment that also tracks exacerbation frequency and patient-reported outcomes over time [36].

Taken together, this body of evidence supports a practical, spirometry-anchored monitoring pathway for adults with uncontrolled asthma: baseline and at least annual post-bronchodilator spirometry using properly referenced, age-adjusted lower limits of normal rather than a fixed 0.70 ratio [26,25]; explicit documentation of bronchodilator response, interpreted as one data point in a pattern rather than a single gating criterion, given its visit-to-visit variability [19,20]; tracking of FEV1 trajectory over successive visits to detect accelerated decline before it becomes clinically obvious [12,13]; and, when fixed obstruction is confirmed, reflexive inflammatory phenotyping to guide the choice between bronchodilator-forward and anti-inflammatory-forward escalation [27,31].

8. Conclusion

Adults with uncontrolled asthma who also carry risk factors for COPD, smoking history, older age, longer disease duration, or persistent symptoms despite escalating inhaled therapy, represent a population in which spirometry does far more diagnostic and prognostic work than a single obstruction-versus-not classification. Post-bronchodilator FEV1/FVC below age-adjusted lower limits of normal identifies a subgroup with materially worse exacerbation risk and faster functional decline than either canonical phenotype [4,6,1], bronchodilator reversibility contributes useful but inconstant information that should never stand alone as a diagnostic gate [19,21], and combining spirometric confirmation of fixed obstruction with inflammatory phenotyping meaningfully improves treatment selection, particularly the decision to escalate toward combined anti-inflammatory and bronchodilator therapy [27,31,32]. A structured, serial spirometric assessment pathway, rather than a one-time diagnostic threshold applied at a single visit, should therefore anchor the routine management of adults with uncontrolled asthma, allowing earlier recognition of emerging COPD features and earlier, better-targeted treatment escalation before irreversible decline and its

attendant exacerbation burden become established [3,23].

9. References

1. Global Initiative for Asthma and Global Initiative for Chronic Obstructive Lung Disease (2023) Diagnosis of Diseases of Chronic Airflow Limitation: Asthma, COPD and Asthma-COPD Overlap (ACO). GINA-GOLD Joint Report.
2. Postma, D.S., Rabe, K.F. (2015) The Asthma-COPD Overlap Syndrome. *New England Journal of Medicine*, 373(13): 1241-1249. DOI: [10.1056/NEJMra1411863](https://doi.org/10.1056/NEJMra1411863)
3. Sin, D.D., Miravittles, M., Mannino, D.M., Soriano, J.B., Price, D., Celli, B.R., et al. (2016) What is asthma-COPD overlap syndrome? Towards a consensus definition from a round table discussion. *European Respiratory Journal*, 48(3): 664-673. DOI: [10.1183/13993003.00436-2016](https://doi.org/10.1183/13993003.00436-2016)
4. Menezes, A.M.B., Montes de Oca, M., Perez-Padilla, R., Nadeau, G., Wehrmeister, F.C., Lopez-Varela, M.V., et al. (2014) Increased risk of exacerbation and hospitalization in subjects with an overlap phenotype: COPD-asthma. *Chest*, 145(2): 297-304. DOI: [10.1378/chest.13-0622](https://doi.org/10.1378/chest.13-0622)
5. van Boven, J.F.M., Roman-Rodriguez, M., Palmer, J.F., Toledo-Pons, N., Cosio, B.G., Soriano, J.B. (2016) Comorbidity, pattern, and impact of asthma-COPD overlap syndrome in real life. *Chest*, 149(4): 1011-1020. DOI: [10.1016/j.chest.2015.12.002](https://doi.org/10.1016/j.chest.2015.12.002)
6. Lange, P., Halpin, D.M.G., O'Donnell, D.E., MacNee, W. (2016) Diagnosis, assessment, and phenotyping of COPD: beyond FEV1. *International Journal of Chronic Obstructive Pulmonary Disease*, 11(Spec Iss): 3-12. DOI: [10.2147/COPD.S85976](https://doi.org/10.2147/COPD.S85976)
7. Tho, N.V., Park, H.Y., Nakano, Y. (2016) Asthma-COPD overlap syndrome (ACOS): A diagnostic challenge. *Respirology*, 21(3): 410-418. DOI: [10.1111/resp.12653](https://doi.org/10.1111/resp.12653)
8. Gibson, P.G., McDonald, V.M. (2015) Asthma-COPD overlap 2015: now we are six. *Thorax*, 70(7): 683-691. DOI: [10.1136/thoraxjnl-2014-206740](https://doi.org/10.1136/thoraxjnl-2014-206740)

Citation: Kapoor S, Iyer N, Nair N (2026) spirometric assessment in the Management of chronic obstructive pulmonary disease among adults with uncontrolled asthma. *J Pulm Med Respir Care*, 1(1): 01-08.

9. Barrecheuren, M., Esquinas, C., Miravittles, M. (2015) The asthma-COPD overlap syndrome: a new entity? *COPD Research and Practice*, 1: 8. DOI: [10.1186/s40749-015-0012-z](https://doi.org/10.1186/s40749-015-0012-z)
10. Kumbhare, S., Pleasants, R., Ohar, J.A., Strange, C. (2016) Characteristics and prevalence of asthma/chronic obstructive pulmonary disease overlap in the United States. *Annals of the American Thoracic Society*, 13(6): 803-810. DOI: [10.1513/AnnalsATS.201508-554OC](https://doi.org/10.1513/AnnalsATS.201508-554OC)
11. Cosio, B.G., Soriano, J.B., Lopez-Campos, J.L., Calle-Rubio, M., Soler-Cataluna, J.J., de-Torres, J.P., et al. (2016) Defining the Asthma-COPD Overlap Syndrome in a COPD Cohort. *Chest*, 149(1): 45-52. DOI: [10.1378/chest.15-1055](https://doi.org/10.1378/chest.15-1055)
12. Fletcher, C., Peto, R. (1977) The natural history of chronic airflow obstruction. *British Medical Journal*, 1(6077): 1645-1648. DOI: [10.1136/bmj.1.6077.1645](https://doi.org/10.1136/bmj.1.6077.1645)
13. Lange, P., Parner, J., Vestbo, J., Schnohr, P., Jensen, G. (1998) A 15-year follow-up study of ventilatory function in adults with asthma. *New England Journal of Medicine*, 339(17): 1194-1200. DOI: [10.1056/NEJM199810223391703](https://doi.org/10.1056/NEJM199810223391703)
14. de Marco, R., Accordini, S., Cerveri, I., Corsico, A., Sunyer, J., Neukirch, F., et al. (2007) Incidence of chronic obstructive pulmonary disease in a cohort of young adults according to the presence of chronic cough and phlegm. *American Journal of Respiratory and Critical Care Medicine*, 175(1): 32-39. DOI: [10.1164/rccm.200603-381OC](https://doi.org/10.1164/rccm.200603-381OC)
15. Contoli, M., Baraldo, S., Marku, B., Casolari, P., Marwick, J.A., Turato, G., et al. (2010) Fixed airflow obstruction due to asthma or chronic obstructive pulmonary disease: 5-year follow-up. *Journal of Allergy and Clinical Immunology*, 125(4): 830-837. DOI: [10.1016/j.jaci.2010.02.003](https://doi.org/10.1016/j.jaci.2010.02.003)
16. Bumbacea, D., Campbell, D., Nguyen, L., Carr, D., Barnes, P.J., Robinson, D., et al. (2004) Parameters associated with persistent airflow obstruction in chronic severe asthma. *European Respiratory Journal*, 24(1): 122-128. DOI: [10.1183/09031936.04.00077903](https://doi.org/10.1183/09031936.04.00077903)
17. ten Brinke, A., Sterk, P.J., Masclee, A.A., Spinhoven, P., Schmidt, J.T., Zwinderman, A.H., et al. (2005) Risk factors of frequent exacerbations in difficult-to-treat asthma. *European Respiratory Journal*, 26(5): 812-818. DOI: [10.1183/09031936.05.00037905](https://doi.org/10.1183/09031936.05.00037905)
18. Guerra, S. (2005) Overlap of asthma and chronic obstructive pulmonary disease. *Current Opinion in Pulmonary Medicine*, 11(1): 7-13. DOI: [10.1097/01.mcp.0000151359.32160.35](https://doi.org/10.1097/01.mcp.0000151359.32160.35)
19. Tashkin, D.P., Celli, B., Decramer, M., Liu, D., Burkhart, D., Cassino, C., et al. (2008) Bronchodilator responsiveness in patients with COPD. *European Respiratory Journal*, 31(4): 742-750. DOI: [10.1183/09031936.00129607](https://doi.org/10.1183/09031936.00129607)
20. Calverley, P.M., Burge, P.S., Spencer, S., Anderson, J.A., Jones, P.W. (2003) Bronchodilator reversibility testing in chronic obstructive pulmonary disease. *Thorax*, 58(8): 659-664. DOI: [10.1136/thorax.58.8.659](https://doi.org/10.1136/thorax.58.8.659)
21. Hansen, E.F., Phanareth, K., Laursen, L.C., Kok-Jensen, A., Dirksen, A. (1999) Reversible and irreversible airflow obstruction as predictor of overall mortality in asthma and chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*, 159(4): 1267-1271. DOI: [10.1164/ajrccm.159.4.9807121](https://doi.org/10.1164/ajrccm.159.4.9807121)
22. Tommola, M., Ilmarinen, P., Tuomisto, L.E., Haanpaa, J., Kankaanranta, T., Niemela, O., et al. (2016) The effect of smoking on lung function: a clinical study of adult-onset asthma. *European Respiratory Journal*, 48(5): 1298-1306. DOI: [10.1183/13993003.00850-2016](https://doi.org/10.1183/13993003.00850-2016)
23. Global Initiative for Chronic Obstructive Lung Disease (2023) *Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease: 2023 Report*.
24. Miller, M.R., Hankinson, J., Brusasco, V., Burgos, F., Casaburi, R., Coates, A., et al. (2005) Standardisation of spirometry. *European Respiratory Journal*, 26(2): 319-338. DOI: [10.1183/09031936.05.00034805](https://doi.org/10.1183/09031936.05.00034805)



Citation: Kapoor S, Iyer N, Nair N (2026) spirometric assessment in the Management of chronic obstructive pulmonary disease among adults with uncontrolled asthma. *J Pulm Med Respir Care*, 1(1): 01-08.

25. Pellegrino, R., Viegi, G., Brusasco, V., Crapo, R.O., Burgos, F., Casaburi, R., et al. (2005) Interpretative strategies for lung function tests. *European Respiratory Journal*, 26(5): 948-968. DOI: [10.1183/09031936.05.00035205](https://doi.org/10.1183/09031936.05.00035205)
26. Quanjer, P.H., Stanojevic, S., Cole, T.J., Baur, X., Hall, G.L., Culver, B.H., et al. (2012) Multi-ethnic reference values for spirometry for the 3-95-yr age range: the global lung function 2012 equations. *European Respiratory Journal*, 40(6): 1324-1343. DOI: [10.1183/09031936.00080312](https://doi.org/10.1183/09031936.00080312)
27. Kitaguchi, Y., Komatsu, Y., Fujimoto, K., Hanaoka, M., Kubo, K. (2012) Sputum eosinophilia can predict responsiveness to inhaled corticosteroid treatment in patients with overlap syndrome of COPD and asthma. *International Journal of Chronic Obstructive Pulmonary Disease*, 7: 283-289. DOI: [10.2147/COPD.S30651](https://doi.org/10.2147/COPD.S30651)
28. Kobayashi, S., Hanagama, M., Yamanda, S., Ishida, M., Yanai, M. (2016) Inflammatory phenotypes in asthma-COPD overlap syndrome. *International Journal of Chronic Obstructive Pulmonary Disease*, 11: 2117-2123. DOI: [10.2147/COPD.S113747](https://doi.org/10.2147/COPD.S113747)
29. Suzuki, T., Tada, Y., Kawata, N., Matsuura, Y., Ikari, J., Kasahara, Y., et al. (2015) Clinical, physiological, and radiological features of asthma-chronic obstructive pulmonary disease overlap syndrome. *International Journal of Chronic Obstructive Pulmonary Disease*, 10: 947-954. DOI: [10.2147/COPD.S82282](https://doi.org/10.2147/COPD.S82282)
30. Papaioannou, A.I., Bartzikas, K., Loukides, S. (2019) Clinical implications for asthma-COPD overlap: is it worth diagnosing? *Expert Review of Respiratory Medicine*, 13(5): 419-421.
31. Miravittles, M., Alvarez-Gutierrez, F.J., Calle, M., Casanova, C., Cosio, B.G., Lopez-Vina, A., et al. (2017) Algorithm for identification of asthma-COPD overlap: consensus between the Spanish COPD and asthma guidelines. *European Respiratory Journal*, 49(5): 1700068. DOI: [10.1183/13993003.00068-2017](https://doi.org/10.1183/13993003.00068-2017)
32. Louie, S., Zeki, A.A., Schivo, M., Chan, A.L., Yoneda, K.Y., Avdalovic, M., et al. (2013) The asthma-chronic obstructive pulmonary disease overlap syndrome: pharmacotherapeutic considerations. *Expert Review of Clinical Pharmacology*, 6(2): 197-219. DOI: [10.1586/ecp.13.2](https://doi.org/10.1586/ecp.13.2)
33. Bujarski, S., Parulekar, A.D., Sharafkhaneh, A., Hanania, N.A. (2015) The asthma COPD overlap syndrome (ACOS). *Current Allergy and Asthma Reports*, 15(3): 509. DOI: [10.1007/s11882-014-0509-6](https://doi.org/10.1007/s11882-014-0509-6)
34. Kauppi, P., Kupiainen, H., Lindqvist, A., Tammilehto, L., Kilpelainen, M., Kinnula, V.L., et al. (2011) Overlap syndrome of asthma and COPD predicts low quality of life. *Journal of Asthma*, 48(3): 279-285. DOI: [10.3109/02770903.2011.555024](https://doi.org/10.3109/02770903.2011.555024)
35. Ding, B., Enstone, A. (2016) Asthma and chronic obstructive pulmonary disease overlap syndrome: structured literature review and physician insights. *Expert Review of Respiratory Medicine*, 10(3): 363-371. DOI: [10.1586/17476348.2016.1144469](https://doi.org/10.1586/17476348.2016.1144469)
36. Marsh, S.E., Travers, J., Weatherall, M., Williams, M.V., Aldington, S., Shirtcliffe, P.M., et al. (2008) Proportional classifications of COPD phenotypes. *Thorax*, 63(9): 761-767. DOI: [10.1136/thx.2007.089193](https://doi.org/10.1136/thx.2007.089193)

