



Editorial

Moving Towards a New Focus on COPD. The Spanish COPD Guidelines (GESEPOC)[☆]

Hacia un nuevo enfoque en el tratamiento de la EPOC. La Guía Española de la EPOC (GESEPOC)

GESEPOC Workgroup

October 6, 2009 was an important day in the fight against chronic obstructive pulmonary disease (COPD). On this day, the Spanish National Health-care System (*Sistema Nacional de Salud* – SNS) COPD strategy was presented, which was later approved on June 3, 2009 by the SNS Interterritorial Committee.¹ All the scientific societies, patient associations, and central and autonomic administrations involved formed an alliance in order to promote COPD care and research. Some things have changed since then, for instance we now have a new anti-tobacco law and COPD plans of action in several autonomous Spanish communities. Another of the consequences of the strategy was the drive to develop an interdisciplinary clinical guide for COPD treatment, in which the members of the Strategy participated. SEPAR accepted the challenge and took the initiative to bring together the different societies in order to develop the Spanish COPD Guidelines (GESEPOC).

GESEPOC has 3 areas:

- Scientific-medical, in charge of elaborating guidelines for the diagnosis and treatment of the disease adapted to all the collectives involved. The document is structured based on scientific evidence, giving explicit recommendations that facilitate its final implementation in accordance with the standards of quality health care.²
- Patients. The worries and needs of people with COPD are compiled in order for the active participation of patients in the development and writing of the guidelines. In addition, formative strategies and strategies for self-care are proposed,³ and information materials are prepared for the patients affected by this disease.
- Diffusion-communication, responsible for the preparation of the promotional material, press communications, and relations with social and economic agents in order to spread information about the reality of COPD and the people who suffer from it.

SEPAR, together with other similar scientific societies, has elaborated COPD treatment guidelines which have served as a reference in Spain.^{4,5} This involves updating the content based on the multitude of advances that have arisen in recent years. The most basic advance is the patient approach based on phenotypes, a denomination that in recent years has been more widely used in referring to the clinical forms of COPD patients.^{6,7} A group of international experts have defined phenotypes as “those attributes of disease that, either alone or combined, describe the differences between individuals with COPD related to parameters that have clinical significance (symptoms, exacerbations, response to treatment, speed of the progression of the disease or death)”.⁶ Therefore, the phenotype should be able to classify the patients into subgroups with prognostic value that are able to determine the best therapy for achieving better results from a clinical perspective.^{6,8}

There are multiple studies that try to identify and quantify the prevalence of the different COPD phenotypes^{9,10} using diverse populations, severity, and characteristics. There is no consensus on the number or on the definition of the different phenotypes, but there should be an intermediate point between the excessive simplification of the term COPD, as a definition that covers the entire spectrum of patients with obstruction that is not completely reversible to airflow, and the complexity of considering each patient individually as an orphan disease.¹¹ This mid-point includes identifying and describing some phenotypes that are interesting not only regarding biology or epidemiology, but also regarding prognosis and especially therapy. From the analysis of these studies came the proposal to define 3 different phenotypes with clinical, prognostic, and therapeutic impact:

- Emphysema-hyperinflation.
- Overlapping or mixed COPD-asthma.
- Exacerbation phenotype.

The emphysema phenotype is characterized by parenchymatous destruction, air trapping, dyspnea, and the tendency towards a low body mass index.^{12,13} The COPD-asthma phenotype is characterized by an obstruction that is not completely reversible to the air flow accompanied by symptoms or signs of a reversibility increased by the obstruction¹⁴ and can include asthmatic

[☆] Please cite this article as: Grupo de trabajo de GESEPOC. Hacia un nuevo enfoque en el tratamiento de la EPOC. La Guía Española de la EPOC (GESEPOC) Arch Bronconeumol. 2011;47:379–81.

E-mail address: marcm@separ.es

individuals that smoke, asthmatics with a long evolution who develop obstruction that is not completely reversible to the air flow and non-smokers that develop chronic airflow obstruction. Smokers with asthma have features that are similar to COPD, with a lower frequency of eosinophilic inflammation and a greater probability of neutrophilia in the airways.^{15,16} The exacerbation phenotype is defined by patients who present 2 or more exacerbations per year.^{17,18}

Other possible phenotypes have been defined, but their transcendence in directing treatment is not established. Thus, a so-called “fast decliner” would be a patient who experiences a fall in lung function, expressed by FEV₁, which is faster than average.¹⁹ The practical problem is that it is impossible to identify this phenotype without a strict follow-up of the lung function for at least 2 years; on the other hand, no specific treatment has been identified for this type of patients. Another possible phenotype would be chronic bronchitis, defined as cough and expectoration for at least 3 months a year for 2 consecutive years. This phenotype is usually associated with airway disease, which can be visualized by HRCT.^{12,20} However, chronic bronchitis can accompany any of the 3 phenotypes mentioned previously, so we therefore prefer to describe it as a modifying factor in any of the 3 main phenotypes. Also, the so-called “systemic” phenotype, with significant metabolic or cardiovascular comorbidity, has been recently described,²¹ but the comorbidity should be considered as a characteristic that should always be kept in mind and that may accompany or complicate any of the 3 basic phenotypes. Last of all, a special phenotype is emphysema due to alfa-1-antitripsin deficiency, which is characterized by predominantly basal emphysema that appears at early ages, especially in smokers, and has a genetic base.²² Due to its low prevalence, we prefer to consider it apart from the general classification.

The importance of establishing these phenotypes is that the treatment is initially directed by the characteristics of each patient, which constitutes a personalized focus of the pharmacological treatment and rehabilitation^{10,23} that is able to be modulated according to severity. Another characteristic of the guidelines is that they contemplate not only the increase in treatment according to severity, but also the possible reduction of treatment depending on a sustained improvement in the symptoms. The evaluation of the severity has also been updated with the incorporation of multidimensional scales. The severity of the obstruction, measured by FEV₁, is fundamental, but not enough. Multidimensional indices, like BODE,²⁴ have demonstrated an excellent prognostic value. When the 6-min walk test cannot be done in the usual manner, the incorporation in the index of the frequency of exacerbations instead of exercise (BODEx index) offers similar prognostic properties.²⁵ The HADO score can be an excellent alternative in patients with an FEV₁>50%,²⁶ but in these patients, the survival is greater and a prognostic index is less necessary.

We are aware that this perspective is a significant change in the treatment of COPD, from an approach centered on the severity of the obstruction to a personalized approach centered on clinical characteristics. Parallel to this clinical perspective, GESEPOC wants to highlight the crucial role of the patients and their care-takers in the improvement of the results of the health-care process. As in all chronic diseases, self-care is an essential element. In order to achieve the best possible results, it is necessary to improve the health skills and understanding of the patients and their caretakers.²⁷ Some specific strategies, such as personalized plans of action, training an “expert patient”, or group visits can be useful.^{28,29}

A change of this magnitude requires the contributions, opinions, and finally the consensus of an important number of professionals related with COPD and the patients themselves who participate from the onset in the elaboration of the guidelines. With this

objective, a survey has been performed through the SEPAR COPD Observatory, followed by a meeting of close to 100 professionals from the various scientific societies participating in GESEPOC, in which a high degree of agreement was reached regarding these new directives.

GESEPOC (www.gesepoc.com) was born from the desire for continuity, for exploring new platforms for communicating with doctors, patients, the media, and health-care authorities, and to support the evaluation of its implementation and impact in the treatment of COPD. Such initiatives should contribute to optimizing the treatment of COPD patients, without forgetting the role that they can play in spreading the knowledge and understanding of this disease among the general population and politicians.

The GESEPOC Organization

Coordinator: Marc Miravittles, Spanish Society of Pulmonology and Thoracic surgery (SEPAR). Member of the workgroup: Myriam Calle and Juan José Soler-Cataluña (SEPAR); Joan B. Soriano (SEPAR-epidemiology); Julio Ancochea, Scientific Coordinator of the COPD Strategy of the SNS; Joan Escarrabill, Director of the del Pla Hospital and Director of Diseases of the Respiratory Apparatus (PDMAR) of the Department of Health (Barcelona); Pere Almagro, Spanish Society of Internal Medicine (SEMI); Daniel López (SEPAR-Physiotherapy); Ester Marco, Spanish Society of Rehabilitation and Physical Medicine and Cardio-Respiratory Rehabilitation Society (SERMEF/SORECAR); Juan Antonio Riesco, National Committee for Smoking Prevention (CNPT); José Antonio Quintano, Spanish Society of Primary Care Physicians (SEMERGEN), Juan Antonio Trigueros, Spanish Society of Spanish General and Family Physicians (SEMG); Jesús Molina, Spanish Society of Family and Community Medicine (semFYC) and Society of Respiratory Medicines in Primary Care (GRAP); Mercè Marzo (semFYC-Methodology); Pascual Piñera and Adolfo Simón, Spanish Society of Emergency Medicine (SEMES); Antonia Cachinero (SEPAR-Nursing); María Dolors Navarro, Spanish Patient Forum (FEP); Montse Llamas (UOC-AlaOeste-Communication). The strategic partners of GESEPOC include: Almirall, AstraZeneca, Boehringer Ingelheim-Pfizer, Faes Farma, Grupo Ferrer, Bayer Schering, GlaxoSmithKline, Novartis and Nycomed-Merck Sharp & Dhome. GESEPOC collaborators are Chiesi, Esteve Teijin and Grupo Uriach Pharma.

References

1. Ministerio de Sanidad y Política Social. Plan de Calidad para el Sistema Nacional de Salud. Estrategia en EPOC del Sistema Nacional de Salud. Sanidad 2009. Ministerio de Sanidad y Política Social. Available from: <http://www.msc.es/organizacion/sns/planCalidadSNS/docs/EstrategiaEPOCSNS.pdf> [accessed 10.03.11].
2. Soler-Cataluña JJ, Calle M, Cosío BG, Marín JM, Monsó E, Alfageme I. Estándares de calidad asistencial en la EPOC. Arch Bronconeumol. 2009;45:196–203.
3. Bourbeau J, van der Palen J. Promoting effective self-management programmes to improve COPD. Eur Respir J. 2009;33:461–3.
4. Peces-Barba G, Barberà JA, Agustí A, Casanova C, Casas A, Izquierdo JL, et al. Guía Clínica SEPAR-ALAT de diagnóstico y tratamiento de la EPOC. Arch Bronconeumol. 2008;44:271–81.
5. SEPAR-SemFYC. Atención integral al paciente con enfermedad pulmonar obstructiva crónica. Semfyc Ediciones; 2010.
6. Han MK, Agustí A, Calverley PM, Celli BR, Criner G, Curtis JL, et al. Chronic obstructive pulmonary disease phenotypes. The future of COPD. Am J Respir Crit Care Med. 2010;182:598–604.
7. Calle Rubio M, Rodríguez-Hermosa JL, Ortega-González A, Alvarez-Sala Walther JL. Fenotipos de la enfermedad pulmonar obstructiva crónica. Med Clin Monogr. 2007;8:22.
8. García-Aymerich J, Agustí A, Barberà JA, Belda J, Ferrero E, Ferrer A, et al. La heterogeneidad fenotípica de la EPOC. Arch Bronconeumol. 2009;45:129–38.
9. Soriano JB, Davis KJ, Coleman B, Visick G, Mannino D, Pride NB. The proportional Venn diagram of obstructive lung disease. Chest. 2003;124:474–81.
10. Miravittles M. Tratamiento individualizado de la EPOC: una propuesta de cambio. Arch Bronconeumol. 2009;45 Suppl. 5:27–34.
11. Rennard SI, Vestbo J. The many “small COPDs”: COPD should be an orphan disease. Chest. 2008;134:623–7.

12. Patel BD, Coxson HO, Pillai SG, Agustí AGN, Calverley PMA, Donner CF, et al. Airway wall thickening and emphysema show independent familial aggregation in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2008;178:500-5.
13. Casanova C, Cote C, De Torres JP, Aguirre-Jaime A, Marín JM, Pinto-Plata V, et al. Inspiratory-to-total lung capacity ratio predicts mortality in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2005;171:591-7.
14. Gibson PG, Simpson JL. The overlap syndrome of asthma and COPD: what are its features and how important is it? *Thorax*. 2009;64:728-35.
15. Chalmers GW, MacLeod KJ, Thomson L, Little SA, McSharry C, Thomson NC. Smoking and airways inflammation in patients with mild asthma. *Chest*. 2001;120:1917-22.
16. Boulet LP, Lemiere C, Archambault F, Carrier G, Descary MC, Deschesnes F. Smoking and asthma: clinical and radiological features, lung function, and airway inflammation. *Chest*. 2006;129:661-8.
17. Hurst JR, Vestbo J, Anzueto A, Locantore N, Müllerova H, Tal-Singer R, et al. Susceptibility to exacerbation in chronic obstructive pulmonary disease. *N Engl J Med*. 2010;363:1128-38.
18. Soler-Cataluña JJ, Martínez-García MA, Román Sanchez P, Salcedo E, Navarro M, Ochando R. Severe acute exacerbations and mortality in patients with chronic obstructive pulmonary disease. *Thorax*. 2005;60:925-31.
19. Celli BR, Thomas NE, Anderson JA, Ferguson GT, Jenkins CR, Jones PW, et al. Effect of pharmacotherapy on rate of decline of lung function in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2008;178:332-8.
20. Martínez-García F, Soler-Cataluña JJ, Donat Y, Catalán P, Agramunt M, Ballestin V, et al. Factors associated with bronchiectasis in chronic obstructive pulmonary disease patients. *Chest*. 2011;101758, doi:10.1378/chest.10-1758.
21. Mair G, Maclay J, Miller JJ, MacAllister D, Connell M, Murchison JT, et al. Airway dimensions in COPD: relationship with clinical variables. *Respir Med*. 2010;104:1683-90.
22. Garcia-Aymerich J, Gómez FP, Benet M, Farrero E, Basagaña X, Gayete A, et al. Identification and prospective validation of clinically relevant chronic obstructive pulmonary disease (COPD) phenotypes. *Thorax*. 2011;66:430-7.
23. Vidal R, Blanco I, Casas F, Jardí R, Miravittles M, Comité del Registro Nacional de Pacientes con Déficit de Alfa-1-antitripsina. Normativa SEPAR: Diagnóstico y tratamiento del déficit de alfa-1-antitripsina. *Arch Bronconeumol*. 2006;42:645-59.
24. Anderson D, MacNee W. Targeted treatment in COPD: a multi-system approach for a multi-system disease. *Int J Chron Obst Pulm Dis*. 2009;4:321-35.
25. Celli BR, Cote CG, Marín JM, Casanova C, Montes de Oca M, Méndez RA, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. *N Engl J Med*. 2004;350:1005-12.
26. Soler-Cataluña JJ, Martínez-García MA, Sánchez L, Perpiña M, Román P. Severe exacerbations and BODE index: two independent risk factors for death in male COPD patients. *Respir Med*. 2009;103:692-9.
27. Esteban C, Quintana JM, Moraza J, Aburto M, Aguirre U, Aguirregomoscorta JJ, et al. BODE-index vs HADO-score in chronic obstructive pulmonary disease: which one to use in general practice? *BMC Med*. 2010;8:28.
28. Roberts NJ, Ghiassi R, Partridge MR. Health literacy in COPD. *Int J Chron Obstruct Pulm Dis*. 2008;3:499-507.
29. Bischoff EW, Hamd DH, Sedeno M, Benedetti A, Schermer TR, Bernard S, et al. Effects of written action plan adherence on COPD exacerbation recovery. *Thorax*. 2011;66:26-31.