Chronic Obstructive Pulmonary Disease, Antibiotics, and Prediction of Bacterial Infection

Sir—Prescription of antibiotics to patients with acute exacerbation of chronic obstructive pulmonary disease (COPD) is perceived to be excessive and sometimes irrational. The recent article by van der Valk et al. [1] provides evidence in support of the potential shortcomings in the clinical practice of prescribing antibiotics to patients with acute exacerbations of COPD. The accompanying editorial commentary [2] pointed out some of the shortcomings of this work, including the lack of clinical outcome measures and the absence of information concerning prevalent bacterial or viral causative pathogens.

We appreciated the effort by van der Valk et al. [1] to identify new and improved methods to guide antibiotic prescription in everyday clinical practice for patients with acute exacerbations of COPD, as well as their rigorous approach to defining exacerbation-related events and evaluating clinical procedures. However, in addition to the limits of this work that were pointed out by Niederman [2] (whose views we share), there are 2 additional aspects that are of concern.

First, the study involved only 116 patients, of whom 22 (19%) had a bacterial infection. There is a general experimental agreement that ~40% of acute exacerbations of COPD have a viral etiology [3, 4]. Thus, exacerbations in ~40% of 116 patients seen by van der Valk et al. [1] would be expected to have a viral etiology; exacerbations in 22 (19%) were determined to be of bacterial origin, whereas exacerbations in ~40% of 116 would have been of noninfectious etiology. Indeed, this proportion of non–infection-driven acute exacerbations of COPD is somewhat excessive, compared with published data [5] and with my experience in routine clinical practice. Failure to correctly identify bacterial infections or bias toward identification of non–infection-driven acute exacerbations could explain the lack of correlation with other parameters (e.g., Anthonisen criteria and previous cold) and affect the evaluation of inappropriate antibiotic use (with respect to providing or withholding treatment) reported in the study.

Second, one of the reasons for the clinical success of the criteria developed by Anthonisen et al. [6] to classify acute exacerbation of COPD lies in their easy, quick use at the bedside. Although we agree that Gram staining is not a complicated technique, it requires (as van der Valk et al. [1] note) technical skill, proper equipment, and 30–45 min of dedicated processing time. For some health care systems, routine use of Gram staining may well prove to be unfeasible or even unreliable if the operator is not used to processing the samples and interpreting the results.

Thus, we suggest that it could be misleading to draw general conclusions or even indications for standard care on the basis of the results of this study. The local epidemiological conditions related to both patient selection and microorganism circulation need to be carefully verified in a study involving a much larger number of patients from different health care settings. Confirmation of the superiority of a new strategy for patient selection that would be widely and easily accepted can only be based on clinical experience and on results of controlled trials with clinical or functional outcomes.

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References


Reply to De Maria

We appreciate the comments of De Maria [1] regarding our recently published article [2] and would like to address his concerns. The first concern, apart from the limitations expressed by Niederman [3], pertains to the excessive proportion of non–infection-driven cases of acute exacerbation of chronic obstructive pulmonary disease (COPD) reported in our study, compared with published data and the proportion encountered during routine clinical practice [4]. Failure to correctly identify bacterial infections could be one of the reasons for this difference and would modify the evaluation of inappropriate antibiotic use (with respect to providing and withholding treatment). We argue that this oversight is unlikely, because we used rigorous microbiological techniques to detect potential bacterial infections [2]. We are aware that differences in