There Are Still Problems in Establishing a Diagnosis of Gastroesophageal Reflux-Related Chronic Cough

To the Editor:

A recent study of 281 patients with extraesophageal syndromes of gastroesophageal reflux disease, of whom 50% had a cough, estimated that 52% of the overall US national annual economic burden of extraesophageal syndromes was attributable to proton pump inhibitor therapy. It is becoming increasingly important to identify patient populations likely to be responsive, avoiding unnecessary or inappropriate treatment. Xu et al. should be congratulated on their study recently published in CHEST (June 2014) applying the Gastroesophageal Reflux Disease Questionnaire to predict patients with gastroesophageal reflux-induced chronic cough (GERC) who may be responsive to antireflux therapy. We have some questions and comments.

First, were any other common causes ruled out in the potential subjects with suspected GERC prior to participation? A prospective multicenter study showed that cough-variant asthma (32.6%), upper airway cough syndrome (18.6%), eosinophilic bronchitis (17.2%), and atopic cough (13.2%) are among the most common causes of chronic cough in China, with GERC in only 4.6% of cases. Xu et al. did not report any information about bronchial provocation test, induced sputum test, peripheral blood eosinophil count, or serum IgE level, suggesting that some common causes of chronic cough could not have been included, which might lead to unnecessary invasive tests and excessive treatment. Additionally, other information, such as smoking, body weight, and lifestyle modification, was not reported, which would be useful and important.

Second, was it reasonable to establish the diagnosis based on favorable response to antireflux therapy? Empirical trials have been widely used in the diagnosis of GERC, since no diagnostic gold standard is available. However, diagnosis based exclusively on therapeutic response has been challenged with the increasing evidence that patients benefit little from acid-suppressive therapy and with the likely mechanisms of hypersensitivity and vagal reflex.

Third, we wonder about the presence of other causes of chronic cough in some patients. Cough in 8.0% of patients resistant to initial acid-suppression therapy was resolved using baclofen as an add-on therapy. Indeed, baclofen could improve refractory cough attributed to gastroesophageal reflux disease. However, baclofen, an agonist of γ-aminobutyric acid, has also been shown to inhibit reflux cough due to other causes. In such cases, other possible causes responsive to baclofen would be considered in addition to GERC, impairing the predictive value of the Gastroesophageal Reflux Disease Questionnaire.

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Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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DOI: 10.1378/chest.14-0643

REFERENCES


Response

To the Editor:

We thank Drs Jiang and Mao for their insightful comments regarding our recent article. The goal of the study is to select the appropriate patients for empirical antireflux trial and to improve therapeutic gains. Actually, the patients were lifetime nonsmokers or ex-smokers for at least 2 years. The patients received an initial laboratory workup that included all the examinations Drs Jiang and Mao mentioned except for those patients with the obvious reflux-related symptoms, who accepted the evaluation of gastroesophageal reflux-induced chronic cough (GERC). Therefore, no other common causes of chronic cough were identified or were the explanation for their cough in the patients with potential GERC when recruited. Because of the limit of words in the