


Response

To the Editor:

We thank Dr Nair for his pertinent comments concerning the use of fluorodeoxyglucose (FDG)-PET scanning in the evaluation of pleural disease. As we made clear in our description of a case of pleural disease associated with ankylosing spondylitis (AS) recently published in CHEST (October 2010), 1 FDG-PET scan-avid pleural lesions only denote increased metabolic activity within the area of interest but do not provide specific information concerning the source of that activity, which may be neoplastic or inflammatory. 2 As is true for many imaging modalities, on some occasions the clinical scenario and/or radiographic appearance are such that a specific nonmalignant diagnosis can be made with enough confidence to obviate the need for an invasive procedure. Clearly, in our patient with a history of tobacco use and possible asbestos exposure it was mandatory that a firm, histologic diagnosis be pursued.

Nevertheless, we believe it is important to remind clinicians that FDG-PET scan avidity has been demonstrated in a wide variety of inflammatory disorders affecting the lungs and pleura, including infection as well as noninfectious granulomatous disease, such as those produced by talc pleurodesis or due to sarcoidosis. 3,4 However, the appearance of pleural disease associated with collagen vascular disorders on FDG-PET scan has only rarely been reported. Rheumatoid arthritis with FDG-PET scan-avid pleural involvement has been described in only one previous report. 5 Spinal lesions with FDG-PET scan avidity are known to occur in patients with AS, 6 but it appears that our case is the first to describe FDG-PET scan positivity in pleural disease due to AS.

Lee K. Brown, MD, FCCP
Albuquerque, NM
Shadi Battah, MD
Anchorage, AK
Cecilia Wu, MD
Allison Richards, MD
Lida Crooks, MD
Michael Hawthorne, MD
Albuquerque, NM

Affiliations: From the Division of Pulmonary, Critical Care, and Sleep Medicine, Department of Internal Medicine (Dr Brown), the Department of Pathology (Drs Wu and Crooks), and the Department of Radiology (Drs Richards and Hartshorne), University of New Mexico School of Medicine; and The Alaska Hospitalist Group (Dr Battah).

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Correspondence to: Lee K. Brown, MD, FCCP, Department of Internal Medicine, University of New Mexico School of Medicine, 1101 Medical Arts Ave NE, Bldg #2, Albuquerque, NM 87102; e-mail: lbrown@alum.mit.edu

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Silicosis: Hidden Behind TB?

To the Editor:

We read with great interest the case report of a 53-year-old man with dysphagia, anorexia, and night sweats by Ferguson and Schwarz in a recent issue of CHEST (November 2010). 1 We believe a few issues need to be addressed after going through this report.

Although a diagnosis of TB was confirmed on sputum cultures, coexistence of a concomitant pneumoconiosis (eg, silicosis in this case) cannot be ruled out because there is a history of exposure to the cement industry. Silicotuberculosis is a well-described entity in literature, and it is said that silicosis increases the predisposition toward TB. 2 The parenchymal nodules on chest radiograph and diffuse miliary pattern with mediastinal lymphadenopathy on CT scan described in this patient can be associated with silicosis. 3 In this patient, the miliary shadows could be present because of preexisting silicosis and right upper lobe infiltrate because of superadded TB infection. Flexible bronchoscopy (transbronchial lung biopsy and BAL) or, preferably, open lung biopsy might have

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helped to rule out concomitant silicosis in this case. Patients with silicobronchitis may have resistance to antituberculous medications, and they may need a longer duration of treatment as compared with TB alone; hence, it is important to recognize concomitant silicosis in the context of this case report.

Pleuritic chest pain and pericardial effusion on echocardiogram suggest the diagnosis of pericardial TB, but the authors did not mention this in their final diagnosis. Moreover, steroids should have been added for treatment of pericardial TB in addition to standard anti-TB regimen.

Alkesh Kumar Khurana, MD, FCCP
Chandigarh, India

Anup Kumar Singh, MD, FCCP

Affiliations: From the Department of Pulmonary Medicine (Dr Khurana), Government Medical College and Hospital; and the Department of Internal Medicine (Dr Singh), Unity Hospital.

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Correspondence to: Alkesh Kumar Khurana, MD, FCCP, Government Medical College and Hospital, Pulmonary Medicine, GMCH Sec 32 Chandigarh, 160030 India; e-mail: lungcancer@rediffmail.com

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What Justifies a Placebo-Controlled Trial of Varenicline for Smoking Cessation in Patients With COPD?

To the Editor:

In a recent issue of CHEST (March 2011), Tashkin et al1 present results of a double-blind, placebo-controlled, two-arm trial of varenicline for smoking cessation in patients with COPD. The question arises: Why was a trial conducted in which varenicline was compared only with a placebo (and not with another active smoking cessation drug) in this patient group?

According to article 32 of the World Medical Association’s Ethical Principles for Medical Research Involving Human Subjects, “the effectiveness of a new intervention must be tested against those of the best current proven intervention.”2 The use of a placebo is only acceptable in studies in which no current proven intervention exists. However, nicotine replacement therapy and bupropion were available interventions recommended by international evidence-based guidelines as first-line pharmacologic treatments for smoking cessation in patients with respiratory disease.3 For example, an earlier trial in patients with COPD, notably, conducted by Tashkin et al2 as well, had shown that smokers who received bupropion for smoking cessation achieved higher continuous abstinence rates than smokers treated with placebo. Furthermore, two large-scale trials4,5 had clearly shown the greater benefit of varenicline compared with placebo and bupropion in the general smoking population—1 year before the current trial by Tashkin et al was initiated. The trial by Tashkin et al is the first to investigate the efficacy of varenicline in patients with COPD, but the authors do not provide compelling and scientifically sound methodologic reasons that justify the use of a placebo control instead of existing evidence-based smoking cessation medication. They only state that smokers with COPD have higher levels of nicotine dependence and are “more resistant to smoking cessation interventions” than smokers without COPD, but this is true for other subtypes of smokers as well, for example smokers with a lower socioeconomic background.

Two-arm placebo-controlled trials should no longer be conducted because they do not provide sufficient information on the effectiveness and safety of a new smoking cessation drug in relation to existing drugs. Given the evidence base of available pharmacologic aids for smoking cessation, future trials with varenicline (or other drugs) that provide good reasons for using a placebo as a comparator should at least incorporate a third study arm in which the best alternative pharmacologic treatment of smoking cessation is administered. However, a search of international registers (http://apps.who.int/trialsearch) shows that several trials are still recruiting smokers into two-arm placebo-controlled trials with varenicline (for example trials in patients with depression, schizophrenia, bipolar disorder, and HIV) and trials in smokers receiving alternative dosing schedules and varenicline for relapse prevention. Researchers, medical ethics committees, and regulatory authorities should keep in mind that the health of smokers in a placebo group is at stake. Smokers from the placebo group have a decreased chance of successful quitting, and each unsuccessful attempt increases the risk of smoking-related disease and reduced life expectancy, especially in a vulnerable group like patients with COPD.

Daniel Kotz, PhD
Onno C. P. van Schayck, PhD
Maastricht, The Netherlands

References