marijuana smoking in a patient without clinical evidence of an immune deficiency. However, two aspects of this report require further discussion.

First, instead of invasive pulmonary aspergillosis (IPA), we believe the precise diagnosis should be chronic necrotizing pulmonary aspergillosis (CNPA). To start with, the patient does not show any clinical symptoms of IPA, nor are her COPD symptoms getting any more severe. The occurrence of IPA in COPD patients will intensify symptoms like shortness of breath. Next, the CT scan of her chest shows no evidence that her left upper lobe nodule is progressing any further. This suggests that the nodule is of a slow progressive nature.

In addition, the findings of the two CT scan-guided biopsies were both nondiagnostic and revealed predominantly fibrous tissue. In clinical practices, the diagnosis of CNPA commonly stands on the presence of multiple cultures that are positive for Aspergillus organisms, chest radiographs with abnormal findings, and test findings for bronchoscopy biopsy specimens that are consistent with tissue invasion.3

Lastly, the authors’ diagnosis rests mainly on the results of a biopsy performed with video-assisted thoracic surgery, showing caseating granulomas and granulomatous pleuritis, with evidence of Aspergillus species and thrombosed vessels in the center of one of the nodules. However, Yousum1 has also reported 4 of 10 persons with conditions resembling necrotizing granulomatous pneumonia centered around a central zone of infarct-like necrosis of the parenchyma, resulting from angiinvasive Aspergillus. This is incredibly similar to the histopathology reported by Sakkour et al.1

In conclusion, in contrast to IPA, CNPA is a chronic process that progresses slowly over months to years. IPA, on the other hand, is a severe and commonly fatal disease that is seen in immunocompromised patients. Therefore, we incline to believe that the diagnosis in this case should be CNPA.

Second, we totally agree with the authors in regarding marijuana smoking as the main factor for pulmonary aspergillosis in this patient. Nonetheless, we believe that the fluticasone therapy the patient received (since the report did not specify the dosage of fluticasone) can only take the recommended dosage of fluticasone for a severe pulmonary patient into consideration) is also one of the key factors for the occurrence of pulmonary aspergillosis in this patient. Reports4–5 have shown that therapy with not only oral steroids, but also with inhaled steroids might promote IPA or CNPA in COPD patients.

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Does Biofilm Formation Play a Role in Ventilator-Associated Tracheobronchitis?

To The Editor:

I enjoyed reading “Ventilator-Associated Tracheobronchitis” by Craven et al1 in a recent issue of CHEST (February 2009). It may be useful to determine whether this condition includes biofilm formation. The authors mentioned contiguous biofilm sites including the endotracheal tube and oral cavity. Dental plaque is a form of biofilm that supports the growth of pulmonary pathogens and is a probable source of pneumonia. Oral hygiene decreases rates of ventilator-associated pneumonia in some groups.2

Pseudomonas and Staphylococcus aureus are biofilm formers,3–5 Biofilm colonies anchor to mucosal surfaces or foreign bodies and are composed of layers of slow-growing bacteria embedded in the glyocalyx (exopolysaccarides). The close proximity of bacteria facilitates chemical quorum sensing when the colony achieves high density, triggering the production of virulence factors and/or launching free-living bacteria to infect other sites such as the alveolae. Glyocalyx interferes with antibiotic penetration, and slow growth makes bacteria resistant to growth-dependent antibiotic killing. Killing bacteria in biofilm requires antibiotic concentrations 10 to 1,000 times that needed to kill free-living bacteria. Conceivably, topical or aerosolized antibiot- ics could achieve local concentrations high enough to suppress biofilm formation and avoid exposing bacteria to sub-minimum inhibitory antibiotic concentrations if the biofilm was not fully mature. Other novel therapies that can be directed toward the formation of biofilms or are capable of breaking the chemical bonds of the biofilms may become available.3–5

The exopolysaccarides in biofilm cannot be visualized by conventional light microscopy.4 Detection requires scanning electron or laser microscopy. Fluorescent in situ hybridization is required to identify specific bacteria.3 These techniques have demonstrated the presence of biofilm and clarified the pathophysiology of ear, nose, and throat infections, including chronic sinusitis, otitis media with effusion, and adenotonsillitis.5 Perhaps it is time to apply the same techniques to determine whether biofilm formation is part of the pathophysiology of ventilator-associated tracheobronchitis.

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Unify the Evaluative Procedures and Involve Peers for Increasing Use of Guidelines in Daily Practices

To the Editor:

Recently, Mazmanian et al have shown the impact of continuing medical education (CME) interventions for increasing the use of clinical practice guidelines (CPGs) in daily practices and illustrated the important issue of physician adherence to CPGs. In France, the main interventions intended to change physicians’ behavior and daily practice are included in the evaluation of medical practices (EMP) step. The EMP is a key feature of continuous quality improvement, and its aim is to help doctors to reflect on their own practices and enhance adherence to CPGs. Beginning in 2005, EMP has been a legal obligation, and French physicians have to continually evaluate their practice.2

Due to the development of evaluative procedures (ie, certification, accreditation of medical teams, EMP, and CME), confusion regarding who is responsible for these procedures has occurred. Many organizations are involved in the EMP (eg, hospital medical committees, specialty societies, French National Institute of Health, and private organizations), some of which are neither acknowledged committees, specialty societies, French National Institute of Health, nor CME but are also involved in the EMP implementation. Indeed, EMP is a “professional thing,” and the involvement of medical specialty societies in EMP is a key component of its success. The influence of medical specialty societies is probably the most important contributor to doctors’ behavioral changes. In a study3 carried out in 2005, we found that hospital physicians generally valued guidelines and hence adhered to them, according to their promoter, more than to the scientific consistency of guidelines.

We propose that the involvement of medical specialty societies also contributes to the success of EMP activities, as has been demonstrated in the literature. Grobl1 has recommended targeting each specific kind of medical professional to achieve the best integration of CPGs and to have a real impact on clinical practices. Starting with this viewpoint, the national health agencies should integrate medical specialty societies into the EMP development process to enhance the participation of medical professionals in peer teaching activities, which are still an under-recognized source of education in the medical education continuum.5

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