are basically in agreement with his statements, we feel a few comments are in order.

The extremely high rate of specific benign diagnoses reported by Khouri et al. are not the experience of most practitioners, and is probably due to their unique expertise, as well as the fact that in most instances they used an 18 or 20 ga needle to obtain a core of tissue for histologic evaluation, rather than aspiration by a 22 ga needle for cytology, as is the practice in most centers. In less skilled hands one would expect an increased incidence of complications using their technique.

A recent negative prior radiograph increases the likelihood that the lung nodule is malignant, and would be a reason not to do needle aspiration.

One of our patients developed chest wall implantation following needle aspiration biopsy of a completely resected T1 N0 M0 lung cancer. Seyfer et al. recently reported a similar occurrence. Chest wall or pleural recurrence from tumor implantation in lesions that are potentially curable certainly is a "significant factor in the decision process" and is a tragic occurrence. We continue to feel strongly that needle aspiration biopsy, with its attendant risks, should be reserved for patients who are not surgical candidates.

We agree that if specific patterns of calcification which indicate benignity can be demonstrated by either standard or high-resolution CT scans, neither needle aspiration nor thorocotomy would be required. Perhaps the use of the phantom scan will be refined enough in the future to discriminate benign from malignant lung nodules.1,4

As surgeons, we are very sensitive to local recurrence following resection of potentially curable lesions. The point of our editorial is that this is a very real possibility following needle aspiration of lung cancers, and should be an important consideration in the decision-making process for the evaluation of solitary pulmonary nodules which have a high likelihood of being malignant.

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REFERENCES


L-Tryptophan Induced Cough and Pleural Effusions Associated with the Esophagitis-Myalgia Syndrome

To the Editor:

I would like to report a patient with cough and pleural effusions associated with the esophagitis-myalgia syndrome which was caused by the ingestion of L-tryptophan. L-tryptophan is an over-the-counter preparation used to treat a variety of psychiatric disorders.1

A 34-year-old white woman presented with a three-week history of severe tightness in the chest, cough and diffuse myalgias. One week prior to the onset of her symptoms she started taking one gram of tryptophan. On physical examination she appeared in pain with simple movement and was unable to take a deep breath without coughing. Chest x-ray revealed a faint interstitial infiltrate and small bilateral effusions. Her WBC count was 29,000 with 45 percent eosinophils, which dropped to 11,000 with 38 percent eosinophils on day 21. Aldolase was elevated to 15 mu/ml (normal 1.2 to 7.6). The patient improved upon withdrawal of the tryptophan.

The CDC has reported more than 300 cases of a new syndrome called the esophagitis-myalgia syndrome associated with the ingestion of L-tryptophan products. It is interesting to speculate on the possible role of the eosinophil major basic protein in causing the considerable respiratory findings in this patient.4

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REFERENCES


Spontaneous Resolution of Endobronchial Mycobacterium avium-Intracellulare Infection in a Patient with AIDS

To the Editor:

We previously reported a case of endobronchial Mycobacterium avium-intracellulare infection (MAI) in a patient with AIDS whom we evaluated for massive hemoptysis.1 Bronchoscopy examination initially disclosed no specific abnormality with the exception of a fresh clot in the posterior segment of the right upper lobe bronchus. A second bronchoscopy performed for evaluation of recurrent massive hemoptysis revealed a clot in the same location. Bronchial artery embolization of the corresponding vessels was therefore performed to control bleeding. One month later a third bronchoscopy performed as a follow-up evaluation disclosed multiple, partially obstructing, polypoid endobronchial lesions. Endobronchial biopsies were performed. Histologic examination revealed necrotizing and non-necrotizing granulomas, and the tissue cultures ultimately grew MAI. Five months later, recurrent hemoptysis, fever, and a chest radiograph typical of bronchiectasis necessitated endobronchial examination. Polypoid masses occluding multiple bronchial segments were partially removed using biopsy forceps, which allowed drainage of purulent material from the distal airways. During the next nine months, the patient’s clinical course was dominated by recurrent maxillary sinusitis resistant to multiple courses of antibiotics (amoxicillin-clavulanate, ciprofloxacin); progressive bilateral, interstitial lower lobe infiltrates; and progressive CMV retinitis. Intravenous DHFG and prophylactic therapy with aerosolized pentamidine and oral zidovudine in addition to cyclic antibiotics (erythromycin, tetracycline, cefadroxil) and monthly gamma globulin injections for control of lower airway infection were utilized over this same period. Bronchoscopy performed for evaluation of the lower lobe interstitial infiltrates 14 months after the initial airway examination demonstrating endobronchial lesions revealed absence of polypoid airway masses (Fig 1). Transbronchial biopsies were unremarkable for infectious organisms or a specific pathologic abnormality. Cytomegalic virus was recovered from...