Trees Don’t Grow in the Lungs!

To the Editor:

We read with interest a recent article1 on the BBC Web site of a 5-cm fir tree discovered by doctors in the lungs of a Russian botanist who underwent resection for a “lung tumor” after he presented with chest pain. The surgeon who operated on him commented that “The branch was green, as if it had just been taken from the wood. It’s still a mystery how the tree got in there.” It was thought that the patient had inhaled a seed, which was then grown into a tree inside his body! Two pulmonologists from South Africa were skeptical and believed it to be a medical hoax.2

Coincidentally, in another recent article published around the same time in the April 2009 issue of the Journal of Bronchology and Interventional Pulmonology, Davis et al3 described the case of a 45-year-old man in whom recurrent pneumonias developed 26 years after an impaling accident on a tree sucker and who was found to have a 5-cm splinter from a piece of wood in his right mainstem bronchus following a thoracotomy. The article was entitled “A tree grows in bronchus”; yet, the incident on aspiration was well documented. The title of the article was chosen to express the patient’s perception and to make it “catchy.”

We would like to bring to the attention of readers that trees do not grow in humans. To the best of our knowledge, there has not grown in humans. To the best of our knowledge, there has never been a single report in the medical literature of seeds and/or plants growing in humans. If they did, watermelon seeds and peanuts, which are the most commonly aspirated foreign bodies, would be growing out of control from our lungs. Moreover, it makes no biological sense that in the absence of sunlight and appropriate nutrient medium, photosynthesis and germination of a seed can take place. Foreign-body aspiration often goes undetected if the initial choking episode is not obvious. In adults, a reason for the lack of acute symptoms may be the larger caliber of airways, resulting in most foreign bodies lodging in distal airways. Seeds and plant material by themselves, however, are radiolucent, and any radio-opacity seen is likely from complications. A high index of suspicion is required. A bronchoscopic examination of the airway will establish the diagnosis.

In the realm of scientific observation, the adage “trees do not grow in the lungs” indeed holds true in every sense.

References


Could Fiberoptic Bronchoscopy and CT Lung Scan Differentiate Ventilator-Associated Tracheobronchitis From Ventilator-Associated Pneumonia?

To the Editor:

I read with interest the article in CHEST (February 2009) by Drs. Chua and Mehta1 on ventilator-associated tracheobronchitis (VAT). In this general review, the authors elegantly discussed recent findings on the impact of targeted antibiotic therapy on patient outcomes.2 They outlined the difficulty in differentiating VAT from ventilator-associated pneumonia (VAP) and suggested fiberoptic bronchoscopy and CT lung scan to confirm the diagnosis of VAP. However, some clarification would be helpful for ICU physicians.

The authors stated that quantitative samples obtained from the distal airway using bronchoscopic or nonbronchoscopic lavage or specimen brush were used to confirm VAP. Do the authors...
suggest performing fiberoptic bronchoscopy in all patients with suspected VAT? According to the definition of VAT suggested by the authors, these patients do not have a new pulmonary infiltrate and might represent a large proportion of patients receiving mechanical ventilation because fever and purulent endotracheal aspirate are common in these patients. However, postmortem animal and human studies demonstrated an acceptable diagnostic accuracy of quantitative endotracheal aspirate in diagnosing VAT compared with bronchoscopic lavage or specimen brush. Fiberoptic bronchoscopy is considered to be a relatively safe procedure. Serious complications such as bleeding, bronchospasm, arrhythmia, pneumothorax, and pneumonia occur rarely. However, a recent study found fiberoptic bronchoscopy to be frequently associated with decreased mesenteric blood flow, which may place the patient at risk for mesenteric ischemia and GI bacterial translocation. Further, Baram et al demonstrated that stable patients receiving prolonged mechanical ventilation had a high alveolar burden of bacteria, exceeding the commonly accepted threshold for diagnosing VAT in most patients.

The authors also suggested that CT lung scan could differentiate VAT from VAP. However, recent guidelines require the presence of new pulmonary infiltrate to diagnose VAP. Therefore, to diagnose a new infiltrate on CT scan, a baseline examination is mandatory. One could wonder whether performing CT scan in all patients at ICU admission to differentiate potential subsequent VAT from VAP would be cost-effective. In addition, intrahospital transport is required because this diagnostic procedure is unrealistic inside the ICU. Intrahospital transport is associated with considerable potential for misadventure and can be a life-threatening endeavor. In addition, a recent case-control study identified intrahospital transport (odds ratio, 2.9; 95% confidence interval, 1.4 to 5.7) as an independent risk factor for VAT. The supine position during intrahospital transport, which increases the risk of aspiration of gastric content or of contaminated secretions, and the frequent manipulations of the ventilator circuits needed during intrahospital transport, are well-known risk factors for VAT.

In fac, procalcitonin would be an interesting marker to differentiate VAT from airway tract colonization. In a recent, prospective observational study performed in COPD patients, procalcitonin was independently associated with community-acquired bacterial bronchitis. Future studies should determine whether these data are applicable to patients with VAT.

**References**


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**Carriage Classification of Pneumonia Rather Than Time Improves Survival**

To the Editor:

We always enjoy Craven’s editorials on pneumonia prevention as they are accurate and constructive. In his previous editorial in CHEST (July 2006),1 Craven acknowledged that antibiotic prophylaxis with selective digestive decontamination (SDD) is a valid strategy that reduces mortality. The SDD philosophy is based on the carrier state concept, which distinguishes among three different types of pneumonia (primary endogenous, secondary endogenous, and exogenous pneumonia), due to a limited range of potentially pathogenic microorganisms (six “normal” organisms and nine “abnormal” organisms). Each of the three types of pneumonia requires different prophylactic interventions. Parenteral antimicrobial agents control primary endogenous pneumonia, enteral antimicrobial agents prevent secondary endogenous pneumonia, and only a high level of hygiene can control exogenous pneumonia. SDD using hygiene, and parenteral and enteral antimicrobial agents is a prophylactic protocol that aims at the control of exogenous, primary endogenous, and secondary endogenous pneumonias, and at a reduction in mortality.

In line with the SDD philosophy, Craven, in his recent editorial in CHEST (November 2008),2 acknowledged that different prophylactic interventions are required to control the different types of pneumonia. He suggested the use of subglottic drainage to control early-onset pneumonia, while parenteral agents are required to control lower airway infections due to respiratory pathogens present in the flora at hospital admission, and silver-coated tubes to control late-onset pneumonia.4

This proposal is puzzling, as neither subglottic drainage nor silver-coated tubes have been associated with a survival benefit,4 while therapy with parenteral antimicrobial agents without enteral antimicrobial agents promotes the emergence of resistance.5

Additionally, in the subglottic drainage study pneumonia was not significantly reduced when all randomized patients were considered or when the intention-to-treat analysis was used. Only a post hoc analysis of a small subset of patients who had received ventilation for > 48 h demonstrated a significant, albeit borderline, impact from subglottic drainage (relative risk, 0.40; 95% confidence interval, 0.16 to 0.99; p = 0.04).

Similarly, in the silver-coated endotracheal tube study the difference in pneumonia rate was marginally significant in the intention-to-treat analysis, and the exclusion of pneumonia episodes due to coagulase-negative staphylococci and enterococci, which usually do not cause pneumonia, might importantly change the results. Finally, exogenous pneumonias, in which microorganisms are introduced directly into the lower airways due to poor