To the Editor:

In a recent issue of CHEST (January 2011), Gounant et al. showed that dedicated linear echoendoscope endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) needles are able to release metal particles, probably by friction between the stylet and the needle, with a potential risk of injecting particles into nodes. After reading this article, we are a bit confused for several reasons.

First, we do not understand the primum movens and the intrinsic aim of designing a study like this. Logically, we should perform similar investigations on all surgical, endoscopic, and radiologic procedures in which metal tools are used. Indeed, a simple blood test with a needle could release metal particles. Therefore, we do not understand the need to focus specifically on dedicated EBUS-TBNA needles.

Second, the article does not indicate the concentrations of iron, titanium, nickel, and chromium that can be potentially harmful to the body. This is important information, considering how many times a patient may undergo EBUS-TBNA over a lifetime (one, maybe two times). Accordingly, we have some doubts that the concentrations released in the lymph nodes are so high as to be potentially harmful to the body.

In conclusion, we completely agree with the authors that transbronchial needle aspiration using a flexible bronchoscope (conventional transbronchial needle aspiration) or linear echoendoscope (endobronchial ultrasound) allowing real-time guided lymph node aspiration are minimally invasive procedures for the diagnosis of mediastinal lymphadenopathy, with a very high sensitivity, a very low morbidity, and no reported mortality. Although the release of metal particles by an EBUS-TBNA needle may be reported to the manufacturers of these needles, this must not lead to a reduction in, or questioning of, the use of EBUS-TBNA in the diagnosis of mediastinal lymphadenopathy.

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REFERENCES


Response

To the Editor:

We thank Dr. Casoni and colleagues for their interest in our recent article published in CHEST (January 2011) on the release of metal particles from ViziShot needles (Olympus Ltd; Tokyo, Japan) used for endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA). We believe that Dr. Casoni et al. failed to understand why this study was designed. In fact, transbronchial needle aspiration (conventional and endobronchial ultrasound-guided) was introduced in our respiratory disease center in 2007, and we very rapidly became intrigued by the deposition of foreign material on slides prepared for cytopathologic examination. This is a very unusual finding. Many specimens are examined each day in the cytopathology laboratory, and such deposits had never been previously observed, regardless of the type of needle used for sampling (eg, pleural, peritoneal, or cerebrospinal fluids; peripheral lymph node or skin nodule; tracheobronchial needle aspiration). This study was designed to define the nature of this foreign material and to identify its origin. The blind review of 141 cytospins clearly demonstrated that particles were only observed on EBUS-TBNA samples and not on conventional transbronchial needle aspiration samples. We, therefore, focused our investigations specifically on ViziShot needles, as these particles may interfere with pathologic interpretation by cytopathologists (false diagnosis of anthracnosis), and we also wanted to find a scientific explanation for these findings. This study confirmed that the particles released were metal alloys used in the manufacture of the needles (iron, titanium, nickel, and chromium) and that these EBUS-TBNA ViziShot needles are potential contaminants of aspirated lymph nodes. The hypothesis of poor quality control was proposed, especially because, at the time of the study, these needles were the only dedicated EBUS-TBNA needles available.

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COPD and Lung Cancer Linked at a Molecular Genetic Level

To the Editor:

We read with interest the recent editorial by Strange (December 2010),¹ who suggests that with a greater understanding of COPD may come a greater understanding of lung cancer biology. Here, we review evidence from recently published genetic epidemiologic studies that supports this view by showing COPD and lung cancer are linked at a molecular genetic level.

In 2009, we showed that COPD affects between 50% and 50% of those diagnosed with lung cancer (depending on diagnostic criteria), more than sixfold that seen in smokers with comparable smoking histories randomly recruited from the community.² Recent advances in genetic epidemiology have identified genetic loci implicating three genes in the development of COPD: the nicotinic acetylcholine receptor gene (α4/α7 subunits) on chromosome 15q25 (CHRNA3/5) (susceptibility effect), the hedgehog interacting protein on chromosome 4q31 (HHIP) (protective effect), and the family with sequence similarity 13 member A gene on chromosome 4q22 (FAM13A) (protective effect). Although these genes were first identified through genome-wide association studies involving thousands of subjects, their dual role in COPD and lung cancer was established in a case-control study wherein these genetic associations were examined in smokers with normal lung function, those with COPD (GOLD [Global Initiative for Chronic Obstructive Lung Disease] 2+ criteria) and those with histology-confirmed primary lung cancer (subgrouped by COPD subphenotype).³ Using this approach, we found the CHRNA3 gene, originally associated with an increased risk of lung cancer, was also associated with an increased risk of COPD;⁴ and the HHIP and FAM13A genes originally associated with reduced risk of COPD were independently associated with a reduced risk of lung cancer.⁵ These findings provide compelling evidence that COPD and lung cancer are directly linked at a molecular genetic level.

Importantly, CHRNA3/5 subunits are expressed throughout the bronchial epithelium, are activated by nicotine, and appear to modulate pulmonary inflammation.² Similarly, HHIP is expressed on bronchial epithelium and modulates epithelial repair, smoke-induced epithelial-mesenchymal transition (premalignant transformation), and cigarette smoke-induced oncogenic transformation of bronchial epithelial cells.⁶ Although little is known of FAM13A function, it is also expressed on the bronchial epithelium and sequence analysis indicates FAM13A has Rho GTPase activating protein activity, suggesting both antiinflammatory and tumor suppressor function.³ Although these genetic associations and their biologic effects require confirmation in further studies, these findings provide the first evidence that COPD and lung cancer share pathogenic mechanisms that are mediated by bronchial epithelial (airway) responses to cigarette smoke exposure.

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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.11-0504

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DOI: 10.1378/chest.11-0504

266