
Endobronchial Ultrasound and Extended Roles

Know Thy Limitations

To the Editor:

In the December 2012 issue of CHEST, Kennedy et al1 raise an important point about acknowledging the boundaries of endobronchial ultrasound-guided transbronchial fine-needle aspiration (EBUS-TBNA), citing infectious complications after sampling a nonsubsternal thyroid nodule. There are a number of issues to consider.

The first is infection. Proximity to the vocal cords is likely to be an issue with oropharyngeal contamination. Substernal thyroid nodules, and avoidance of more invasive surgical excision or mediastinoscopy, may well be a better indication for an extended role of EBUS-TBNA, but more data are needed to clarify this.2 The risks for infection in this scenario may be reduced by greater distance from the vocal cords (although the EBUS-TBNA needle is not sterile), and this might better justify EBUS-TBNA over surgical exploration.

Second, what is the best test in this situation, and who is the best trained person to do it? Radiologists (especially those with a subspecialist interest in head and neck) are very adept at sampling thyroid nodules accessible via ultrasound-guided fine-needle aspiration under asepsis. Moreover, it is somewhat harder to justify complications when doing a procedure that someone else is more optimally trained for.

The third issue is training. The learning curve for EBUS-TBNA is notoriously idiosyncratic and longer than previously thought, even for experienced bronchoscopists.3 Adding in extended roles for EBUS-TBNA introduces a need to consider this in curriculum requirements and training standards. Such roles have not been addressed currently in the UK national guidelines for EBUS-TBNA.4 As another example, a few EBUS-TBNA operators perform transesophageal endoscopic ultrasound (EUS) with bronchoscope-guided fine-needle aspiration (EUS-B-FNA)5 6 using the endobronchial ultrasound scope when full-blown EUS is not available. This also requires competency with esophageal intubation, is not covered in the EBUS-TBNA curriculum, and is not a substitute for complete EUS-guided fine-needle aspiration (EUS-FNA). Moreover, it has been suggested by some that such a procedure might be better performed by experienced EUS operators.3 Again, it might be harder to justify complications when doing EUS-B-FNA as opposed to EUS-FNA by a trained EUS operator. As a counter argument, however, there may be occasions where EUS-FNA is not available (given its considerable cost) and a trained EUS-B-FNA operator could perform this as an alternative if only endobronchial ultrasound equipment is available.

In summary, there is a natural inclination, as with any innovative technology, to extend the roles of EBUS-TBNA with time, but this needs to be recognized in EBUS-TBNA curricula and training programs. Careful consideration in the light of existing and new data is required as to what extended roles are reasonable and which of these roles are perhaps better performed by other specialist operators; that is, know thy limitations.

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Relationship Between Pulmonary Emphysema and Renal Function in Smokers

To the Editor:

We read with interest the article by Chandra et al1 in CHEST (September 2012). These authors reported that in smokers, more severe emphysema is associated with kidney dysfunction independent of the common risk factors for kidney disease. They further
noted that no prior study has investigated the relationship between kidney function and emphysema and that the mechanisms of kidney dysfunction in patients with emphysema need further investigation.

Chandra and colleagues studied 508 cases, assessing the severity of emphysema by CT scan and comparing the results with glomerular filtration rates. However, we note that from Figure 3 of their publication, only 16 patients had an emphysema percentage of ≥20%. In this regard, we would like to call to the attention of the authors and the readership of CHEST a study published in 1988 that one of us (V.L.R.) participated in.

Pratt et al examined the cause of death in a consecutive series of 1,033 autopsies and observed that chronic renal disease is a much less common cause of death in people with emphysema, as compared with those without emphysema (P = .0003). The trend persisted when individuals who died of smoking-related diseases were eliminated from the analysis (P < .0003). When only those cases with discernible emphysema were examined (n = 272), the percentage of emphysema in patients dying of chronic renal disease was significantly lower than in all other causes of death (P < .006).

The percentage of emphysema was assessed by point counting of inflation fixed lung specimens to determine the volume percentage of emphysema. From a mechanistic perspective, Pratt et al proposed that destruction of the pulmonary vascular bed in emphysema reduces the efficiency of conversion of angiotensin I to angiotensin II. This, in turn, could interrupt, or at least ameliorate, the vicious cycle of renal injury and release of renin leading to production of angiotensin, with a resulting increase in BP and further renal injury. Thus, an individual with emphysema might be less likely to progress to fatal end-stage renal disease. These observations are also consistent with epidemiologic studies that have shown that smokers have, on average, a lower BP than that of nonsmokers. This, in turn, could interrupt, or at least ameliorate, the vicious cycle of renal injury and release of renin leading to production of angiotensin, with a resulting increase in BP and further renal injury. Thus, an individual with emphysema might be less likely to progress to fatal end-stage renal disease. These observations are also consistent with epidemiologic studies that have shown that smokers have, on average, a lower BP than that of nonsmokers. What is not contradictory is that in considering the results of each study, one must conclude that further research is required into the development of renal comorbidities in patients with COPD. Both studies suggest that renal disease may be an important contributor to the disproportionate burden of cardiovascular disease and cardiovascular death in patients with COPD.

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Response

To the Editor:

We welcome the comments on our article by Dr Palvisko and colleagues and thank them for drawing our attention to the large autopsy series published in 1988 where the presence of emphysema was predictive of reduced mortality from kidney disease. At first glance, this study may appear to contradict our finding that emphysema is associated with an increased prevalence of mild kidney insufficiency.

However, we believe that this paradox might have a rather uncomplicated explanation. The key difference is that mortality from kidney disease is very different from suffering from mild kidney disease. Epidemiologic studies demonstrate that patients with mild kidney disease do not usually die of end-stage renal disease but, rather, of cardiovascular causes. Therefore, patients with emphysema may have a disproportionately high prevalence of early stage kidney disease but may die preferentially of cardiovascular disease instead of advanced kidney disease.

What is not contradictory is that in considering the results of each study, one must conclude that further research is required into the development of renal comorbidities in patients with COPD. Both studies suggest that renal disease may be an important contributor to the disproportionate burden of cardiovascular disease and cardiovascular death in patients with COPD.

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