reactive oxygen metabolites production in the pleural cavity and their role in the pathogenesis of pleural effusions.

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REFERENCES

Is Endobronchial Ultrasound Necessary for Transbronchial Lung Biopsy in Solitary Pulmonary Nodule?

To the Editor:

I read the article "Endobronchial Ultrasound-Guided Transbronchial Lung Biopsy in Fluoroscopically Invisible Solitary Pulmonary Nodules" by Herth et al (January 2006) with interest. In consideration of the outcomes of this article, several questions arose, as follows:

1. With lesion size being 1.4 to 3.3 cm, why were lesions not seen under fluoroscopy in 54 of 138 patients? Were they entirely indiscernible or somewhat visible?
2. The authors noted that the "suspected area" was approached by a catheter with an ultrasound probe. Was fluoroscopy used in any way during the procedure? Why was the suspected area not investigated initially by obtaining the four to six specimens without endobronchial ultrasound, including cytology?
3. Six apical lesions from both upper lobes were not found to be abnormal by ultrasound. Was this due to the fact that the catheter and ultrasound probe were not able to reach the suspected area?

The design of this study cannot result in any of the following potential recommendations:

1. Replace the conventional method. The new method is better, safer, and more accurate. It should be used in all patients.
2. The new method should be additive to the conventional method. It increased the yield in some circumstances and can be used in selected cases.
3. This new technique is neither able to replace nor to be added to the conventional method.

The technology for diagnosing these lesions has evolved over the past several decades. The major determining factor for diagnostic yield is whether the sampling device can reach the lesion or get close to it, confirming whether the lesion is reached or not by any means beyond fluoroscopy before sampling is of great interest. At least it might have the "ROSE" (rapid on-site cytologic evaluation) effect with lesser specificity.

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To the Editor:

We thank Dr. Wang for his thoughtful comments regarding our recent study (January 2006) showing the benefit of endobronchial ultrasound guidance for the transbronchial biopsy of solitary pulmonary nodules that are not visible on standard fluoroscopy.

In answer to the questions posed, we offer the following comments: the lesions were not visible at all during the procedure, which is certainly a well-known problem for most bronchoscopists. The suspected area was determined as the most likely lesion or get close to it, confirming whether the lesion is reached or not by any means beyond fluoroscopy before sampling is of great interest. At least it might have the "ROSE" (rapid on-site cytologic evaluation) effect with lesser specificity.

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identified. This may represent a technical problem, as all of those lesions were in the apical segments.

Our article convincingly showed that in this particular circumstance the addition of endobronchial ultrasound to conventional bronchoscopy (not the replacement) can be very helpful and certainly can be recommended. It avoids aborting an otherwise nonpromising bronchoscopy by providing an acceptable yield, does not expose the patient to unnecessary radiation, and is less invasive than primary surgical procedures.


REFERENCES

To the Editor:

Hamdulay et al3 described two similar cases of severe reversible cardiac depression, temporarily related to exposure to chemotherapy agents for the treatment of lymphoma or prior to haploidentical bone marrow transplantation. Both cases required continuous venovenous hemofiltration, which had been reported to result in the reversal of septic shock and hemodynamic improvement over time because of the plasma clearance of myocardial depressant cytokines. It could be argued that the hemodynamic recovery witnessed was not related to insulin-glucose infusion but was explained by time-dependent plasma clearance of inflammatory cytokines because of the earlier initiation of continuous renal replacement therapy.

There are several studies that have indicated that indiscriminate intensive insulin therapy to maintain a blood glucose level at <6.1 mmol/L (110 mg/dL) can result in attributable mortality. A large randomized controlled trial2 in patients with acute myocardial infarction reported that insulin therapy at a blood glucose level of 7 mmol/L (126 mg/dL) increased the mortality rate to 8.3% (control mortality rate, 6.6%; p = 0.045). Murcia et al2 reported that the cumulative risk for total mortality including cardiovascular mortality and morbidity increased with insulin treatment in diabetic patients with acute myocardial infarction and left ventricular failure. In a recent study by Van den Berghe et al6 intensive insulin therapy in patients with a short length of stay in the ICU and low severity of illness had a much higher mortality rate (27%) compared to patients receiving conventional insulin therapy (19%) [i.e., a relative increase in mortality of 42%; p = 0.045]. The premature and indiscriminate use of intensive insulin therapy in the ICU, which is based on 2004 recommendations without robust scientific evidence, may have resulted in preventable death across the United States. The early resolution of stressors related to the acute illness and minimizing the iatrogenic interventions that exacerbate hyperglycemia rather than prescribing intensive insulin therapy in critically ill patients is the safest method for improved glycemic control and survival.

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Dr. Rady has no affiliations or financial involvement with any organization or entity with a direct financial interest in the subject matter or materials discussed in the article.

REFERENCES


To the Editor:

We thank Dr. Rady on his comments regarding our review on glucose-insulin and potassium infusions in septic shock.1 However, we disagree on his suggestion that the hemodynamic improvement that occurred in our patients could be attributed to the continuous veno-venous hemofiltration (CVVH).

CVVH has been widely used for the treatment of critically ill patients with acute renal failure, and the effects of CVVH on inflammatory responses have been aggressively investigated.2–5 Although circulating inflammatory cytokines were removed by ultrafiltration and adsorption, studies failed to show a decrease in plasma cytokine levels,2,4 even with an aggressive high-volume hemofiltration.5 Having said that, high-volume hemofiltration can significantly improve hemodynamic instability and decrease the vasopressor dose in septic shock patients.6 Nearly all of our patients with sepsis and renal failure are receiving CVVH, yet such dramatic reductions in...