Questions About Adenosine Deaminase Testing and Drug Choice in an Unusual Presentation of TB

To the Editor:

We read with great interest the case report by Sergew et al (November 2009) wherein they reported a case of TB that presented in an unusual fashion as sepsis and ARDS. There are, in our opinion, a couple of issues to be answered.

First, although the authors have mentioned the role of adenosine deaminase (ADA) in ascitic fluid for the diagnosis of TB as the etiology in the discussion of the case report, we wonder why this simple bedside investigation was not done in the case described. ADA levels in ascitic fluid have been suggested as a useful, noninvasive screening test in the diagnosis of peritoneal TB. Although not diagnostic, ADA levels in serous fluids, when considered in collaboration with the clinical scenario, can guide the clinician to clinch an early diagnosis and start the required anti-TB therapy in time. Second, the standard treatment regimen for a fresh case of TB consists of four drugs: isoniazid, rifampicin, pyrazinamide, and ethambutol. Use of potent second-line drugs such as quinolones and an aminoglycoside (amikacin in this case) at the initial phase is not recommended. Inadequate drug regimens promote the selection of drug-resistant strains, which magnify the threat of drug-resistant TB. As the incidence of multidrug-resistant TB and extensively drug-resistant TB is steadily increasing throughout the world, judicious use of antitubercular therapy is recommended to keep the drug resistance to a minimum.

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Correct Diffusing Capacity of Lung for Carbon Monoxide and Carbon Monoxide Transfer Coefficient in Considering Respiratory Function in Patients With Stable Anorexia Nervosa

To the Editor:

There is another explanation for the low diffusing capacity of the lung for carbon monoxide (DLco) values of patients with moderate-to-severe anorexia reported by Gardini Gardenghi and colleagues in CHEST (November 2009) other than “the progressive enlargement of peripheral lung units without relevant alveolar septa destruction.” Because DLco varies with hematocrit, and patients with anorexia nervosa often have anemia, it is particularly important to adjust predicted DLco and carbon monoxide transfer coefficient (Kco) for hematocrit, which was not done in their study.

The authors mistakenly refer to Kco as “lung diffusion capacity corrected for alveolar ventilation.” In fact, Kco = DLco/VA, where VA is the alveolar volume, which equals the volume of distribution of the tracer gas (usually helium) minus predicted dead space. Kco changes much more with lung volume than does DLco, so Kco does not “correct for VA.” Both DLco and Kco have known changes with lung volume, as would be expected with membrane conduction (DM) varying linearly with VA and blood conduction (θVC) not changing. Because the surface area for diffusion is less at lower lung volume, DLco decreases with lung volume. Because DLco decreases less than lung volume, DLco/VA or Kco increases at lower lung volumes. One can correct DLco (Daco) and Kco (Kaco) for VA. The predicted Daco = predicted DLco (0.58 + 0.42 VAtlc) and Kaco = Kco (0.42 + 0.58(VA/VA+tlc)), where VA is the measured VA and VA+tlc is the predicted VA at total lung capacity (predicted total lung capacity [TLC] minus predicted dead space). Percent predicted Daco equals percent predicted Kaco and provides a good indication of the lung’s diffusion correcting for lung volume.

The data demonstrate a problem with the European Community for Coal and Steel equations for predicted Kco. For control subjects, DLco was 95% predicted and TLC 105% predicted, yet Kco was only 82% predicted. This occurred because of inconsistent equations for predicted Kco and DLco. Instead, predicted Kco (Dlco/VA) should be calculated as predicted DLco divided by predicted VA, with predicted VA equal to predicted TLC minus predicted dead space. Because their patients had normal TLC and should not have elevated dead space, their predicted Kco should nearly equal predicted DLco.

The authors should recalculate their data and report DLco and Kco as percent predicted, adjusting predicted values for hemoglobin. They should also report percent predicted VA. My guess is that anemia will explain some, but not all, of the reduction in DLco. Finally, they should correct DLco and Kco for lung volume, by reporting Daco (DLco as percent predicted adjusted both for hemoglobin and for VA) and Kaco. By adjusting for hemoglobin and properly correcting DLco for lung volume (Daco), their study can provide further evidence for an impairment of gas exchange in patients with moderate-to-severe anorexia nervosa.

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Response

To the Editor:

Regarding our recent article in CHEST (November 2009), 1 the hemoglobin (Hb) of the subjects with anorexia was 13 ± 1 g/dL. The diffusing capacity of lung for carbon monoxide (DLCO) values of subjects with anorexia (and controls) were always corrected, according to the following equation proposed by Cotes et al: DLCO (corrected) = DLCO (observed) × (10.2 + [Hb])/1.7 × [Hb], assuming a membrane diffusing capacity/pulmonary capillary blood volume ratio of 0.7 and a reference [Hb] of 14.5 g/dL. Regrettably, we did not mention this correction (which is automatically performed by the computer every time the entered value of Hb is different from 14.5 g/dL) in the Materials and Methods section of our article.

Dr Johnson is perfectly right, because in the article alveolar volume (VA) is erroneously defined as alveolar ventilation, while it is obvious that VA means alveolar volume (in this case obtained by single-breath helium dilution technique). 2 We are very sorry about making such a mistake in the manuscript. On the other hand, Dr Johnson made the same mistake referring to a paper by Dr Plummer in a previously published letter to the editor in CHEST. 3

There are no doubts that DLCO and the diffusing coefficient of lung for carbon monoxide (KCO) change with VA in opposite and different ways and that these changes are relevant for interpretation of gas transfer in patients with low lung volumes. 4 In our subjects with anorexia and controls, however, VA was 99 ± 10% predicted and 103 ± 8% predicted (ie, of VA at total lung capacity = total lung capacity predicted = anatomical dead space predicted), respectively. Thus, there is no substantial reason to recalculate DLCO and KCO in percent predicted of volume-corrected DLCO and volume-corrected KCO. In fact, the values obtained are similar (DLCO: 74 ± 14% predicted vs 75 ± 15% predicted in subjects with anorexia and 95 ± 9% predicted vs 94 ± 9% predicted in controls; KCO: 66 ± 18% predicted vs 66 ± 15% predicted in subjects with anorexia and 82 ± 11% predicted vs 83 ± 10% predicted in controls). Dr Johnson must admit that neither anemia nor VA can explain the difference in DLCO and KCO between subjects with anorexia and matched controls.

We used the European Community for Steel and Coal equations to give the percent predicted of DLCO and KCO, and we agree that those equations seem less consistent for KCO. This applies, of course, for both groups (controls and subjects with anorexia).

By using the formula suggested by Dr Johnson for calculating predicted KCO, actual KCO amounted to 76 ± 18% predicted for subjects with anorexia and 92 ± 11% predicted for controls, nearly equal to DLCO (as percent predicted) for both groups. Therefore, the presentation of our data could be criticized for KCO in terms of percent predicted, but this does not influence the difference in KCO between subjects with anorexia and matched controls that remains unchanged.

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Morning Rounds Becoming Mourning Rounds?

To the Editor:

In their elaborate study, Afessa et al (December 2009) 5 suggest a relationship between ICU mortality and admission during morning rounds (8:00 AM-11:00 AM). A relatively small number of the patients (7.2%) were admitted during round time. However, these patients differed from the average ICU admission: They had a higher severity of illness, were less likely to be postoperative, and were more frequently admitted to the medical ICU.

Based on standardized mortality ratio (SMR), which is used to compare observed mortality with predicted mortality, the authors conclude that mortality rate during morning rounds is higher than predicted mortality rate. They pose the question whether patient care during round times falls short. We also work at a mixed ICU with 24/7 coverage of inhouse intensivists/fellows and have rounds from 11:00 AM to 1:00 PM. We recognize this type of patient admitted early in the morning, but we do not think that they get

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