son, suggest chest CT. " Prospective studies should address the number of CT scans being ordered to evaluate lesions that are already established by roentgenogram as benign. It is also alarming to us that a CT scan can precede the Gram stain or tuberculosis smears on patients with infectious histories and new infiltrates. Our colleagues at Dartmouth are equally aware of how the advances in diagnostic imaging are not always the best way to "deal with uncertainty." We hope that Dr. DiMarco's important editorial message will hit home before the next ACCP section report on clinical staging of patients with non-small-cell lung cancer comes to press.

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The opinions and assertions contained herein are the private views of the authors and do not necessarily reflect the views of the Department of Veterans Affairs or the Department of Defense.

Increased Adenosine Deaminase Activity in Q Fever Pneumonia With Pleural Effusion

To the Editor:

There is presently a growing awareness about the increased activity of adenosine deaminase (ADA) in pleural effusions that are unrelated to tuberculosis, in spite of the established value of that variable in the diagnosis of this disease. Among the infectious diseases, particularly the atypical pneumonias, we have found a report of one case of psittacosis that showed augmented ADA activity. We present a case of Q fever pneumonia with increased ADA activity levels (> 45 IU/L) in the pleural fluid.

A 55-year-old man was admitted with fever, chills, cough, scarce mucous discharge, and right pleuritic pain for a period of 9 days. He had received treatment with cefonicid for 4 days, without any clinical improvement. Physical examination revealed diminished sounds and the presence of crackles at the right lung base. A standard blood analysis disclosed elevations in the erythrocyte sedimentation rate (82 mm/h [normal, up to 20 mm/h]), serum glutamic oxaloacetic transaminase level (137 IU/L [normal, up to 37 IU/L]), and glutamic pyruvic transaminase level (128 IU/L [normal, up to 40 IU/L]). Arterial blood PaO2 and PaCO2 values were 69 and 32 mm Hg, respectively (normal, 80 and 35 to 45 mm Hg, respectively). A chest film showed air-space consolidation in the right lower lobe and a small pleural effusion. A skin test with 2 U of purified protein derivative was negative.

Ziehl-Nielsen staining and Legionella direct immunofluorescence study of the sputum were also negative, and no growth was seen in the blood cultures. A sample of pleural fluid obtained by thoracocentesis revealed a xanthochromic liquid with biochemical features of exudate and a lymphocyte-prevalent cell count (4 percent neutrophils, 52 percent lymphocytes, 39 percent histiocytes, 5 percent mesothelial cells). The ADA level was 64 IU/L. Bacteriologic stainings of the cultures of the pleural fluid were also negative. Liver ultrasonography was normal. Serology was negative for mycoplasma and Legionella, but the Coxiella burnetii complement fixation immunofluorescence technique showed seroconversion at the third week, with a dilution titer of 1/128.

The patient received therapy with erythromycin. Analytical measurements showed rapid improvement, and a full clinical and radiologic recovery was evident afterward.

The elevation of ADA activity in pleural fluid during Q fever pneumonia might be related to the intracellular behavior of C burnetii, with associated lymphocytic activation. This may extend to other intracellular agents causing pneumonia. The most interesting finding in our case, aside from the lack of previous similar reports, is related to the fact that in regions with epidemic outbreaks and sporadic cases of Q fever, such as the Basque country of Spain, the rate of associated pleural effusion may reach as high as 35 percent. The noncritical interpretation of ADA values, even when considering the potential differences in clinical presentation between Q fever and tuberculosis, may lead to a diagnostic mistake between these diseases.

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Errata


The editors of Chest wish to apologize to Jahan W. de Fijter, M.D., for a typographical error that appeared in his article, "Sepsis Syndrome and Death After Bronchoalveolar Lavage" (Chest 104: 4:1296-97 [Oct]). On page 1296, right-hand column, the following sentence appeared as, "Therefore, the patient underwent a diagnostic BAL, using 2,000 ml of 0.9 percent sterile saline solution at body temperature." It should have read, "... using 200 ml of 0.9 percent sterile saline solution at body temperature."

A Communication to the editor, "Critical Care Board Examination Resources" by Len Scarpinato, D.O. (Chest 1993; 104:1311 [Oct]), refers in the text to a book by Dr. James M. Rippe, entitled Critical Care Medicine; it is actually entitled Intensive Care Medicine, 2nd ed.

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