To the Editor:

We thank Dr. Tashkin for bringing to our attention that indeed some of the quoted studies, particularly the ones performed in his laboratory did take hemoglobin into account in the calculation of the diffusion capacity. Nevertheless, in the remaining studies there is no mention as to whether or not this was performed. We concur with regard to his list of other confounding factors that may be associated with a drop in diffusion capacity in crack users. Whether or not a well-designed control study will ever be carried out on this issue is difficult to say as these patients tend to be unreliable, often using more than one drug, and suffering from other complications of intravenous drug use.

We thank him for bringing this matter to our attention and stand corrected with regard to the statements made regarding the diffusion capacity in the articles from his laboratory.

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Adverse Pulmonary Effects of Mesalamine

To the Editor:

We have read in the 1992 May issue of CHEST the case report described by Reinoso et al (CHEST 1992; 101:1469-71), concerning pulmonary disease associated with the oral administration of mesalamine for ulcerative colitis. We describe a similar case of pulmonary reaction to mesalamine in a patient with Crohn’s disease.

A 60-year-old woman who smoked 25 packs/yr, underwent surgery in 1985 for Crohn’s disease. After few months of therapy, first with steroids and subsequently with sulfasalazine, she began a therapy of mesalamine 1,600 to 2,400 mg/d and had good control of the basic disease.

In January 1994 she began showing dyspnea under limited effort, 38°C, and cough without sputum. Since the symptoms persisted, she was admitted to the hospital. Chest x-ray films showed moderate signs of enhancement of the interstitial tissue. Pulmonary function tests showed a vital capacity of 1.57 L (69%), an FEV1, 0.91 L/s (52%), and a significant hypoxemia (PaO2, 41 mm Hg). Diffusion of carbon monoxide was not carried out owing to the strong dyspnea affecting the patient. The physical examination identified rhonchi in the upper fields and rales in the lower fields; the hemochrome showed 10,500 WBCs, 70% of which were neutrophils, 3% eosinophils, 1% basophils, 2% lymphocytes, and 5% monocytes. Erythrocyte sedimentation rate was 56 mm Hg. C-reactive protein was 1.4 (normal value less than 0.5), while fibrinogen was 743 mg/mL (normal value 150 to 400 mg/mL). The values of sodium, potassium, creatinine, total protein, and urinary sediment were normal, while an increase in alkaline phosphatase was observed (353 U/L) (normal value 90 to 300 U/L).

Bronchoscopy identified a pattern of mucous inflammation with the presence of mucus on the walls. BAL showed the presence of 800 cells/mL 10x3, 40% macrophages, 2% neutrophils, 3% eosinophils, 55% lymphocytes. The search for common germs, fungi, and Koch’s bacillus was negative. Lymphocytes (CD) in blood resulted in CD3+ 73%, CD4+, 44%, and CD8+ 35%. BAL results were 92% CD3+, 56% CD4+, and 31% CD8+. The patient did not consent to a biopsy. High-resolution computed tomography of the lung showed scattered ground-glass patches, with bronchiectasis at parahilar level. After this investigation, the decision was made to interrupt mesalamine to ascertain a correlation with the respiratory problems. After a 2-week interruption, an improvement of cough, dyspnea, PaO2 (66 mm Hg), fibrinogen, erythrocyte sedimentation rate (34), and C-reactive protein (0.3), then normalization of the radiologic pattern at high-resolution computed tomography could be observed. After another week, the patient resumed the mesalamine treatment, with no reappearance of the respiratory symptoms. Two months later, there was a normalization of the vital capacity and FEV1, while PaO2 showed a clear improvement (74 mm Hg).

It is our opinion that mesalamine (if administered for a long time) can cause an adverse pulmonary reaction. This damage is reversible even by interrupting drug administration only temporarily, and it does not appear to recur when a new administration of the same drug is required. Such behavior has already been described1-2 as with the use of methotrexate in rheumatoid arthritis.

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References


Effects of Ipratropium Bromide on Pulmonary Hemodynamics in COPD

To the Editor:

I have read with great interest the article by Ashutosh and colleagues in the January 1995 issue (CHEST 1995; 107;173-78) devoted to the nonbronchodilatory effects of ipratropium bromide. I agree with authors’ conclusions and would like to add some data supporting their views. In the discussion, the authors state that anticholinergic agents “have no effect on pulmonary vessels, do not interfere with the hypoxic pulmonary vasoconstrictive reflex . . . .” No reference, however, was proposed to support this statement. It happens that some years ago, we studied the effects of ipratropium bromide (IB) on pulmonary hemodynamics in COPD patients. That