Kaposi’s Sarcoma of the Tip of the Nose as a Sentinel Sign for Kaposi’s Sarcoma of the Lung

To the Editor:

We have noticed in several of our patients with bronchoscopy-proven pulmonary Kaposi’s sarcoma (KS) the association of a striking, bulbous, tip-of-the-nose KS lesion. To study this association further, we reviewed the records at Thompson Hospital for bronchoscopies in HIV-infected patients from 1992 to 1995, searching for those with a diagnosis of pulmonary KS. Nineteen patients were found to have pulmonary KS diagnosed by classic appearance on bronchoscopy. Average helper T-cell count at the time of bronchoscopy was 106 (range 40 to 220). The predominant race was Hispanic and the average age was 32 (range 25 to 41). Fifteen of these patients also had oral-facial KS noted on the chart, and in 13 of these patients a prominent tip-of-the-nose KS lesion was noted. Only six patients lacked the tip-of-the-nose lesion and yet had pulmonary KS. Of these six patients, only four did not have associated oral-facial KS.

Our review would suggest that in our patient population, a tip-of-the-nose lesion is commonly associated with pulmonary KS and may be a sentinel sign for the same. We would welcome the comments and experiences of others with regard to this peculiar lesion.

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Communications to the Editor

Pulmonary Toxicity During Granulocyte Colony Stimulating Factor Administration and Neutrophils

To the Editor:

We read with interest the article "Impairment in Gas Exchange After Granulocyte Colony Stimulating Factor (G-CSF) in a Patient With the Adult Respiratory Distress Syndrome" by Schilero and colleagues (CHEST 1995; 107:276-78). Interstitial pneumonitis also developed during G-CSF administration in two of our patients whose malignant lymphomas were being treated with chemotherapy. In addition, transient pulmonary dysfunction developed in another elderly patient who was receiving G-CSF. Schilero et al detected the early signs of exacerbation in pulmonary function during G-CSF, and they promptly discontinued the administration of G-CSF. We1,2 and others3 continued G-CSF administration, and in some cases, administered pulmonary toxic drugs like bleomycin sulfate on the same day as G-CSF. In addition to recovery of neutropenia, phagocytic activity of neutrophils was enhanced in our patients during development of interstitial pneumonitis, which support the potentiation of lung disorders by G-CSF-activated neutrophils as discussed by Schilero et al. There has been no discussion by Schilero et al or others4-6 on lymphocytes in pulmonary disorders potentiated by G-CSF; however, we now believe that lymphocytopenia, which was present in our patients, and/or dysfunction of lymphocytes in these patients may participate in the development of pulmonary dysfunction with G-CSF-activated neutrophils. In a recent controversial discussion on pulmonary disorders and G-CSF, the incidence of pulmonary toxicity in patients receiving G-CSF was reported not to increase in placebo-controlled trials in which bleomycin sulfate was administered. Furthermore, G-CSF is reported to be related to, but not the cause of, an increase in bleomycin pulmonary toxicity. As very early onset of pulmonary toxicity was demonstrated by Schilero et al, we suggest that compromised hosts, like the elderly and infants with malignancies receiving chemotherapy or those with multiple-organ failure who underwent orthotopic liver transplantation (CHEST 1995; 107:276-78), are more sensitive to the G-CSF-induced pulmonary toxicity.

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