Respiratory Medicine (1995) **89,** 237–239

Letters to the Editor

Dear Editor

Atypical localization of pulmonary infiltrates in sarcoidosis

Sarcoidosis is a systemic granulomatous disease of unknown aetiology that most commonly involves the lung (1). More than 90% of patients with sarcoidosis will show abnormalities on chest X-ray films sometime during the course of their disease (1). The common roentgenographic presentations of pulmonary sarcoidosis are well known, and include lymph node enlargement and/or diffuse, parenchymal interstitial infiltrates (2). We report an unusual case of thoracic sarcoidosis which showed bilateral, peripheral, patchy parenchymal infiltrates of the lung, without any mediastinal or hilar lymphadenopathy.

A 27-year-old female patient was admitted to our outpatient clinic with an abnormal chest X-ray film which had been taken during a routine check-up examination. They started anti-tuberculosis therapy with three drugs in the other hospital to which she had referred before. When there was no change in the chest X-ray film after 2 months of treatment, she referred to our department for further examination.

Physical examination revealed normal findings. Chest radiograph showed bilateral, diffuse, patchy infiltrations being more prominent in the peripheral regions. Smear and culture of the sputum were negative for acid-fast bacilli. Erthyrocyte sedimentation rate, complete blood count (including differential count of the white blood cells), serum electrolyte concentrations and kidney and liver function tests were in normal ranges. ACE concentration was 5 UI^{-1}. Arterial blood gases revealed; pH 7·43, PCO_2 35 mmHg, PO_2 80 mmHg and HCO_3 24 $mEql^{-1}$. Pulmonary function tests showed mild small-airway obstruction, restriction and decrease in the diffusion capacity of the lung. Computed tomography of the thorax disclosed diffuse, bilateral, multiple infiltrative lesions being more prominent at the periphery of the lung. Gallium scanning of the lung was normal. Thoracotomy was performed to obtain a specimen, which on examination revealed granulomatous disease consistent with sarcoidosis. We started to treat the patient with corticosteroid drugs, and noticed improvement in her chest X-ray films from the end of the first month of therapy, at the monthly follow-up visits. She developed spontaneous pneumothorax on the third month of treatment, for which she has been treated in our radiology department by tube drainage using a Heimleich valve. She recovered in 24 h and is still receiving tapered doses of corticosteroid therapy.

Isolated, peripheral parenchymal infiltrates in the lung are a rare initial manifestation of sarcoidosis (3). Glazer *et al.* (1) mentioned eight patients having infiltrates of this type, all of whom had hilar or mediastinal adenopathy. Scott and Pinstein (4) described a case with a chest X-ray film which showed mainly peripheral pulmonary infiltrates without any mediastinal or hilar adenopathy. Also, Judson *et al.* (3) presented a patient with chest CT scans showing isolated, peripheral pulmonary infiltrates. As a result, to our knowledge, our case is the third sarcoidosis case in which the chest CT scans showed isolated, peripheral pulmonary infiltrates.

G. Kisacik, L. Çöplü, Y. Karakoca and A. U. Demir
Department of Chest Diseases, Hacettepe University Hospital Ankara, Turkey
7 September 1994

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Dear Editor

Necrotizing vasculitis and exacerbation of psoriasis after granulocyte colony-stimulating factor for small cell lung carcinoma

Granulocyte colony-stimulating factor (G-CSF) is widely used as an adjunct to chemotherapy in patients with small cell lung cancer (1). We report on a case simultaneously presenting two cutaneous

0954-6111/95/030237+03 $08.00/0

complications, necrotizing vasculitis and exacerbation of a pre-existing psoriasis, after use of G-CSF for treatment of a small cell lung cancer.

A 45-year-old woman was treated for a limited small cell lung cancer with a chemotherapy regimen consisting of cyclophosphamide (1 g m^{-2}), doxorubicin (50 mg m^{-2}), cisplatin (100 mg m^{-2}), and etoposide (200 mg m^{-2}). Because the patient had febrile neutropenia after the first course of chemotherapy, she was treated with 300 μg day^{-1} of G-CSF subcutaneously on days 3–10 following the end of the second course of chemotherapy. Four days later, she developed palpable purpuric lesions on the legs characteristic of a necrotizing vasculitis, and we observed exacerbation of psoriasis over thorax and legs. No biopsy was done as clinical aspects were rather characteristic.

There were no antibodies detected for nuclear cytoplasm antigens, hepatitis B and C viruses or human immunodefiency virus. Cryoglobulinemia and proteinuria were absent. The leucocyte count was $3{\cdot}5 \times 10^9\,l^{-1}$ with $2{\cdot}8 \times 10^9\,l^{-1}$ neutrophils; platelets were $211 \times 10^9\,l^{-1}$. Treatment with topical corticosteroid resolved psoriasis within 10 days, and purpuric lesions disappeared in about the same time. Considering the relative severity of the cutaneous lesions, the patient did not receive further G-CSF. Two additional courses with cisplatin and etoposide were administered simultaneously with thoracic radiotherapy, without recurrence of the cutaneous adverse effects.

Large reports of G-CSF treated patients with small cell lung cancer have been reported without cutaneous adverse effects (1,2). However, one patient with small cell lung cancer developed pyoderma gangraenosum during G-CSF therapy, and another patient showed necrotizing vasculitis with granulocyte-macrophage stimulating factor (3,4). Six other cases of cutaneous vasculitis have been reviewed in patients with various tumours, treated with leucocyte colony-stimulating factors. The eruptions are unrelated to the primary diseases and developed after 1 or 2 weeks of therapy. It is interesting that exacerbation of psoriasis has been noted in a patient with aplastic anemia subsequent to granulocyte macrophage-stimulating factor (5). To our knowledge, our case report is the first with both vasculitis and psoriasis. The mechanisms by which G-CSF exacerbate pre-existing psoriasis or induce vasculitis remain unknown. Various cytokines may be an indirect cause (tumour necrosis factor, interleukin-1) (6). We conclude that patients and physicians should be aware of the development of various inflammatory processes during G-CSF treatment. Patients with pre-existing psoriasis should be treated with caution, while receiving G-CSF.

L. J. COUDERC*, B. PHILIPPE*, N. FRANCK†,
E. BALLOUL-DELCLAUX* AND
M. LESSANA-LEIBOWITCH†
**Service de Pneumologie*
Hopital Foch
Suresnes
†*Service de Dermatologie*
Hopital Tarnier
Paris, France.
14 September 1994

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Dear Editor

Gallbladder wall thickening as a sign of pulmonary embolism

Alvarez-Sala *et al.* described a patient with acute pulmonary embolism, in whom thickening of the gallbladder wall was shown by abdominal sonography (1). The authors suggest that this could be an initial sign of increased cystic and systemic venous hypertension, due to right cardiac failure, and hence to acute pulmonary embolism. The validity of the sign, however, is not established, since other cases have not been reported (2). We wish to report a case of acute pulmonary embolism we have observed, in which this sign was present.

A 92-year-old woman was admitted because of vomiting and tachycardia of several days duration. She denied dyspnoea and thoracic or abdominal pain. The remaining history was unremarkable. Blood pressure was 120/90 mmHg, heart rate was