



**Fig. 1.** The patient on admission. (A) Erythema and oozing crusts in the face and on the chest. (B) Papulo-pustules on the dorsal side of the hands.

5 mg prednisolone daily and was later followed up monthly at the out-patient clinic. Tetracyclin 1 g daily was continued for the next 3 months. When the patient got pregnant a few months later, she had a flare-up of her rosacea, but only in the facial area.

#### DISCUSSION

The patient described had extrafacial lesions, which usually points in the direction of acne fulminans. Plewig et al. (2) studied 20 patients with clinical rosacea fulminans and reported that extrafacial lesions were isolated and few, if present. But extrafacial lesions have been described by many authors in patients with ordinary rosacea. The patient described had no comedones, and she had facial erythema, telangiectasia and papules before and after the aggravation. The disease activity was probably too great for tetracycline to show any quickly improving effect the period before admission. Due to the possibility of infection, prednisolone was not increased at hospital

admission. Huge leukocytosis (pseudoleukemoid reaction) is reported in other severe dermatoses, like acne fulminans and pustular psoriasis. A methotrexate drug eruption seems unlikely in the present case, since she had a new outburst of facial rosacea in the year after the hospital stay. It is, however, possible that methotrexate caused a flare-up of an "underlying" rosacea.

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### Disseminated Morphea in Small Cell Lung Cancer

Sir,

A 34-year-old woman, non-smoker, presented with the complaints of cough and hemoptysis since 1 month. Physical examination and routine laboratory investigations were normal. In the chest radiograph and computerised tomo-

graphy, a mass was observed in the right hilus, with mediastinal invasion. Neuron-specific enolase was elevated at 170 ng/ml (normal, 0–12.5). Bronchoscopy revealed a mass obliterating the lower lobe segment of the right main bronchus. The pathologic diagnosis was anaplastic small cell type lung cancer.

After complete staging work-up, she was accepted as having limited disease and a chemotherapy regimen consisting of cyclophosphamide-adriamycin-etoposide was started. After six cycles, a complete remission was achieved and radiotherapy to thorax was applied.

Two months after completion of the radiotherapy, multiple wax-colored oval and round plaques with a smooth shiny surface appeared all over the trunk (Fig.). A skin biopsy confirmed the clinical diagnosis of morphea. The dermis was thickened, with thick collagen bundles, which replaced largely subcutaneous fat cells and entrapped the sweat glands. The fibroblasts were slightly increased and a mild perivascular mononuclear inflammatory cell infiltrate was detected. The epidermis was slightly atrophic. Antinuclear antibodies were negative and no sign of systemic involvement with scleroderma was found.

In the ensuing weeks, the patient's malignancy rapidly progressed, with bone, liver and bilateral suprarenal gland metastasis. The skin lesions remained unchanged. Chemotherapy with ifosfamide, mesna and etoposide was initiated, with no clear response. The patient's condition progressively deteriorated and she expired 4 months after the beginning of the second chemotherapy.

In our patient, the emergence of sclerotic skin changes in the absence of systemic involvement approximately 1 year after the diagnosis of lung cancer suggested a probable paraneoplastic manifestation (1). Tumor-made substances, such as hormones, cytokines, biologically active proteins and their precursors, may activate dermal fibroblasts and cause sclerotic cutaneous lesions. Small cell lung cancer is particularly associated with the induction of a variety of paraneoplastic conditions (2). Paraneoplastic skin lesions resembling scleroderma have rarely been detected during the course of lung cancer (3), although it is well recognised that primary lung cancers may arise in patients with scleroderma. The reported cases of lung cancer have appeared in established scleroderma, usually in the presence of extensive pulmonary fibrosis. However, morphea as a paraneoplastic manifestation, appearing after the diagnosis of lung cancer, is very unusual.

The underlying basis of localised scleroderma is still unclear. Some chemotherapeutic agents such as bleomycin may cause fibrosis (4). But none of the drugs administered to our patient is known to induce skin fibrosis. Radiation has been identified recently as an etiologic factor of cutaneous sclerosis (5). Trauma of radiation injury was incriminated as the triggering mechanism for morphea. In these reports, morphea appeared in the irradiated areas or nearby, so it was suggested that irradiation might be the factor provoking sclerotic skin changes. This mechanism may also be applicable to our patient, as she received radiotherapy of the chest. However, the presence of similar lesions scattered in the inguinal and flank regions indicates additional possible explanations for the development of localised scleroderma.

Morphea may represent an additional distinct paraneoplastic manifestation of small cell lung cancer. Radiation may also have played a role, or both of these mechanisms may be operative in the development of morphea.



Fig. 1. Multiple, wax-coloured oval and round plaques on the trunk.

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