Systemic sclerosis and organic solvents: early diagnosis in industry

In 1957 Rein¹ and Walder² described the first cases of systemic sclerosis (SS) after contact with organic solvents. An increasing number of cases have subsequently been reported, mostly frequent involving aliphatic hydrocarbons (vinyl chloride, perchoroethylene, trichloroethylene),³ and 20 cases related to aromatic hydrocarbons (benzene, toluene, xylene, white spirits and diesel).³ Aromatic amines (cyclohexylamine and m-phenylene-nediamine) and formaldehyde derivatives were involved in about 10 cases.³ ⁴

Our 56 year old patient developed SS with skin, lung and pericardial involvement after intense and prolonged exposure to toluene (aromatic hydrocarbon), heptane (aliphatic hydrocarbon), dimethylbuthylphenylida-mine, (aromatic amine), and octaphenyl formaldehyde (formaldehyde hydrate), cutaneously and by inhalation. Exposure to nonchloronated hydrocarbon and sulphated substances was also assessed.

For 23 years he had worked in the rubber transformation division of a tyre factory. However, a period of eight years he developed progressive thickening of the skin of the fingers, Raynaud’s phenomenon and progressive effort dyspnoea. He was first seen by us in May 1981 because of dysnoea on minimal exertion.

Clinical findings on admission were sclerodactyly, mild generalised cutaneous sclerosis (more intense on both shoulders and some on the lower limbs) and telangiectasia. Despite the trunk and palm telangiectasia. A trunk skin biopsy showed a severe sclerosis of the dermal collagen, with few fibroblasts, sclerosis of the sweat glands and subcuticular dermis, with poor vascularity and septa thickening of subcutaneous tissue. Fine crackles were present in both lung bases. A chest radiograph showed cardiomegaly. Echocardiography revealed a small pericardial effusion and enlargement of the right cavities with mild tricuspid insufficiency that yielded a pulmonary arterial hypertension of 46 mm Hg. Cardiac catheterisation showed it to be pre-arteriolar. Respiratory function tests showed moderate-severe restriction, (FEV1:1960 cc-59%; VC-IN:2:260 cc-52%) alteration on diffusing capacity (TLCO 57, 5%) and arterial gasometry with hypoxaemia (PPO2 73 mm Hg) and increase of the alveolar-arterial O2 gradient (A–aO2 = 43) compatible with moderate lung fibrosis. A radiograph of the right hand showed small subcutaneous calcification in one digit. A barium swirl only showed reflux. Renal function was normal. Antinuclear antibodies were positive at a 1:400 titre with a nucleolar pattern. Anticentromere and antiScI 70 antibodies were negative.

The patient was treated with nifedipine (30 mg/day) and prednisone (1 mg/kg a day initially with subsequent tapering). A few months later he complained of dysnoea at rest, and clinical signs of right sided heart failure. PAP control by echocardiography (Doppler) had raised to 80 mm Hg. He died 12 months after diagnosis from cardio-respiratory failure. Renal function remained normal until his death. Necropsy was refused.

SS is a multisystem disorder characterised by an overproduction of collagen with involvement of the skin, blood vessels and visceral organs.

Over the past 25 years there have been increasing reports of environmentally induced SS.³ Organic solvents penetrate the skin, can be inhaled, and may produce metabolic changes in many organs, due both to a direct toxic effect and a possible immunogenenic susceptibility to SS.³ ⁴ In most cases, avoiding exposure does not result in clinical remission.¹ ⁵,³ ⁶ Nevertheless, early diagnosis should be achieved. Raynaud’s phenomenon is the first symptom in up to 70% of patients with SS.⁷ We suggest that a review of solvent exposure should include an anamnetics and occupational history test followed up workers from relevant industries. In patients in whom Raynaud’s phenomenon is present a complete physical examination, a nailfold capillaroscopy and a selective autoimmune study (anticentromer and anti-ScI 70 antibodies) should be carried out,⁸ and further exposure avoided if positive.

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1 Rein L W. Sclerodermae durch trichloroethylen einwirkung. Haut Lfg 1957; 88: 678.

Isospora belli reactiv arthritis in a patient with AIDS

Isospora belli has been recognised as an opportunistic protozoan in patients with the acquired immunodeficiency syn

drome (AIDS).³ ⁴ Parasitic infestation of the gastrointestinal tract has been previously reported as a possible cause of seronegative arthritis.³ ⁴ The common features were eosino-

philia, asymmetric oligoarthritis affecting large joints of the lower limbs, and full improvement after elimination of the parasite.³ ⁴ We report a case of reactive arthritis due to infestation by I belli in a patient with AIDS.

The patient, a 57 year old white woman, with human immunodeficiency virus (HIV) was infected by heterosexual transmission. In 1991, she started having dysarth-
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