Systemic sclerosis and organic solvents: early diagnosis in industry

In 1957 Rein and Walder described the first cases of systemic sclerosis (SS) after contact with organic solvents. An increasing number of cases have subsequently been reported, the most frequent involving aliphatic hydrocarbons (vinyl chloride, perchloroethylene, trichloroethylene), and 20 cases related to aromatic hydrocarbons (benzene, toluene, xylene, white spirits and diesel). Rein and Walder yielded aromatic hydrocarbons (dimethylbuthylphenyldiameine) and formaldehyde derivatives were involved in about 10 cases. 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), dimethylbuthylphenyldiameine, (aromatic amine), and octophenol formaldehyde (formaldehyde derivates), cutaneously and by inhalation. Exposure to nonchloronitrohydrocarbons and substituted substances was also assessed.

For 23 years he had worked in the rubber trade, manufacture of a tyre factory. Now, 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 1991 because of dyspnoea on minimal exertion.

Clinical findings on admission were sclerodactyly, mild generalised cutaneous sclerosis (more intense on both shoulders and some on the face) and some Raynaud phenomenon. Despite his thick trunk and pulsate telangiectasia. A trunk skin biopsy showed a severe sclerosis of the dermal collagen, with few fibroblasts, sclerosis of the sweat glands and subdermiclemic debris, with poor vascularity and septa thickening of subcutaneous tissue. Fine cracks 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-artericular. 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 (PaO2 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 swallow only showed reflux. Renal function was normal. Antinuclear antibodies were positive at a 1/400 titre with a nuclear pattern. Anticentromere and antiScI 70 antibodies were negative.

The patient was treated with nifedipine (30 mg/day) and prednisonone (1 mg/kg a day initially with subsequent tapering). A few months later he complained of dyspnoea 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. 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 immunogenetic susceptibility to SS. In most cases, avoiding exposure does not result in clinical remission. 7 Nevertheless, early diagnosis should be achieved. Raynaud’s phenomenon is the first symptom in up to 70% of patients with SS. 8 We suggest that a review of solvent exposure should include an anamnestic data collection and a search for signs of occupational overexposure from workers relevant to industries. In patients in whom Raynaud’s phenomenon is present a complete physical examination, a nailfold capillaroscopy and a selective autoimmunological study (anticentromer and anti-Scl 70 antibodies) should be carried out, and further exposure avoided if positive.

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11. Isospora belli reactive arthritis in a patient with AIDS

Isospora belli has been recognised as an opportunistic protozoan pathogen in patients with the acquired immunodeficiency syndrome (AIDS). 9 Parasitic infestation of the gastrointestinal tract has been previously reported as a possible cause of seronegative reactive arthritis. 9 The common features were eosinophilia, asymmetric oligoarthritis affecting large joints of the lower limbs, and full improvement after elimination of the parasite. 9 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 Raynaud’s phenomenon. Four months later she developed inflammatory arthritis affecting both hands and wrists, accompanied by morning stiffness. A year before her admission her husband, who had been diagnosed with AIDS died of pneumocystis carinii pneumonia complications. On physical examination, she had peripheral lymphadenopathy and xanthelasma, but no other cutaneous manifestations. Laboratory tests revealed no evidence of autoimmune disease. In the peripheral blood picture, the erthrocyte sedimentation rate (ESR) of 38 mm/hour, a haemoglobin of 12:1 g/dl, a white blood cell (WBC) count of 5·6 × 10^9/l with an eosinophlic count of 0·56 × 10^9/l and platelets of 260 × 10^9/l. Blood chemistry and urinalysis findings were within normal limits. Results of the test for IgM rheumatoid factor, antinuclear antibody and HLA-B27 were negative. Anti-HIV antibody, performed by ELISA assay, was positive. After 2 months, the Western blot analysis. She had a CD4:CD8 ratio of 0·13 and an absolute CD4 count of 264 cells/mm^3. Radiographs of the chest and upper limbs showed some areas of fibrosis. Before the development of polyarthritis, 12 months after the infestation of parasite, 3 she was isolated from her stool samples an organism identified as Isospora belli. The patient was treated with oral trimethoprim (160 mg) and sulphamethoxazole (800 mg), given four times daily for 10 days and then twice daily for three weeks and diclofenac, 50 mg given three times a day. Three days later, her arthritis progressively improved and diarrhoea disappeared. Despite the control of the reactive arthritis, arthritis did not recur. Unfortunately, this patient died in June 1993 due to AIDS.

Reactive arthritis has been reported in association with a number of enteric parasitic pathologies including isospora dientamoeba and cryptosporidium. Chronic enteric infections with coccidial parasites have been associated with immunodeficient patients. Cryptosporidium and I belli have been implicated as a cause of chronic diarrhoea in patients with AIDS. Reactive arthritis has been reported in HIV-infected patients mainly in homosexual men. After parasite enteric infection has been described, seronegative oligo- or polyarthritis asymmetric, additive or migratory with predominant involvement of joints of the lower limbs, but upper limb joints can be affected.

Our patient may have had reactive arthritis after enteric infection with I belli. She developed symmetrical polyarthritis without extra-articular features of systemic rheumatoid arthritis, involving both wrists and hands, accompanied by morning stiffness resembling rheumatoid arthritis. The diagnosis of parasite reactive arthritis is suggested by: eosinophilia, blood chemistry profile, and the temporal sequence that this arthritis was triggered by I belli infestation, and rheumatic manifestations, which improved after trimethoprim-sulphamethoxazole therapy. However, the mechanisms of reactive
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