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 since been reported, mainly involving aliphatic hydrocarbons (vinyl chloride, perchloroethylene, trichloroethylene), and 20 cases related to aromatic hydrocarbons (benzene, toluene, xylene, white spirits and diesel). Aromatic amines (cyclohexylamine and m-phenyl-enediamine) and formaldehyde derivatives were involved in about 10 cases. 1-7

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), dimethyldihexylidiamine, (aromatic amine), and octylphenol formaldehyde (formaldehyde derive), cutaneously and by inhalation. Exposure to nonchlorinated hydrocarbon and sulphated substances was also assessed.

For 23 years he had worked in the rubber transcription department of a tyre factory. After 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 forehead and cheeks). Despite harsh upper 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 subcutaneous dermis, with poor vasculosity and septa thickening of subcutaneous tissue. Fine cracks were present in both lung bases. A chest radiograph showed cardiomegaly. Echocardiography revealed a small peri- cardiac 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-artrial. Respiratory function tests showed moderate-severe restriction, (FEV1:1960 cc-59%; VC-IN:2-260 cc-52%) alteration on diffusing capacity (TTCO 57, 5%) and arteriolar hyperoxygenation by 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. Anticientromere 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 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 due 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. 1 Organic solvents penetrate the skin, can be inhaled, and may produce toxic metabolic changes in many organs, due both to a direct toxic effect and a possible immunogenetic susceptibility to SS. 1,8 In most cases, avoiding exposure does not result in clinical improvement. Nevertheless, early diagnosis should be achieved. Raynaud’s phenomenon is the first symptom in up to 70% of patients with SS. 1 We suggest that a review of solvent exposure should include an immunological research in the annual check up of workers from relevant industries. In patients in whom Raynaud’s phenomenon is present a complete physical examination, a naijof capillaroscopy and a selective autoimmune study (anticientromer and anti-ScI 70 antibodies) should be carried out, and further exposure avoided if positive. 2

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Isospora bellii reactive arthritis in a patient with AIDS

Isospora bellii has been described as an opportunistic protozoan pathogen in patients with the acquired immunodeficiency syndrome (AIDS). 1,2 Parasitic infestation of the gastrointestinal tract has been previously reported as a possible case of seronegative arthritis. 3 The common features were eosinophilia, asymmetric oligoarthritis affecting large joints of the lower limbs, and full improvement after elimination of the parasite. 3 We report a case of reactive arthritis due to Isospora bellii infection 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 her first diarrhoea. 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 revealed activitis of the reticuloendothelial system and the second, third and fourth metacarpophalangeal (MCP) joints in both hands. There were no aphthous ulcerations, skin rashes or evidence of conjunctivitis.

In our clinical test we measured the erythrocyte sedimentation rate (ESR) of 38 mm/hour, a haemoglobin of 121 g/dl, a white blood cell (WBC) count of 5·6 × 10^9/l with an eosinophilic count of 0·56 × 10^9/l and platelet count of 620 × 10^9/l. Blood chemistry and a urine analysis were within normal limits. Results of the test for IgM rheumatoid factor, anti nuclear antibody and HLA-B27 were negative. Anti-HIV antibody, performed by ECLUSA assay, was positive, and the Western blot analysis. She had a CD4/CD8 ratio of 0-13 and an absolute CD4 count of 264 cells/mm³. Radiographs of the chest and abdomen were normal. After a few days before developed polyarthritis, we isolated from her stool samples an organism identified as Isospora beli. 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 dicrofenc, 50 mg given three times a day. Three days later, her arthritis progressively improved and diarrhoea also. Described above. Nevertheless, after the reaction 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 pathogens including I. bellii. 4,5 More recently Blastocystis hominis 6 and Cryptosporidium. 7 Chronic enteric infections with cooccidial parasites have been associated with immunodeficient patients. Cryptosporidium and I bellii 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. 6 After parasite enteric infection has been described, sero- negative oligo- or polyarthritis asymmetric, additive or migratory with predominant involvement of joints of the lower limbs, but upper joint involvement may also be present.

Our patient may have had active arthritis after enteric infection with I bellii. She developed symmetrical polyarthritis without enthesitic features of her arthritis, involving both wrists and hands, accompanied by morning stiffness resembling rheumatoid arthritis. 7 The diagnosis of parasite reactive arthritis is suggested by eosinophilia, seronegative polyarthritis, and the temporal sequence that this arthritis was triggered by I Belli infestation, and rheumatic manifestations, which improved after trimethoprim-sulfamethoxazole therapy. However, the mechanisms of reactive arthritis...