Stevens-Johnson Syndrome after Albendazole

Sir,

Albendazole is a benzimidazole anthelmintic active against various nematodes, including *Toxocara* spp (1). Although it is generally well tolerated, adverse reactions have been reported, including fever, leucopenia, thrombocytopenia, raised transaminase levels and hair loss (2). We report a patient who developed Stevens-Johnson syndrome after starting albendazole (Zentel®).

A 57-year-old man was admitted for a widespread eruption on his trunk, proximal limbs and neck. The cutaneous lesions were erythematous or purpuric macular and slightly popular spots, showing a flat atypical target aspect (3) without significant epidermal detachment. Nikolsky’s sign was negative. Painful oral and ocular erosions were present. Histological examination of a skin biopsy showed marked oedema of the superficial dermis and perivascular mononuclear cell infiltrates. General examination showed arthralgia and raised temperature (39.5°C). Blood cell count was normal except for slight eosinophilia (700/mm3). Erythrocyte sedimentation rate was accelerated (78 mm first hour). CRP (155 mg/l) and ALT and AST serum levels (172 U/l and 98 U/l, respectively) were increased. Serology for herpes virus I and II and *Mycoplasma pneumoniae* showed previous immunization. Albendazole 400 mg/day had been initiated 15 days before the eruption for toxocariasis. Albendazole was withdrawn and the cutaneous eruption disappeared in 20 days. Biological abnormalities returned to normal. The patient took no other drug. According to the standards of the French drug surveillance system, albendazole was the only culprit (4).

The diagnosis of Stevens-Johnson syndrome was retained because of widespread macular and purpuric lesions, showing a flat atypical target aspect with predominant distribution on the trunk, associated with involvement of two different mucosal sites (3). The syndrome is related to drug intake in at least 50% of cases (5). This is the first reported case observed after albendazole treatment.

REFERENCES

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Scar Depigmentation in Systemic Sclerosis

Sir,

Pigmentary changes are commonly seen in systemic sclerosis and usually occur in 3 patterns (1): (i) a diffuse brown melanoderma that mimics Addison’s disease, (ii) patchy areas of depigmentation interspersed with perifollicular pigmentation, so-called “salt and pepper” pigmentation, and (iii) focal hypopigmentation and hyperpigmentation on sclerosed skin. We have recently observed depigmentation in the scars of patients with systemic sclerosis, a clinical feature that appears not to have been described previously.

CASE REPORTS

The sign was observed in 5 patients with advanced systemic sclerosis. There were 4 women and one man, whose ages ranged from 22 to 58 years. All patients fulfilled the American Rheumatism Association criteria for the diagnosis of systemic sclerosis and had disease for periods ranging from 2.5 to 7 years. All the patients had Raynaud’s phenomenon and binding down of the skin of the extremities, face and trunk. Four patients had sclerodactyly, while all had finger tip ulcers and/or scars. Pulmonary functions were deranged in all the patients and 4 had clinical dyspnoea. Barium swallow revealed decreased esophageal motility in 4 patients. One patient had proteinuria.

All patients had patchy depigmentation with residual perifollicular pigmentation within the macules (“salt and pepper pigmentation”). Diffuse hyperpigmentation of the skin was seen in 2 patients. In

Fig. 1. Depigmentation in muscle biopsy scar on arm. Note “salt and pepper” pigmentation on adjacent skin and on the chest.

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