Stevens-Johnson Syndrome after Alendazole

Sir,

Alendazole 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 alendazole (Zenite®).

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. Alendazole 400 mg/day had been initiated 15 days before the eruption for toxocariasis. Alendazole was withdrawn and the cutaneous eruption disappeared in 20 days.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, alendazole 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 alendazole treatment.

REFERENCES


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Sciar 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 perilocular 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 perilocular 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.

Acta Derm Venereol (Stockh) 77
addition, all patients had depigmentation in the scars following muscle biopsy (1) (Fig. 1), skin biopsies (2), smallpox vaccination (1), a healed auricular sinus (1) and in long-standing scars (2), whose cause the patients were unable to recall. Three scars were normally pigmented before the onset of systemic sclerosis and depigmented subsequently; 3 scars developed after the onset of disease and were depigmented de novo, while one patient was unable to date the onset of depigmentation in the scar. In no patient did the depigmentation of scars precede the onset of the "salt and pepper" depigmentation of systemic sclerosis.

COMMENT

The patchy depigmentation of systemic sclerosis has many clinical, histological, histochemical and ultrastructural similarities to vitiligo (2). Koeber's phenomenon is known to occur in vitiligo, and a similar process may explain the depigmentation that developed in the fresh scars following trauma, skin and muscle biopsies in our patients. However, depigmentation of the pre-existing scars of smallpox vaccination and previous injury cannot be thus explained.

An alternative explanation is suggested by the clinical observation that focal depigmentation develops on sclerosed skin (2). If this finding indicates that depigmentation supervenes when a critical level of dermal sclerosis has been reached, it could explain the predilection of depigmentation for fibrotic scars, new or old.

While our finding is unlikely to be of any diagnostic value, since it appears in patients with well-established, unmistakable systemic sclerosis, it may provide clues to the pathogenesis of this ill-understood disease.

REFERENCES


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Transient Acantholytic Dermatosis after Treatment with 2-Chlorodeoxyadenosine

Sir,

We agree with Meunier et al. (1) that cutaneous reactions secondary to 2-chlorodeoxyadenosine (2-CdA) in patients receiving this drug for the treatment of hairy cell leukemia have rarely been reported. In their retrospective study, 7 of 33 patients (21%) developed cutaneous manifestations while receiving daily intravenous continuous infusion of 2-CdA (at a dose of 0.1 mg/kg/day for 7 days); a maculopapular exanthem in 6 patients and toxic epidermal necrolysis in one (1). To the best of their knowledge, only one other hairy cell leukemia patient had previously been described who developed a diffuse maculopapular skin rash 4 days after initiating 2-CdA treatment at the same dose schedule (2). However, the cutaneous adverse reaction was attributed to a concurrently administered drug in 6 of these 8 patients (1, 2); in only 2 patients no other drug than 2-CdA was given (1).

Several patients with hairy cell leukemia have been treated at the University of Texas M.D. Anderson Cancer Center. One of the authors (RK) has managed the care of over 100 hairy cell leukemia patients during the past 9 years; most of these patients were treated with interferon alfa. However, at least 40 patients have been treated with 2-CdA (at a daily dose of 4 mg/m² for 7 days) as either their initial management or subsequent therapy (3). Two of these men developed biopsy-confirmed transient acantholytic dermatosis within 3 days after treatment with 2-CdA had been initiated or had been discontinued. Both patients (ages 45 and 51 years) were febrile; also, there was no improvement of the dermatosis after topical and/or intravenous antibiotics. The lesions were initially suspected to represent a disseminated herpesvirus infection in one of the men; however, there was no improvement after systemic acyclovir therapy. The lesions resolved within 1 to 2 weeks and have not subsequently recurred.

REFERENCES