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Reply

To the Editor: I thank Dr. Driesch for his important comments. In our case 1, a series of the episodes may be attributable both to the association with the myelodysplastic syndrome (MDS) and to treatment with granulocyte colony-stimulating factor (G-CSF).

First, as to the relation between G-CSF and skin lesions, our opinion is in accordance with Dr. Driesch's. It is possible that G-CSF was related to the skin manifestation. G-CSF is reported to be a potent myelopoietic growth and differentiation factor in vivo.[1] It may also be a potent activator of granulocyte functions such as superoxide release[2] or phagocytosis.[1] Therefore in case 1 it is suggested that neutrophils activated by G-CSF might be mobilized and might induce the skin lesions.

Second, G-CSF might contribute to the appearance of pseudo-Pelger-Huët neutrophils or hypersegmented ones. The presence of those anomalous neutrophils is one of the characteristics of dysgranulopoiesis, which is often seen in patients with MDS. However, there is a possibility that G-CSF differentiated the MDS clone into neutrophils with abnormal nuclei and mobilized them from bone marrow to peripheral blood. Toyama et al.[3] reported that those anomalous neutrophils appeared in peripheral blood during G-CSF treatment and disappeared after discontinuation of that therapy.

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Progressive facial hemiatrophy (Parry-Romberg syndrome) and antibodies to *Borrelia*

To the Editor: Abele et al. recently reported (J AM ACAD DERMATOL 1990;22:531-3) a case of progressive facial hemiatrophy (PFH) with high titers of antibodies to *Borrelia* detected by indirect immunofluorescence (IF) (IgM 1:16 and IgG greater than 1:128). In addition, they found in skin sections of the patient, microorganisms compatible with *Borrelia* sp. when these sections were stained with the Steiner silver impregnation procedure and by direct IF with the use of a monoclonal antibody specific for *Borrelia*. They therefore propose to examine patients with PFH for borreliosis because early antibiotic therapy could offer the cessation of disease progression.

We became interested in the subject, and we have tested the sera from four patients (one man, three women) with PFH to investigate the presence of antibodies to *B. burgdorferi* by enzyme-linked immunosorbent assay (ELISA) (Diamedix Corporation, Miami, Fla.) and by indirect IF to detect IgG and IgM antibodies (Scimedx Corporation, Danville, N.J.). The patients' ages ranged from 8 to 40 years, and the duration of the disease from 2 to 11 years. None of the patients recalled a tick bite. As a control group we studied 17 healthy persons.

Antibodies to *B. burgdorferi* were negative with both methods in all four patients with PFH and in the control group. These results suggest that there is no relation between *B. burgdorferi* infection and PFH. However, we are aware that the current system of laboratory confirmation of infection by *B. burgdorferi* is not perfect.[1,2] Currently, we are doing some studies to determine the kind of ticks that are more prevalent in our geographic area, and the prevalence of infection with *B. burgdorferi* in those *Ixodes* (Esgleyes-Ribot et al., manuscript in preparation). More studies are needed before a positive correlation can be established between PFH and *Borrelia* infection.

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