Localized Scleroderma Is Not a *Borrelia burgdorferi* Infection in France

L.		Vaillant ^a
A.		Goudeau ^b

^aService de Dermatologie, CHU Trousseau, et ^bUnité de Virologie, Département de Microbiologie Médicale et Moléculaire, URA CNRS, CHU Bretonneau, Tours, France

L. Vaillant, Service de dermatologie, CHU Trousseau, F-37044 Tours Cedex (France)

We read with great interest a recent article by Aberer et al. [1] published in your journal. They investigated different types of mor-phea, and 46% of their patients were sero-positive for *Borrelia burgdorferi*. These authors have hypothesized yet in 1985 that localized scleroderma might be a late manifestation of *B. burgdorferi* infection [2]. This hypothesis has remained controversial since some authors [3, 4] denied any relation between localized scleroderma and *Borrelia* infection and others demonstrated some evidence for a spirochetal origin of localized scleroderma [5, 6]. All positive studies came from Western Europe. To explain these conflicting results, differences in the prevalence of antibodies in the general population [7] or differences between *Borrelia* strains isolated in Europe or the United States have been evoked. We have carried out a prospective case-control study in France to investigate the relationship between *B. burgdorferi* infection and localized scleroderma (participating in this study: L. Vaillant, A. Goudeau, M. Larregue, D. Barrut, E. Gross-hans, J. Maleville, A. Cosnes, M. Avenel, D. Lambert, F. Truchetet, D. Dorcier, P. Thomas, P. Litoux, V. Salagnac, G. Lorette).

In our prospective study were included well-defined localized scleroderma (with a characteristic lilac-colored edge) seen at any time of the evolution. Progressive systemic sclerosis and induced scleroderma were excluded. Sera from patients were investigated for antibodies against *B. burgdorferi* by a standard immunofluorescence assay (Lymix, Diagast Laboratories) at the time of the first evaluation and 2 months after. For each patient 1 control, matched for sex and age (± 5 years), was tested; controls were outpatients who consulted in Dermatology with noninfectious dermatological and not *Borrelia*-related diseases. Titers $\leq 1:128$ were considered as nonreactive, titers 1:256 as borderline and titers $\geq 1:512$ as reactive. Every borderline serum was controlled using a hemagglutination assay (Lymag, Diagast Laboratories) and was tested for treponemal reaction (both VDRL and TPHA assays). Sera were obtained from 67 patients (45 females and 22 males) with long-lasting mor-phea (mean 33.9 months, SE 5.7), and age ranged from 3 to 75 years (mean 37, SE 2.7). No sera of either patients or controls were reactive against *B. burgdorferi* by immunofluorescence assay. Sera from 8 patients and 5 controls were borderline. In these sera, VDRL and TPHA were negative. Two months after the initial test, sera from all 67 patients were still negative or borderline.

Our results clearly demonstrate that localized scleroderma is seldom or never associated with *B. burgdorferi* infection in France. We suggest that discordances between previous studies lie within the difficulty of the clinical diagnosis of localized scleroderma. Sclerotic skin lesions, clinically and histologically similar to localized scleroderma, may occur in about 10% of

cutaneous B. infection [8]. Moreover, cases of association between acrodermatitis chronica atrophicans and the so-called localized scleroderma have been reported [9, 10]. We suggest that the rising number of cases of localized scleroderma associated with evidence of B. burgdorferi infection are not coincidental but due to sclerotic skin lesions of acrodermatitis chronica atrophicans similar to localized scleroderma.

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