Acta Derm Venereol (Stockh) 1994; Suppl. 186: 119–120

Psoriasis and Lyme Arthritis

G. BIANCHI[1], L. BUFFRINI[1], M. C. GRIGNOLO[1], G. ROVETTA[1], F. CROVATO[2] and P. MONTEFORTE[1]

[1]*DIMI, Cattedra di Reumatologia, Centro Reumatologico Instituto E. Bruzzone,* [2]*Divisione di Dermatologia, U.S.L. XVIII, Chiavari, Italy*

Psoriasis is a distinctive skin disease that may sometimes be triggered by different agents, such as streptococci and staphylococci, and this may also occur in psoriatic arthropathy. (1). Moreover, psoriasiform lesions may be the first clinical manifestations of HIV infection (2).

Interesting relationships also exist between the course of psoriasis and infections: frequently, streptococcal infections can aggravate the disease (3), and HIV infection may exacerbate the psoriatic condition, including the joint manifestations, in individuals with pre-existent psoriasis (2).

Reactive arthritides are a group of joint diseases characterized by the fact that arthritis follows infections in other organs, caused by various bacteria (Streptococcus, Yersinia, Chlamydia, Salmonella, Shigella), and that they are frequently linked to the genetic make-up of the patient (4). The clinical manifestations are often similar to that of HLA-B27 related spondyloarthropathies, in particular Reiter's syndrome.

Lyme borreliosis (LB) is a multisystem disease, caused by the spirochaete *B. burgdorferi,* characterized by involvement of the skin, joints, nervous system, and heart (5). Some facts support the possibility that, at least in some cases, the joint manifestations caused by *B. burgdorferi* could be a reactive arthritis.

We studied the relationship between *B. burgdorferi* infection, Lyme arthritis, and psoriasis in a group of patients with suspected LB.

Patients and methods

The study population consisted of 1,321 patients examined at the Rheumatological Centre of the University of Genoa because of signs and symptoms possibly related to LB. Patients, all living in an area endemic for LB, were referred to us by rheumatologists, dermatologists, neurologists, and by general practitioners.

A complete rheumatological examination was performed, and patients were diagnosed as having LB according to a set of criteria (6): living in an area endemic for LB or previous tick bite; presence of serum antibodies to B. burgdorferi; at least two of the signs and symptoms compatible with LB (erythema migrans, systemic, neurological, cardiac, articular).

Antibodies to *B. burgdorferi* were measured by both indirect immunofluorescence (IFA) and enzyme-linked immunosorbent assay (ELISA), with previous absorption with *Treponema phagedenis* to avoid cross-reactions with *Treponema pallidum.*

Antibodies vs *B. burgdorferi*:	Positive *n* (%)	Negative *n* (%)
Psoriasis (*n* 32)	11(34)	21(66)
Psoriatic arthropathy (*n* 16)	2(12)	14(88)
All psoriatics (*n* 48)	13(27)	35(73)
Non-psoriatics (*n* 1273)	320(25)	953(75)

RESULTS

Forty-eight (3.6%) patients out of 1,321 were affected by psoriasis. Eight (16.6%) of these 48 had LB, and 2 of them fulfilled the criteria for both LB and psoriatic arthropathy, male-female ratio was 5/3 and mean age was 44 years. Among the 40 (83.4%) non-LB psoriatic patients, 26 (65%) had psoriasis only, and 14 (35%) psoriatic arthropathy. Male-female ratio was 25/15 and mean age was 47 years in this group.

One-hundred and eighty-seven (14.7%) out of 1,273 non-psoriatic patients had LB, male-female ratio was 66/121, and mean age was 46 years. The remaining 1,086 patients were affected by neither psoriasis nor LB.

LB was diagnosed in 195 (14.8%) out of 1,321 subjects examined. The prevalence of psoriasis in patients with LB was 8/195 (4.1%), and 40/1126 (3.6%) in non-LB patients. The presence of antibodies to *B. burgdorferi* in psoriatic and non-psoriatic patients is shown in the table.

There was no difference in the sero-prevalence of antibodies to *B. burgdorferi* between the psoriatic and the non-psoriatic patients.

DISCUSSION

Both psoriasis and psoriati arthropathy can be triggered by bacterial and viral infections, and recently HIV infection has been related to the appearance and the exacerbation of psoriasis and psoriatic arthropathy.

Infectious agents are also responsible for the appearance of reactive arthritides, and in some cases the arthritis of LB seems to follow the clinical pattern of reactive arthritis. This suggest that the spirochaete *B. burgdorferi* may act as a trigger for joint inflammation.

Thus it is conceivable that *B. burgdorferi* could act as a trigger in psoriasis and/or in psoriatic arthropathy.

Our results do not confirm this hypothesis, as we did not find any difference in the prevalence of LB in psoriatic and non-psoriatic patients. Nor was any difference found in the prevalence of psoriasis between patients with and without LB.

Moreover, the prevalence of antibodies to *B. burgdorferi* did not differ between patients with and without psoriasis. However, the presence of 2 patients who fulfilled the diagnostic criteria for both LB and psoriatic arthropathy raises the possibility that some relationship with *B. burgdorferi* infection could exist, at least in patients with psoriatic arthropathy.

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 ISSN 0365-8341

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