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Anetoderma and borreliosis: is there a pathogenetic relationship?

A 32-year-old man simultaneously developed anetoderma and acrodermatitis chronica atrophicans on his left arm and showed a positive serology for borreliosis with ELISA and Western Blot tests. In addition, a 45 year-old man is presented with anetoderma *without* any associated systemic or cutaneous diseases, with *B. afzelii* confirmed as a singular causality through serology (ELISA, Western Blot) and amplification of *B. afzelii*-specific DNA from the skin by PCR. These two observations highly suggest that anetoderma can be the result of an infection with *B. afzelii*. We conclude that in patients with anetoderma a serological investigation for Borreliosis should be performed.

Key words: *Acrodermatitis chronica atrophicans*, *anetoderma*, *borrelia afzelii*, *pathogenesis*, *single-tube nested PCR*

Anetoderma is a rare disease clinically characterized by localized laxity of the skin with herniation resulting from destruction of normal dermal elastic fibers.

Anetoderma is subdivided into primary idiopathic and secondary variants. Secondary anetoderma (SA) has been reported to occur after various inflammatory or non-inflammatory dermatoses [1, 2]. Primary anetoderma (PA) develops on clinically normal skin without any preceding dermatoses but it may be associated with systemic and/or cutaneous diseases [3-5].

Acrodermatitis chronica atrophicans (ACA) is a well-documented infectious dermatosis caused by *B. burgdorferi*. In addition to lymphadenosis benigna cutis and erythema chronicum migrans, in rare cases other skin diseases have been reported to be associated with an infection by *B. burgdorferi* [2, 6-8].

In 1958, Hauser stated that anetoderma could be found near the edge of ACA (9). It is interesting to note that this association has not been described again since then.

Case one: A 35-year-old man presented with atrophic and reddish skin changes on the back of his left hand, which had developed when he was 25 years old. At the same time spotty and asymptomatic depigmented lesions appeared, restricted to the left arm. In 1991 histology from a spotty lesion was diagnosed as anetoderma. Silver staining could not detect spirochetes. Serology for borreliosis was positive in the ELISA and Western Blot tests. He was treated with doxycyclin for 20 days, which lead to amelioration without resolution. When we saw the patient in 2001, he showed the typical clinical picture of ACA (*Fig. 1*) and anetoderma (*Fig. 2*) on his left hand and arm. A second course of doxycyclin 2 × 100 mg a day for 28 days was carried out and lead to marked amelioration.

Case two: A 45 year-old man complained of a generalized weakness lasting for several months. Routine examination by a general practitioner and neurological examination in a sleep laboratory had already taken place with no conclusive results. His personal history was otherwise



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Figure 1 and 2. Document acrodermatitis chronica atrophicans and anetoderma on the left arm in Case 1.



Figure 3. Shows the typical lesions of anetoderma in Case 2.

uneventful. On physical examination typical flabby lesions of anetoderma were seen. They were scattered over his trunk, both arms and thighs (Fig. 3). The patient himself had noticed the presence of these “scars” over a year before. He presumed them to be the result of mosquito bites, describing them as being red, swollen and itchy spots in the beginning, fading after a few days. He did not recall being bitten by a tick. Hematological and serological examinations ruled out the presence of any autoimmune or hematological disorder. There were no antiphospholipid antibodies detected and syphilis had been ruled out. Protein electrophoresis was normal and no monoclonal gammopathy was detected by immunofixation. The only pathological finding was a positive serology for *Borrelia afzelii* with ELISA and Western Blot tests. The histological examination showed findings that were consistent with the clinical diagnosis of anetoderma. In addition to these results, *B. afzelii*-specific DNA from the skin was detected by PCR [10-12] (Fig. 4). After ruling out neuroborreliosis and cardiac or rheumatologic complications, treatment with 200 mg of doxycycline per day was carried out for thirty days. Soon after the treatment, the generalized weakness disappeared and no new urticarial lesions with the resulting typical scars of anetoderma were observed.

Discussion

In 1958, Hauser noted the simultaneous occurrence of anetoderma and ACA [9]. In 1955 he had already presumed that ACA could be a tick-borne dermatosis, without knowing, at that time, about the true infectious agent. The connection between anetoderma and ACA is mentioned in a textbook of dermatology [13] and in 1991 by Garbe [14], although it had not then been proven that a similar pathogenesis could be responsible for these two clinical entities. Our first case enables us to postulate a pathogenetic relationship between PA and ACA, since these two diseases occurred simultaneously on the left arm of a 32-year-old man with a positive serology for borreliosis. The second case shows PA of the Schwenninger-Buzzi type. An infection with *B. afzelii* as the only pathogenetic result was confirmed by serology and by amplification of *B. afzelii*-specific DNA from the biopsy specimen by PCR.

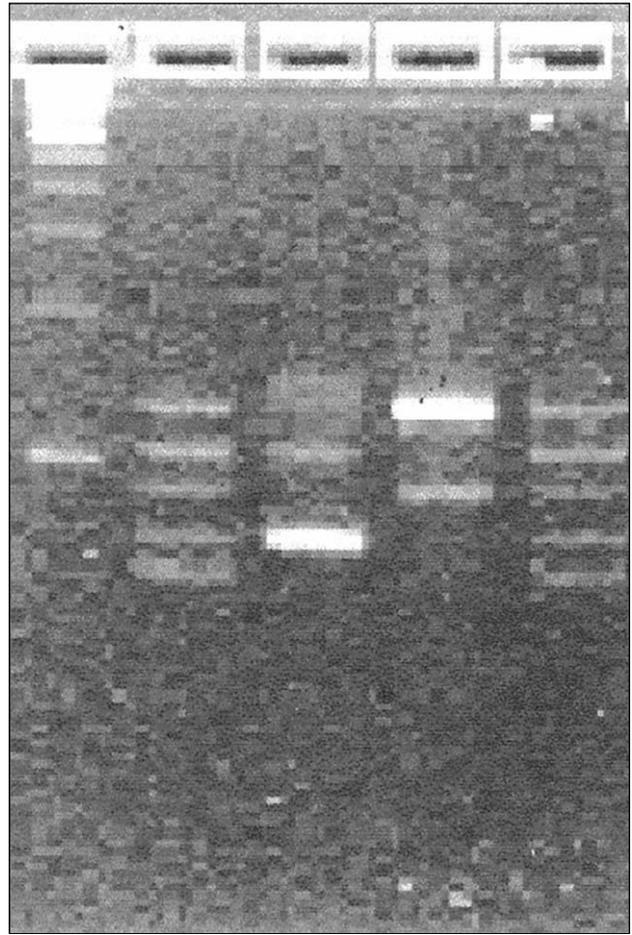


Figure 4. Identification of *Borrelia* DNA after multiplex PCR and high resolution agarose gel electrophoresis.

Lane 1: 100 bp DNA ladder;

Lanes 2,5: DNA marker with the 5 specific fragments (62, 72, 90, 108, 132 bp length);

Lane 3: pattern specific for *B. burgdorferi sensu stricto* (positive control);

Lane 4: pattern specific for *B. afzelii*.

In conclusion: Our two cases illustrate that anetoderma may be related to an infection with borreliosis in rare cases. Therefore in any case of anetoderma serological testing for *B. afzelii* is recommended. ■

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