An Infection of Acrodermatitis Chronica Atrophicans Herxheimer by Borrelia Affzelii

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Introduction
Acrodermatitis Chronica Atrophicans Herxheimer (ACA) is a tick-borne disease due to infection by Borrelia afzelii, the major vector organism is Ixodes ricinici. We report on a 48-year-old male patient who developed extensive livid-erythematous fibrosclerotic symmetrical plaques associated with hyperpigmented widely distributed lesions within the tension lines, and acrocyanosis. The diagnosis of ACA has been confirmed by histopathologic examination of a skin biopsy and laboratory investigations with positive IgG and IgM immunobLOTS. The patient was treated by intravenous ceftriaxone resulting in partial remission of cutaneous and extracutaneous symptoms.

Keywords: acrodermatitis chronic atrophicans herxheimer; borreliosis; cutaneous manifestations; treatment.

Abstract
Acrodermatitis Chronica Atrophicans Herxheimer (ACA) is a tick-borne disease due to infection by Borrelia afzelii, the major vector organism is Ixodes ricinici. We report on a 48-year-old male patient who developed extensive livid-erythematous fibrosclerotic symmetrical plaques associated with hyperpigmented widely distributed lesions within the tension lines, and acrocyanosis. The diagnosis of ACA has been confirmed by histopathologic examination of a skin biopsy and laboratory investigations with positive IgG and IgM immunobLOTS. The patient was treated by intravenous ceftriaxone resulting in partial remission of cutaneous and extracutaneous symptoms.

On this hands, a livid erythema was noted. During inspiration the lower thoracic aperture had a decreased elongation. On the hands there was an incomplete fist circuit notable. No other clinical symptoms were noted. We performed a skin biopsy. Histologic evaluation disclosed dermal changes including broadened and homogenized collagen bundles, perivascular and perinodal lymphocytic infiltrate with some mast cells and plasma cells intermingled. PCR for Borrelia remained negative.

Laboratory investigations
Leukocytes 12.4 (normal range: 3.8-11.0 Gpt/l); neutrophils 9.4 (1.8-7.6 Gpt/l); C-reactive protein 17.6 (< 5 mg/dl); total IgE 269 (0-100kU/l); rheumatoid factor 38 (< 14 IU/ml); Borrelia IgG-antibodies [Enzyme Immuno assay] >200 (<16 RE/ml), IgM-antibodies 19.2 (<16 RE/ml), IgG-immunoblot: positive; IgM-immunoblot positive; serum albumin 47.6 (60.3-71.4 %), Y-globulin 30.0 (8.7-16.0 %). Antibacterial antibodies (ANA) and antibodies against extractable nuclear antigens (ENA): negative.

Imaging diagnostics

Case Report
A 48-year-old male patient was referred to our hospital because of large livid-erythematous fibrosclerotic plaques on his trunk and extremities which developed within half a year. He suffered from arterial hypertension and had a penicillin allergy. He had no memory of any tick bite.

On examination we observed symmetric large livid-erythematous fibrosclerotic plaques on his upper back. Erythematous to brownish lesions along the tension lines of skin were found on the lower back, abdominal, on the shoulders and proximal extremities.
Treatment and course

Based on clinical examination, histopathology and serologic investigations the diagnosis of ACA, edematous stage, was confirmed. Because of the penicillin allergy we treated the patient with intravenous ceftriaxone 2g once daily for 10 days. We combined this with topical steroids, bath-PUVA (Psoralen Plus UVA-irradiation), and complex physio- and ergotherapy. The latter consisted of manual lymph drainage, respiratory therapy, relaxation, and motoric-functional treatment of both hands.

We achieved a partial response with improved motoric ability of the hands. The skin became more softened, erythema vanished, and fibrosis improved.

Discussion

ACA is a tick-borne disease with progressive course. In the adult European population 1-2% of Borreliosis develop ACA [2], among children ACA was observed in about 1% [6]. The clinical presentation may vary. Extracutaneous manifestations are common among our patients [7]. Cutaneous manifestations cover a broad spectrum. Unusual symptoms include chronic venous insufficiency [8], vasculitis racemosa [9], morphea- and lichen sclerosus-like lesions [10], anetoderma [11], juxta-articular nodules [12], small spinous papules [13], foot ulcers [3], and alopeia [14]. A very rare manifestation is facial involvement [15, 16].

Our patient was quite unusual related to cutaneous manifestations. Hyperpigmented lesions along the tension lines of skin and extensive livid-erythematous fibrosclerotic plaques are ambiguous. Only the livid erythema of the hands was a classical presentation. Serologic investigations and histopathology of a skin biopsy, however, confirmed ACA.

Early antibiosis is important to prevent the progress of ACA to an atrophic stage. Intravenous treatment is possible with ceftriaxone, cefotaxime, or penicillin G [17]. We used ceftriaxone since the patient had a penicillin allergy.

ACA remains a diagnostic challenge, this has been illustrated by our patient.

References