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Case Report Open Access

Localised Scleroderma-Patchy Type Morphea

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Abstract

Background: Scleroderma comes in two main forms: systemic and localized. Morphea is a localized form of scleroderma and affects primarily the skin. The cause of morphea is unknown. Literature focuses on Borrelia burgdorferi as a possible etiologic agent for morphea.

Methods: It is a case presentation of 8 years old female. The first cutaneous changes have occurred three months prior to hospitalization with appearance of a solitary red patch surrounded by a violet ring in suprapubical region. Within a short time, several other patches similar to the first one have been appeared close to the first one and one on the spine. A year ahead, she experienced the tick bite on the head.

Conclusions: Although the etiology of morphea is unknown, the Borrelia origin of morphea was verified by the presence of antibodies against Borrelia Burgdorferi. The data obtained in this study suggest that Borrelia Burgdorferi may play a role in the etiopathogenesis of disease.

Background

Morphea is a localized type of scleroderma, which is characterized, with extensive storage of collagen, which leads to the thickening of derma, subcutaneous tissue, or both. Morphea is classified in several types, such as patchy, generalized, linear and deep form [1]. This classification is based on the clinical presentation and extensiveness of the pathologic process that affects the tissue. An autoimmune component is supported by the frequent presence of autoantibodies in affected individuals, as well as the association of morphea with other autoimmune diseases, including systemic lupus erythematosus, vitiligo, type 1 diabetes, and autoimmune thyroiditis [2]. Some patients with classic morphea have sclerosis due to Borrelia burgdorferi infection, and if not to sclerotic, the lesions can disappear with prolonged courses of oral antibiotics [3].

Case Presentation

First cutaneous changes on female, eight years old child, have occurred three months prior to hospitalization with appearance of a solitary red patch, 4x4 cm in diameter with clear border margin with healthy skin, surrounded by a violet ring in suprapubical region. Within a short time, another patch, similar to the first one, appeared close to the first one, 3x3 cm in diameter and the third one on the lumbar spine. The center of changes was slightly indurated. A year ahead, she experienced the tick bite on the head. History of other diseases was negative. Patient's sister suffers from vitiligo.

Laboratorial examinations within physiological levels. Anti-DNA and antinuclear antibodies negative, IgG anti Borrelia Burgdorferi antibodies were positive. The titer of IgG antiborrelia antibodies with ELISA screening was 21.0 (> 11.0 positive). The follow up titer of antibodies was 16.82. Abdomen ultrasound without pathological findings. Neurological examination without focal disturbances and neurological lateralization. Fundus oculi: without pathological findings. Biopsy was taken from the spine changes, approximately 1 cm in diameter, till subcutaneous tissue, showing features for morphea. Pathohistological report revealed thinned epidermis, increase of fibrocollagenous tissue in dermis with dilated blood vessels and peripheral lymphocytic infiltration. Superficial layers of the skin adnexes were atrophic and reduced, whereas the deeper layers were surrounded by fibrotic tissue. These derangements belong to the early stage of sclerodermy (Figure 1).

For treatment systemic antibiotics, firstly parenteral and following oral penicillin was given in several courses over a time span of several months. Initially the treatment with benzatin benzyl penicillin 800.000 IU, im for 10 days, following with amoxicillin, 50 mg/kg tid, for next two weeks, and then three courses of amoxicillin 50 mg/kg tid per ten days for next following three months.

Systemic and topical corticosteroids were used as well. Concomitantly with antibiotics methylprednisolone i.m. 20 mg per diem for ten days, following oral prednisolone with continuously tapering with three weeks. Topical betamethasone valerate 0.1% cream for first seven days during three months was applied, followed with panthenol cream between topical steroids, successively (Figures 2 and 3).

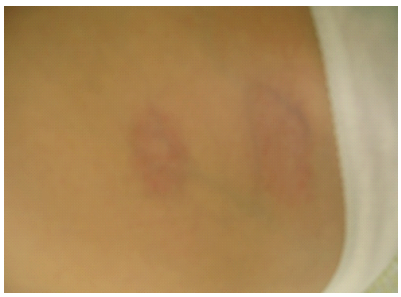

Figure 1: Morphea patches, clear border between the patch and the healthy skin, slight central skin atrophy, abdomen.

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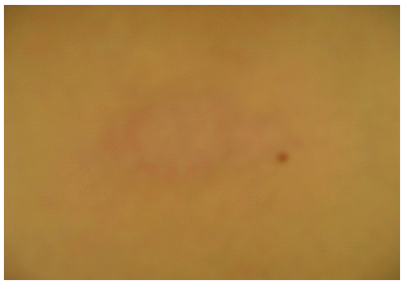

Figure 2: Morphea patches, clear border between the patch and the healthy skin, slight central skin atrophy, spine.

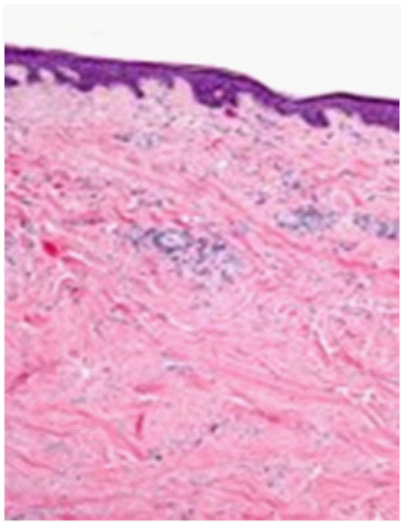

Figure 3: Thinned epidermis, increase of fibrocollagenous with dilated blood vessels and peripheral lymphocytic infiltration.

The skin changes gradually withdrawal within 5-6 months, with certain residual skin hyperpigmentation.

Conclusion

Extensive production of collagen from fibroblasts in the affected tissues is a common pattern for all types of morphea, though the mechanism that induces excessive production is still unknown. Although morphea occurs in persons of all races, it appears to be more common in whites, who comprise 73-82% of patients seen [4]. Females are affected about three times as often as males, including children. The cause of morphea is unknown. Data from various researches didn't reveal any sustainable etiologic factor. An autoimmune mechanism is suggested by an increased frequency of autoantibody formation and a higher prevalence of personal and familial autoimmune disease in affected patients [5]. Infections such as those with Epstein-Barr virus, varicella, morbilli, or borreliosis, have been reported as underlying factors for the onset of morphea and furthermore they have been recommended as a possible trigger factors for morphea. *Borrelia burgdorferi* today is a possible etiologic factor [6]. Antibodies *to B. burgdorferi* and high antinuclear antibody titers have been described in patients with morphea, and it has been suggested that *Borrelia*-associated early-onset morphea may represent a subset of patients with infection-induced autoimmunity. However, results have been conflicting, as other studies have not found a definitive association between Borrelia infection and morphea based on serologic or polymerase chain reaction data [7]. Antinuclear antibodies are present in approximately 20-80% of morphea patients, typically with a homogeneous, speckled, or nucleolar pattern. The prevalence is higher in patients with generalized, linear, and deep subtypes. Anti–single-stranded DNA antibodies are present in 25% of patients with plaque-type morphea, levels correlate with extensive, active disease and joint contractures [8]. The histologic findings of morphea and systemic sclerosis are similar, with a fundamental process of thickening and homogenization of collagen bundles [9]. Patchy type morphea is a self-contained variant of scleroderma with a tendency to evolve slowly with resolution after 3-5-years. Topical potent corticosteroids may contribute in the reduction of inflammation and prevention of progression of the disease. Other topical treatment like calcipotriene, tacrolimus and imiquimod are effective in reducing the erythematous and indurated lesions in a small cohort of patient with morphea [10-12].

Though the etiology of morphea in this case was not completely known, having in mind the anamnestic data on the tick bite, proved presence of antibodies against Borrelia Borgdorferi and positive respond on antibiotic therapy we concluded that disease appeared due to the borreliosis, the data that corresponds with published literature.

Author's Contributions

Blyta Ymran; conception and designee, drafting the article, critical version of the article and final revision, final approval of the version to published.

Daka Aferdita; critical version of the article.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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