Case Report

Friction-Induced Biphasic Cutaneous Amyloidosis

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Abstract: Primary cutaneous amyloidoses (PCA) are a group of conditions characterized by deposition of amyloid in previously normal skin, without association with other skin or systemic diseases. We describe a Kazakhstani female with a 30-year history of increasingly spreading hyperpigmented macular as well papular skin lesions on her upper trunk accompanied by pruritus. Moreover, her medical history included intensely rubbing her skin with a cotton towel following bathing and showering. On the basis of the clinical and histopathological findings, the diagnosis of biphasic cutaneous amyloidosis was made. The present unusual case of biphasic cutaneous amyloidosis can be subsumed under mechanically-induced forms of cutaneous amyloidosis. In conclusion, the present case underscores the necessity to explore carefully the patient’s history in order to discover the cause of PCA.

Keywords: cutaneous amyloidosis; friction dermatosis; friction melanosis

1. Introduction

Primary cutaneous amyloidoses (PCA) belong to a group of conditions characterized by deposition of amyloid in previously normal skin, without association with other skin or systemic diseases [1–4]. Clinically, PCA can be divided into three subtypes: (1) macular, (2) papular (lichenoid), (3) nodular. Biphasic amyloidosis is a rare entity characterized by the presence of concurrent lesions of macular and lichen amyloidosis [1,2]. The confluent pigmented macules with a ripped pattern observed in macular amyloidosis as well as the flesh-colored or pigmented papules seen in lichen amyloidosis are observed. The macular lesions are frequently found in the interscapular area while the papular lesions are most frequently observed on the extremities. We here describe a case of biphasic cutaneous amyloidosis associated with a long history of a mechanical trigger.

2. Case

We describe a 53-year-old Kazakhstani female (photo-skin type III) with a 30-year history of increasingly spreading hyperpigmented skin lesions on her upper trunk accompanied by pruritus. Importantly, she reported on inquiry a 30-year history of intensely rubbing her skin with a cotton towel following bathing and showering. She had no family history of skin disorders or internal medical conditions, except for chronic atrophic gastritis type A. Systemic antihistamines, topical glucocorticosteroids, and skin care products were tried without beneficial effects. On examination, there were V-shaped dirty-brownish macules and patches over her shoulders and smaller brownish-spotted macules on her left and right scapula (Figure 1). Moreover, there were tiny aggregated papules on the sides of her neck and upper arms. Laboratory tests did not reveal pathologies on blood chemistry analysis, protein electrophoresis, serum immunoglobulin tests, coagulation analysis, and urine analysis including Bence-Jones proteins.
Figure 1. V-shaped dirty-brownish confluent large macules and patches over the shoulders, smaller brownish-spotted macules on both scapulae (a,c), and tiny aggregated papules on the sides of the neck (b) and upper arms of a Kazakhstani female with friction-induced biphasic cutaneous amyloidosis.

Abdominal and lymph node sonography as well as thoracic X-ray were normal. Histopathological assessment of papules and macules/patches revealed features of lichen and macular amyloidosis, respectively. In both specimens amorphous eosinophilic material was observed in the papillary dermis which was consistent with amyloid deposits that stained positively with Congo red (Figure 2). On immunohistochemistry staining, the deposits were positive for CK-5, CK-6, and MNF-116.

We instructed the patient to stop the habit of cotton towel rubbing and initiated oral acitretin at a starting dose of 10 mg/d. Due to skin dryness and failing benefit, the patient discontinued acitretin after five weeks and denied any further systemic treatment. We prescribed a polidocanol-containing emollient resulting in improvement of her pruritus.
3. Discussion

It has been proposed that epidermal keratinocytes degenerate into amloid by an apoptotic mechanism that is not yet understood. Clinically, our patient presented with two types of lesions—macular as well as lichenoid (papular). The association of both types is termed biphasic amyloidosis. Indeed, immunohistological investigations have confirmed the presence of keratin epitopes in the amyloid of biphasic amyloidosis [5,6]. PCA is often associated with pruritic disorders such as atopic dermatitis or cholestasis. Some authors suggested that deposition of amyloid is the result of chronic scratching, caused by long-term pruritus [3,4,7]. In this regard, friction melanosis must be mentioned in which amyloid deposits may also be observed. The clinical presentation of friction melanosis resembles macular amyloidosis and the usage of brushing utensils is characteristic for this condition. Similar to PCA, friction melanosis is frequently observed in female Asians. However, it is very likely that conditions called friction melanosis and towel melanosis can be subsumed under mechanically-induced forms of macular amyloidosis [3]. There exists no standard treatment for cutaneous amyloidosis. However, clinical improvement of cutaneous amyloidosis has been reported with oral retinoids, including etretinate, acitretin, and isotretinoin [8]. In conclusion, the present case of biphasic cutaneous amyloidosis underscores the necessity to explore carefully the patient’s history in order to discover the cause of cutaneous amyloidosis.

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References