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Satisfactory treatment of a large connective tissue nevus with intralesional steroid injection

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ABSTRACT Collagenoma is a type of connective tissue nevi, a rare hamartomatous malformation characterized by the predominant proliferation of normal collagen fibers and normal, decreased, or increased elastic fibers. Collagenomas present as multiple or solitary, hereditary or sporadic, asymptomatic, skin-colored papules, nodules, and plaques with variable sizes, and are usually located on the trunk, arm, and back. Here, we report on a 14-year-old boy who presented with an isolated giant collagenoma of the frontal area that dramatically responded to intralesional triamcinolone acetonide.

Case Presentation

A 14-year-old boy presented with an asymptomatic solitary skin-colored plaque on the frontal area that had been present for five years. In the beginning, the plaque was small but gradually progressed to a large 4 x 4 cm plaque on the forehead (Figure 1a). On the dermatological examination, two café-au-lait macules on the trunk were found. General physical examination was normal. The patient's past medical history revealed no significant findings, such as seizure, neurological deficit, or abnormal development, and trauma and manipulation were absent. Family history for similar problem was also negative. Brain CT showed that the lesion was limited to the outside of the skull bones without intracranial extension. Routine laboratory tests, comprising of liver function test, urine analysis, and complete blood count were all normal. He was very unhappy with the lesion and had developed depression due to his classmates' insults.

An incisional skin biopsy was performed which showed increased normal collagen bundles in dermis, mild hyperkeratosis, acanthosis, minimal perivascular inflammation in the upper dermis, and dermal thickening (Figure 2a, b). Verhoeff's elastic stain (VEG) revealed elastorrhexis, that is, fragmentation and thinning of the elastic fibers within the reticular dermis (Figure 2c). Therefore, an isolated cutaneous collagenoma was diagnosed.

Due to the large size and poor cosmetic result of the surgery and the great impact of the lesion on quality of life,

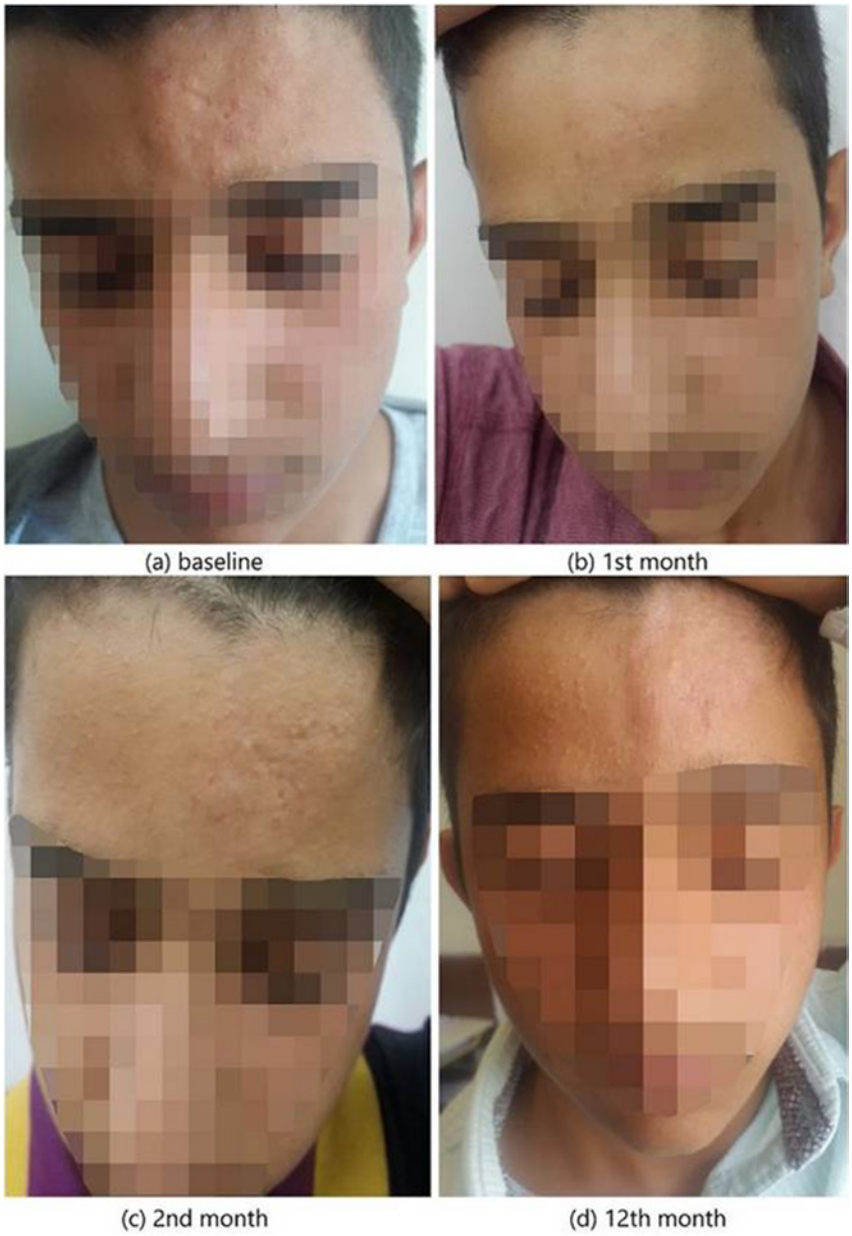

Figure 1. Trend on the improvement of the collagenoma over one year achieved by four intralesional triamcinolone acetonide injections. [Copyright: ©Saki et al.]

we decided to treat it with four monthly sessions of intralesional triamcinolone acetonide injections.

Triamcinolone acetonide 10 mg/ml was injected into the bulk of the connective tissue nevus in the first session and 20 mg/ml in the other three sessions. The patient was regularly followed and showed remarkable response with good results after the third injection. At follow-up, eight months after the last session, only the medial border of the lesion had relapsed, which was resolved with a single injection of triamcinolone acetonide 10 mg/ml (Figure 1b, c, d).

Conclusion

Connective tissue nevi are benign, well-defined hamartomas of the dermis categorized as collagenomas, elastomas, and nevi mucinosis based on the prevailing element of extracellular matrix of dermis. Collagenomas are divided into inherited and acquired types. The inherited form consists of familial cutaneous collagenoma and shagreen patch of tuberous sclerosis, which is autosomal dominant. Familial cutaneous collagenoma presents with multiple lesions and is accompanied by cardiac disorders such as cardiomyopathy and conduction abnormalities [1-5]. Acquired collagenoma includes eruptive collagenoma and isolated collagenoma. Eruptive collagenoma is described as discrete, firm, skin-colored, and elevated papules, nodules, or plaques distributed on the trunk and extremities symmetrically [6]. Isolated collagenoma exhibits numerable lesions localized on the palms, soles, and rarely other parts of the body [7,8]. The pathogenesis of collagenoma is ambiguous; however, sporadic collagenoma might be related to trauma, since it appears in areas subjected to friction. In addition, collagenomas might be associated with pseudohypoparathyroidism, hypogonadism, and Down syndrome [9-11].

Apparently, underproduction of collagenase and the consequent diminished collagen degradation is the key event underlying collagenoma pathogenesis [12]. Additionally, the required time for fibroblasts mitosis, abundant within collagenoma, is reduced and this contributes to the increase in the collagen bundles [13]. Histologically, in all types of collagenoma, coarse and dense collagen fibers accumulate in the dermis and the content of elastic fibers is diminished [14].

To the best of our knowledge, there is no conclusive effective therapy for cutaneous collagenoma except for surgical removal of the lesions [15]. Three studies investigating the therapeutic effect of triamcinolone acetonide on familial cutaneous collagenoma [16], acquired linear nodular collagenoma [17], and isolated corymbose collagenoma [18] reported the improvement of the lesions in varying degrees. The therapeutic effects of triamcinolone acetonide might be the result of the underproduction of transforming growth factor β1 (TGF-β1) in the fibroblasts and overproduction of basic fibroblast growth factor (bFGF), which in turn inhibits fibroblast mitosis and collagen synthesis [19]. In the present case we tried intralesional triamcinolone acetonide and observed

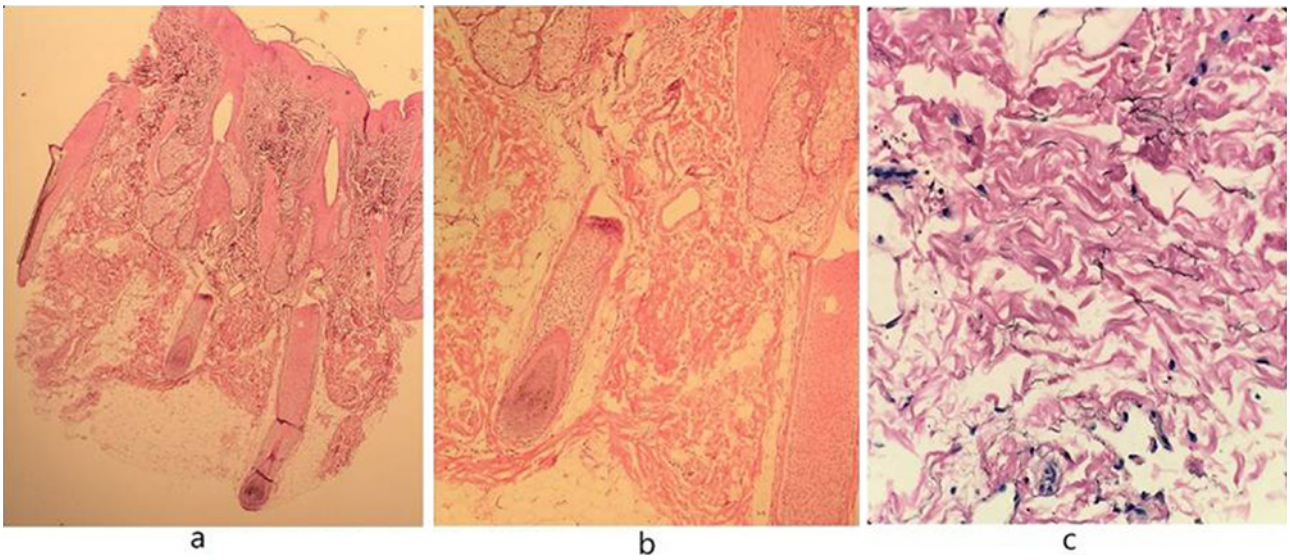

Figure 2. Microscopic findings: (a) Increased normal collagen bundles in the dermis, mild hyperkeratosis, acanthosis, minimal perivascular inflammation in upper dermis, and dermal thickening (H&E x40). (b) Partial replacement of the subcutaneous fat by broad collagen bundles (H&E x200). (c) Elastorrhexis within reticular dermis (Verhoeff's elastin stain x200). [Copyright: ©Saki et al.]

a dramatic response. Hence, intralesional triamcinolone acetonide may be a promising therapy for non-operable forms of collagenoma.

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