CASE REPORT

Keloid mimicking Dermatofibrosarcoma

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ABSTRACT

Keloid scars result from an abnormal healing response to cutaneous injury or inflammation that extends beyond the borders of the original wound. Spontaneous keloid scars forming in the absence of any previous trauma or surgical procedure are rare. Certain syndromes have been associated with this phenomenon, and few reports have discussed the evidence of single spontaneous keloid scar, which raises the question whether they are really spontaneous. In this case, I present a 35-year-old male with progressive spontaneous keloid scar on the left arm which was confirmed histopathologically by the presence of typical keloid collagen. This report supports the fact that keloid scars can appear spontaneously and are possibly linked to a genetic factor.

INTRODUCTION

A 35-year-old male presented with solitary reddish brownish indurated plaque with multinodules and central whitish scarring area in the left arm since 3 years [Fig. 1 A & B]. The main complaint of the patient was ugly look which had an effect over his personal life. The disease had a gradually onset and a progressive course especially in the last 9 months. There was no history of trauma, insect bite or any drug reactions. Also, there was no history of any other skin or systemic illness. There was no personal or family history of keloid formation. The clinical differential diagnoses considered were keloid, Keloidal morphea, sarcoidosis, lobomycosis, and dermatofibrosarcoma protuberance.

Histopathological sections showed hyperkeratosis and acanthosis with increased basal pigments. The reticular dermis showed thickened, hyalinized, eosinophilic collagen bundles within





Fig. 1 A & B Solitary reddish-brown indurated plaque with multinodules and central whitish scarring area on the left arm.

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fibrous stroma [Fig. 2, 3].

The patient was managed with intralesional administration of triamcinolone acetonide 40 mg/ml followed by silicone sheet application and customized pressure garments. Intralesional steroid injection was repeated after two weeks. A total of 4 cycles were given. The lesions showed significant improvement.

FINAL DIAGNOSIS

• Spontaneous keloid



Fig. 2 Hyperkeratosis and acanthosis with increased basal pigmentation. The reticular dermis shows thickened collagen bundles within fibrous stroma.



Fig. 3 Hyalinized, eosinophilic, thickened collagen bundles within fibrous tissue.

DISCUSSION

Keloids are known to occur following epithelial breach. Dark-skin people are more likely to develop hypertrophied scar. Certain anatomical sites like sternum and parasternal area are more likely to develop Keloid. Severity of trauma, loss of tissue, contaminated wounds are more likely to heal with keloid formation.¹ Relaxed suture line and clean wounds have fewer tendencies for hypertrophy. Multilayer closer of wound provide relaxed suture line and so less tendency for hypertrophy. Pressure areas are at a constant risk for trauma and heal with hypertrophy. People having keloid formation tendency on wearing prophylactic pressure garments have prevented formation.² keloid Studies demonstrated that keloids contain an increased level of immunoglobulins suggesting that they may be produced by an abnormal immune reaction.³ Systematic therapy for this type of idiopathic Keloid disease is still an illusion.

Keloid may occur in the context of wound hypoxia. In vitro studies showing that fibroblasts when cultured with keloid keratinocytes suggest that keloid may be a result of abnormal epithelial-mesenchymal interactions. In future the approach to keloid scarring will be to undertake a biomolecular characterization of scar and individualizing the treatment.⁵ The use of stem cell culture is a step ahead in the treatment for keloid. In this patient, conservative management was implemented, which include hygienic relief, washing, drying and intralesional corticosteroids and continuous pressure which gave continually progressive acceptable results. Till date there is no definite prophylactic therapy, or systemic therapy for keloid to cure this deformity. Histopathological and biochemical

mechanism of keloid formation has shown a way for immunosuppressive drugs in an extensive keloid disease.

Moshref and Mufti have extensively studied the possible biological and diagnostically relevant differences between keloid and hypertrophic scar using histopathological and immunohistochemical features. The differences were the presence of small aggregating blood vessels just below the epidermis appearing to grow out in keloids, while in the hypertrophic scars the blood vessels were oriented vertically around the nodules. Moderate degree of perivascular chronic inflammatory infiltrate was seen in keloids showing 73% of mast cells in reticular dermis, whereas only 20-30% of hypertrophic scars showed mast cells.⁶

Because of the rarity of spontaneous keloid scar, it is important to bear in mind the differential diagnosis of lesions resembling keloid scar, such as sarcoidosis and various benign and malignant tumors especially dermatofibrosarcoma protuberance.⁷

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