ISSN 2321 – 7359 EISSN 2321 – 7367

discovery

Medical Science

Emergence of Vitiligo Juxta-Keloid in a Female Patient – A Rare Case Report

Syed Yousuf Ali[1]☼, Harin Reddy M[2], Syed Fiaz Hussain[3]

1. Associate Professor, Department of Dermatology and STD, Shadan Institute of Medical Sciences, Peerancheru, Hyderabad-08, Andhra Pradesh, India
2. Post graduate MD final year, Department of Dermatology and STD, Shadan Institute of Medical Sciences, Peerancheru, Hyderabad-08, Andhra Pradesh, India
3. Post graduate MD second year, Department of Dermatology and STD, Shadan Institute of Medical Sciences, Peerancheru, Hyderabad-08, Andhra Pradesh, India

☼**Corresponding author:** Dr. Syed Yousuf Ali, H.No: 9-4-77/A/79, Al Hasnath Colony, Toli Chowki, Hyderabad-500008, Andhra Pradesh, India, Mail id: syedbidar@gmail.com

Publication History
Received: 02 November 2013
Accepted: 04 January 2014
Published: 15 January 2014

Citation
Syed Yousuf Ali, Harin Reddy M, Syed Fiaz Hussain. Emergence of Vitiligo Juxta-Keloid in a Female Patient – A Rare Case Report. *Medical Science*, 2014, 4(12), 42-44

ABSTRACT

Vitiligo is a common, acquired, pigmentary disorder of skin and hair resulting from the destruction of functional melanocytes. It is characterized by well-circumscribed, asymptomatic depigmented macules. Keloids is a abnormal wound responses in predisposed individuals and represent a connective tissue response to trauma, inflammation, surgery, or burns. Here, we report an unusual case of vitiligo juxta-keloid in a 46-year-old woman.

Keywords: Vitiligo, keloid, autoimmune

1. INTRODUCTION

Vitiligo is a disease in which patchy depigmentation of skin and hair results from autoimmune loss of melanocytes. It is a complex disorder involving multiple susceptibility genes and unknown environmental triggers. Genetic linkage and candidate-gene association studies have implicated several potentially contributory loci, though few have been consistently supported by the data (Jin et al. 2010). Keloids result from an abnormal fibrous wound healing process in which tissue repair and regeneration-regulating mechanism control is lost. These abnormal fibrous growths present a major therapeutic dilemma and challenge to the plastic surgeon because they are disfiguring and frequently recur (Wolfram et al. 2009). Here, we report a case of vitligo juxta-keloid in a 46-year-old woman.

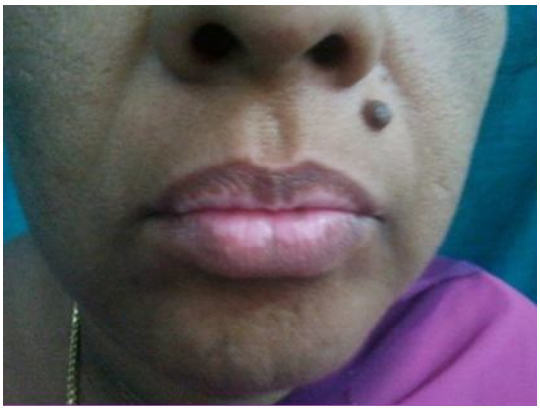

Figure 1

Mucosal Vitiligo

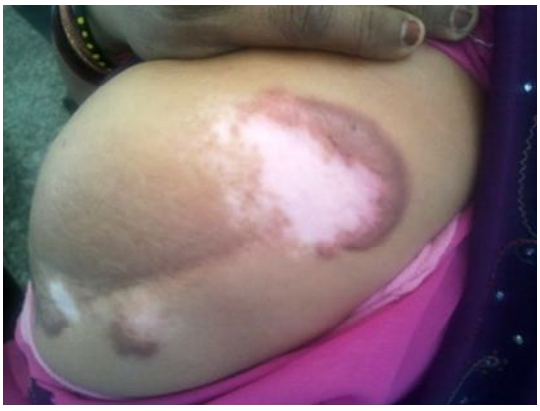

Figure 2

Vitiligo juxta-keloid

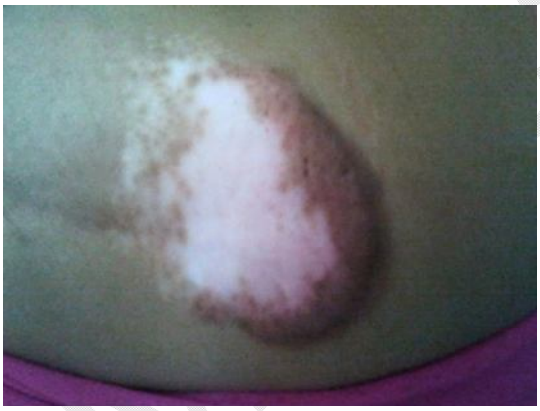

Figure 3

Close up view of Vitiligo juxta-keloid

2. CASE REPORT

A 46-year-old housewife attended our department with symptomatic keloid on left sholder since childhood. Later she developed gradually progressive, asymptomatic, whitish and non scaly macules on left shoulder and both lips of 8 years duration. Initially she developed the depigmented macule on both her lips later she noticed the similar lesions juxta-keloid (Figure 1, 2 & 3). Examination of scalp, and nails showed no abnormality. There was no history of any other illness, drug intake or intralesional therapy for a keloid. The diagnosis of vitiligo and keloid was made clinically.

3. DISCUSSION

This is an unusual case of development of vitiligo juxta-keloid in a female patient. Vitiligo is an idiopathic disease that causes destruction of melanocytes in the skin, mucous membranes, eyes, inner ear, leptomeninges and hair bulbs (Gopal et al. 2007). Although genetic, autoimmune, neurogenic and melanocyte self-destruction hypothesis have time and again been described in the etiopathogenesis of vitiligo. The occurrence of vitiligo after major illness, severe emotional stress, pregnancy, surgical operation and physical trauma has been recorded. Some noxious, melanocyte-destroying chemical entering the body through diet, consumption of readymade preserved stale food/medicines/contaminated water (through industrial waste) or the air might be an important factor in precipitation of vitiligo in a susceptible host. The occurrence of vitiligo following repeated drug intake in as many as 535 (26.75 per cent) patients is far more than just a co-incidence, and warrants serious thought prior to prescribing antibiotic therapy in patients who have a family history of vitiligo (Behl et al. 1999). Patients vitiligo have elevated frequencies of other autoimmune diseases, including autoimmune thyroid disease, rheumatoid arthritis, psoriasis, adult-onset type 1 diabetes, pernicious anemia, systemic lupus erythematosus, and Addison's disease, suggesting that these diseases involve shared genetic components (Gopal et al. 2007). The disease is further classified according to distribution pattern and extent of depigmentation. The presence of several clinical subtypes may reflect the diversity in causative factors. To select appropriate therapeutic measures it is important to distinguish vitiligo from other disorders that affect melanocyte function. Although vitiligo has a characteristic clinical appearance and histological features, the presence of early or atypical lesions often requires the exclusion of other disorders such as postinflammatory hypopigmentation and piebaldism. Therapies aimed at repopulation of lesional skin include phototherapy, application of topical corticosteroids, and transplantation of skin or skin cells. Moreover, micropigmentation or camouflage can be used to restore a pigmented appearance to lesional skin (Le Poole et al. 1997).

Keloids develop as a result of an overgrowth of dense fibrous tissue after skin injury. When an imbalance occurs between the anabolic and catabolic phases of the healing process, more collagen is produced than is degraded, and the scar grows in all directions resulting in keloid formation (Jindal et al. 2010). Experimental evidence implicates the importance of members of the transforming growth factor β(TGF-β) family in cutaneous scarring, as well as scarring in other organs. Virtually every cell in the body, including epithelial, endothelial, hematopoietic, neuronal, and connective-tissue cells, produces TGF- β and has receptors for it. TGF- β regulates the proliferation and differentiation of cells, embryonic development, wound healing, and angiogenesis. TGF- β directly stimulates angiogenesis in vivo. TGF β is released by platelets at the site of injury. Aberrations in the levels of cytokines, including interleukins 6, 13 and 15 may also have a role in keloid formation. Although TGF- β is essential for wound healing, overproduction of TGF- β can result in excessive deposition of scar tissue and fibrosis (Mutalik et al. 2005). The most commonly used modalities are pressure, silicone gel sheet, intralesional steroids, 5-fluorouracil (5 FU), cryotherapy, surgical excision, and lasers. They may be used either singly or, as is done more commonly, in combinations (Gupta et al. 2011).

Syed Yousuf Ali et al.
Emergence of Vitiligo Juxta-Keloid in a Female Patient – A Rare Case Report,
Medical Science, 2014, 4(12), 42-44,
http://www.discovery.org.in/md.htm

4. CONCLUSION

Case study has been made as it is a very rare case and interesting clinically. Vitiligos in association with multiple sclerosis, cutaneous amyloidosis, twenty-nail dystrophy linear scleroderma, Parry-Romberg syndrome, naevoid basal cell carcinoma syndrome, have been reported (Bhargava et al. 1995, Bhargava et al. 2001, Rajashekar et al. 2008, Bonifati et al. 2006, Creus et al. 1994, Muramatsu et al. 2005). The association of vitiligo juxta-keloid is very rare and has not been reported, which prompted us to report this case.

REFERENCES

1. Behl PN, Agarwal A, Srivastava G. Etiopathogenesis of vitiligo : Are we dealing with an environmental disorder ?. *Indian J Dermatol Venereol Leprol.* 1999, 65, 161-7
2. Bhargava P, Mathur KD, Bhargava R. Congenital leopard vitiligo associated with multiple sclerosis. *Indian J Dermatol Venereol Leprol.* 1995, 61, 375
3. Bonifati C, Impara G, Morrone A, Pietrangeli A, Carducci M. Simultaneous occurrence of linear scleroderma and homolateral segmental vitiligo. *J Eur Acad Dermatol Venereol.* 2006, 20, 63-5
4. Creus L, Sanchez-Regana M, Salleras M, Chaussade V, Umbert P. Parry-Romberg syndrome associated with homolateral segmental vitiligo. *Ann Dermatol Venereol.* 1994, 121, 710-1
5. Gopal K, Rama Rao GR, Kumar YH, Appa Rao MV, Vasudev P, Srikant. Vitiligo: A part of a systemic autoimmune process. *Indian J Dermatol Venereol Leprol.* 2007, 73, 162-5
6. Gupta S, Sharma VK. Standard guidelines of care: Keloids and hypertrophic scars. *Indian J Dermatol Venereol Leprol.* 2011, 77, 94-100
7. Jin Y, Birlea SA, Fain PR, Gowan K, Riccardi SL, Holland PJ,et al. Variant of TYR and autoimmunity susceptibility loci in generalized vitiligo. *N Engl J Med.* 2010, 362(18), 1686-97
8. Jindal R, De D, Kanwar AJ. Extensive keloids over lesions of air-borne contact dermatitis: An unusual manifestation. *Indian J Dermatol Venereol Leprol.* 2010, 76, 289-90
9. Le Poole C, Boissy RE.vitiligo. *Semin Cutan Med Surg*. 1997, 16(1), 3-14
10. Muramatsu S, Suga Y, Mizuno Y, Haseeqawa T, Komuro S, Kubo Y, *et al*. A Japanese case of naevoid basal cell carcinoma syndrome associated with segmental vitiligo. *Br J Dermatol.* 2005, 152, 812-4
11. Mutalik S. Treatment of keloids and hypertrophic scars. *Indian J Dermatol Venereol Leprol.* 2005, 71, 3-8
12. Rajashekar TS, Singh G, Rajkumar V. Segmental vitiligo and twenty-nail dystrophy: An unusual association. *Indian J Dermatol Venereol Leprol.* 2008, 74, 661-2
13. Rajkumar V, Okade R, Chakrabarty N, Yellappa K. Vitiligo associated with cutaneous amyloidosis. *Indian J Dermatol Venereol Leprol.* 2001, 67, 263
14. Wolfram D, Tzankov A, Pülzl P, Piza-Katzer H. Hypertrophic scars and keloids--a review of their pathophysiology, risk factors, and therapeutic management. *Dermatol Surg*. 2009, 35(2), 171-81