Multiple keratoacanthomas developing in healing plaques of Psoriasis

Vineet Relhan, Surabhi Sinha, Nita Khurana, Vijay K. Garg

ABSTRACT

A 22 year old male psoriatic patient presented with multiple reddish scaly plaques all over body. After hematological and biochemical investigations the patient was started on oral methotrexate 15 mg weekly. PASI score at the start of treatment was 26.2. After 3 months PASI dropped to 11.5, the dose of methotrexate was tapered to 7.5mg weekly and the patient was maintained on this dose and kept under monthly follow up. Four months later, the patient presented with reddish to hyperpigmented raised firm nodules having a central crater over the healing plaques of psoriasis. Few lesions showed self resolution over a period of 6-12 weeks. Histopathology of the lesion confirmed it to be Keratoacanthoma. We believe the most likely etiologic factors for the multiple KAs in our patient could be a genetic susceptibility stimulated by multiple causes.

Key words: Keratoacanthoma, Psoriasis, Spontaneous, Healing

INTRODUCTION

Keratoacanthoma is a common epithelial tumor of the skin characterized by rapid growth; histopathologic features similar to those of cutaneous sqamous cell carcinoma, and a certain tendency for spontaneous resolution. The exact nosology and classification of keratoacanthomas are a matter of debate. Now the favored view is that keratoacanthomas are low grade sqamous cell carcinomas, which in many cases will regress. The factors which may play a part include sun exposure, minor trauma to skin, immunosuppression, and genetic factors etc.

CASE REPORT

A 22-year-old male patient presented with multiple reddish raised scaly lesions all over his body associated with scaling over the scalp for one year. The patient was shopkeeper by profession dealing in stationary items, there was no history of smoking or tobacco intake. Patient had skin type V, and there was no history of diabetes, tuberculosis, or any other immunosuppressive disease. He had applied various emollients and topical steroids with no lasting relief; however, there was no history of tar application or phototherapy. Clinical examination revealed multiple well-circumscribed erythematous indurated scaly plaques on the scalp, bilateral upper and lower limbs, back, and on buttocks. Auspitz sign was positive over the lesions. The diagnosis of psoriasis vulgaris was confirmed by histopathological examination. The Psoriasis Area and Severity Index (PASI) was estimated to be 26.2. After hematological and biochemical investigations, X-ray chest and HIV test, the patient was started on oral methotrexate 15 mg weekly. He significantly improved on this dose, and PASI dropped to 11.5 after three months. The dose was decreased to 7.5 mg weekly, and the patient was maintained on this dose and kept under monthly follow-up.

Four months later, the patient presented with reddish to hyperpigmented raised nodules over the healing lesions of psoriasis. The nodules were firm and almost all had a central crater.

He also reported self resolution of few of the lesions over a period of 6 to 12 weeks without any specific treatment. Examination revealed the presence of multiple well defined dome shaped erythematous to hyperpigmented nodules, ranging in size from 5 to 15 mm, over both legs, especially shins, and a few over the arms and forearms [Figure 1]. The lesions were present on sun exposed as well as on non exposed areas. The nodules were characteristically present over
the healing plaques of psoriasis vulgaris. Few post inflammatory hyperpigmented macules and atrophic scars were seen at the sites where previous nodules had healed spontaneously.

Histopathological examination of the nodules showed a globular growth having a central crater containing keratinous material. The epidermis surrounding the crater was acanthotic and overlapped the crater laterally ("lipping"). Keratinocytes formed islands and cords and had pale eosinophilic cytoplasm [Figure 2]. These features were consistent with keratoacanthoma, and a diagnosis of multiple keratoacanthomas was made.

The patient was unwilling to opt for any therapy for the keratoacanthomas in view of spontaneous resolution of few of them. Weekly methotrexate was continued for control of psoriasis. The patient was followed up for the next 6 months, during which time keratoacanthomas continued to appear and some resolved leaving behind scarring. The patient was asked to apply tazarotene gel 0.1% daily over the residual lesions.

DISCUSSION

Keratoacanthoma (KA) is a rapidly evolving tumor of the skin, composed of keratinizing squamous cells and often resolving spontaneously. Multiple KAs may occur as part of the Ferguson Smith or Grzybowski’s syndromes or due to other acquired factors. These include ultraviolet (UV) light, viruses, chemical carcinogens, immunosuppression, and trauma.

The occurrence of KAs, mostly on sun-exposed regions of the skin, supports the role of UV light in their causation,[1,2] Prolonged therapy with PUVA or UVB has also been seen to result in large number of KAs.[3-5] Infective factors, especially viral, cannot be excluded as a cause. Various authors have found HPV-related DNA in KAs.[6,7] However, no virus could be demonstrated by Lu et al in their study.[8] Ghadially et al found tar and tobacco smoke to be significantly associated with KAs.[9] Multiple KAs have been known to occur in psoriatic patients treated with tar.[10,11]

Immunosuppression may also play a role as reported in patients with bone marrow transplants.[12]

Numerous reports have been published implicating trauma in the causation of multiple KAs. Ghadially et al, in their study on 238 patients, found 25 to have history of injury / skin disease causing epithelial disruption (psoriasis, lichen planus, discoid lupus erythematosus, rosacea, etc).[5,13,14] Pattee and Silvis reported 2 cases of KAs developing in sites of previous trauma.[15]

Vickers and Ghadially reported the first case of multiple KAs developing in a patient of psoriasis in 1961.[16] Clendenning et al reported KAs appearing in a patient of generalized pustular psoriasis.[17] Many hypotheses have been forwarded for the occurrence of KAs in psoriasis. Walder et al proposed that high levels of sun exposure in these patients may serve as an initiating factor.[18] The role of tar also cannot be ignored.[9,16] In addition, psoriasis itself is an epithelial injury and may contribute to development of KAs. Clendenning et al postulated that methotrexate therapy in these patients could cause immunosuppression and hence stimulate growth of KAs.[17] Annamalai et al proposed that chronic inflammation, tar, and phototherapy were responsible for multiple KAs and SCC in their patient.[19]

Treatment for multiple KAs is mainly medical. Intralosomal and systemic methotrexate and 5 fluorouracil have been tried with varying results.[20,21] Oral retinoids have also been seen to be highly effective.[22]

We believe the most likely etiologic factors for the multiple KAs in our patient could be a genetic susceptibility stimulated by either (a) epithelial injury due to psoriasis, the patient had history of psoriasis for more than a year, and psoriasis itself causes epithelial injury and chronic inflammation; moreover,
the lesions of keratoacanthoma appeared only in areas of healing psoriatic plaques; or by (b) immunosuppression due to methotrexate, which in this case seems less likely as methotrexate was given for a short duration; or by (c) Isomorphic phenomenon: There are single case reports of this phenomenon in keratoacanthoma.[23] The possibility of isomorphic phenomenon in this case seems unlikely as patient had psoriasis for the last one year and no history of keratoacanthoma lesions during that one year; or by (d) it may be sheer coincidence of two diseases occurring together, which again appears unlikely as the lesions of keratoacanthoma appeared only in areas of healing psoriatic plaques.

REFERENCES

1. Schwartz RA. The keratoacanthoma: A review. J Surg Oncol 1979;12:305-17.
2. Chuang TY, Heinrich LA, Schultz MD, et al. PUVA and skin cancer: A historical cohort study on 492 patients. J Am Acad Dermatol 1992;26:173-7.
3. Weinstock MA, Coulter S, Bates J, et al. Human papillomavirus and widespread cutaneous carcinoma after PUVA photochemotherapy. Arch Dermatol 1995;131:701-4.
4. Sina B, Adrian RM. Multiple keratoacanthomas possibly induced by psoralens and UVA photochemotherapy. J Am Acad Dermatol 1983;9:686-8.
5. Craddock KJ, Lauzon GJ, Tron VA. Multiple keratoacanthomas arising post – UVB therapy. J Cut Med Surg 2004;8:239-43.
6. Trowell HE, Dyall – Smith ML, Dyall-Smith DJ. Human papillomavirus associated with Keratoacanthomas in Australian patients. Arch Dermatol 1990;126:1654.
7. Pfister H, Gassenmaier A, Fuchs PG. Demonstration of HPV DNA in two keratoacanthomas. Arch Dermatol Res 1986;278:243-6.
8. Lu S, Syrjanen SI, Havu VK, et al. Known HPV types have no association with keratoacanthomas. Arch Dermatol Res 1996;288:129-32.
9. Ghadially FN, Barton BW, Kerridge DF. The etiology of keratoacanthoma. Cancer 1963;16:603-11.
10. Schwartz RA. Keratoacanthoma. J Am Acad Dermatol 1994;30:1-19.
11. Maddin WS, Wood WS. Multiple keratoacanthomas and Squamous cell carcinomas occurring at psoriatic treatment sites. J Cutan Pathol 1979;6:96-100.
12. Furnkawa M, Hamada T, Shibata H, et al. Keratoacanthoma ensuing from bone marrow transplant for chronic myeloid leukemia. Osaka City Med J 1992;38:83-8.
13. Ahmed AR. Multiple keratoacanthomas. Int J Dermatol 1980;19:496-99.
14. Giesecke LM, Reid CM, James CL, et al. Giant keratoacanthoma arising in hypertrophic lichen planus. Australas J Dermatol 2003;44:267-9.
15. Pattee SF, Silvis NG. Keratoacanthoma developing in sites of previous trauma: A report of 2 cases and review of literature. J Am Acad Dermatol 2003;48:S35-8.
16. Vickers CFH, Ghadially FN. Keratoacanthoma associated with psoriasis. Br J Dermatol 1961;73:120.
17. Clendenning WE, Auerbach R. Keratoacanthoma in generalized psoriasis. Acta Derm Venereol 1962;43:68.
18. Walder BH, Robertson MR, Jeremy D. Skin cancer and immunosuppression. Lancet 1971;ii:1282.
19. Annamalai R, Vasantha M, Umasevam M, et al. Multiple Keratoacanthoma and squamous cell carcinoma in psoriasis. Int J Dermatol 1981;20:606-7.
20. Melton JL, Nelson BR, Stough DB, et al. Treatment of Keratoacanthomas with intralesional methotrexate. J Am Acad Dermatol 1991;25:1017-23.
21. Goette DK, Odem RB. Successful treatment of Keratoacanthoma with intralesional fluorouracil. J Am Acad Dermatol 1980;2:212-16.
22. Street ML, White JW, Gibron LE. Multiple Keratoacanthomas treated with oral retinoids. J Am Acad Dermatol 1990;23:862-6.
23. Thappa DM. The isomorphic phenomenon of koebner. IJDVL 2004;70:187-89.

Cite this article as: Relhan V, Sinha S, Khurana N, Garg VK. Multiple keratoacanthomas developing in healing plaques of Psoriasis. Indian Dermatol Online J 2013;4:202-4.

Source of Support: Nil, Conflict of Interest: None declared