

Case Report

Keloid Formation on Herpes Zoster Scar in a Patient with Renal Transplantation

H. Özge Keseroğlu, MD, Meltem Önder,* MD

Address: Department of Dermatology, Gazi University Medical Faculty, Besevler, Ankara, 06500, Turkey

E-mail: monder@gazi.edu.tr

* Corresponding author: Dr. Meltem Önder, Department of Dermatology, Gazi University Medical Faculty, Beşevler, Ankara, 06500, Turkey

Published:

J Turk Acad Dermatol 2008; **2** (2): 82203c

This article is available from: <http://www.jtad.org/2008/2/jtad82203c.pdf>

Key Words: keloid, herpes zoster, isotopic response, renal transplantation

Abstract

Observations: Keloid is a benign, proliferative type of scar tissue. The exact pathogenesis of keloid is unknown. We report here a case of keloid after herpes zoster infection in a 21-year-old woman with renal transplantation. This is the first case of keloid formation on herpes zoster scar that developed after renal transplantation in English literature.

Introduction

Herpes zoster is an acute vesicular eruption resulting from reactivation of the varicella zoster virus when the immune system weakens with age or immune suppression. The rash may leave scarring, especially if it becomes secondarily infected. The incidence and severity of herpes zoster are markedly increased in recipients of solid organ and bone marrow transplants. In severely immunocompromised patients, because of secondary bacterial infection and superficial gangrene, healing may delay and scar formation may be seen [1].

A variety of cutaneous lesions have been reported in the affected area of skin after herpes zoster infection, including granulomatous reactions, malignancies, immune disorders, infections and others (e.g. acneiform lesions, reactive perforating collagenosis, keloid) [2, 3, 4, 5]. We report here a case of keloid after herpes zoster infection in a 21-year-old woman with renal transplantation.

Case Report

A 21-year-old woman admitted to our clinic with painful, pinkish-red masses on anterior and pos-

terior of her right shoulder. Four years previously, she had undergone renal transplantation because of focal segmental glomerulosclerosis. Four months ago, she had been hospitalized for severe herpes zoster infection and treated with acyclovir, 10 mg/kg intravenously, three times a day for 21 days. Also, for prophylaxis of secondary bacterial infection, sulbactam ampicillin 375 mg orally, once a day was given to patient for 10 days. At the end of the treatment, all vesicular lesions were totally crusted and healed with pruritic and painful masses which developed at the sites previously involved by herpes zoster lesions. She was still on immunosuppressive therapy (tacrolimus 0.09 mg/kg/day, mycophenolate sodium 360 mg twice a day). Physical examination revealed numerous pinkish-red, mildly tender, firm, irregular papules and plaques on the irregular hyperpigmented macule which had a dermatomal distribution (Right C4 dermatomal area)(Figure 1a, b). The lesions were not passing the midline. Any keloidal reaction was not detected elsewhere. The clinical diagnosis was keloid scar. She was successfully treated with intralesional triamcinolone acetonide injection. At the end of the second month, lesions become more superficial and painless (Figure 2a, b).

Discussion

Keloid is a proliferative type of scar tissue which results from excessive collagen depo-



Figure 1a, b. Keloid formation on healed herpes zoster lesions located on the right C4 dermatomal area.

sition following cutaneous injury in predisposed individuals [6, 7]. Together with genetic predisposition, some form of skin trauma play a major role in keloid development. The exact cause and clinical behavior of keloids are still unknown. Beside the molecular defects contributing to keloid scarring, some studies support that immunologic mechanisms play a role in keloid formation. Autoimmune anti-fibroblast antibodies have been detected in keloidal tissue. These antibodies may have a fibroblast stimulating role in the pathogenesis of keloids [7].

Requena et al. investigated cutaneous reactions at sites of herpes zoster scars at 16 patients and described only one patient with keloid formation [3]. In a different study, in which the potential effectiveness of herbal medicine used for herpes zoster in HIV-infected patients was evaluated, 23 patients with keloid formation as a complication was reported among 246 HIV-infected patients who completed the study [4].

The development of a new skin disorder at the site of another, unrelated and already

healed disease is known as *Wolf's isotopic response* [8]. Herpes zoster is the most common preceding disease of this response [5]. In the literature, variable latency periods between the infection and the cutaneous reaction were described, ranging from days to years [5, 8]. The pathogenesis of isotopic response is not totally understood. The viral, immunologic, vascular and neural etiologies were proposed [8]. The viral DNA was detected only in early (less than 1 month) post-zoster isotopic reactions [3, 5]. For this reason, herpes virus is not directly responsible for isotopic phenomenon.

We report here a case of keloid after herpes zoster infection in a 21-year-old woman with renal transplantation. This is the first case of keloid formation on herpes zoster scar that developed after renal transplantation in English literature. In our case, keloid formation on herpes zoster scar can be explained by isotopic phenomenon. Also, deeper, long standing herpes zoster lesions and development of secondary bacterial infection due to the long term immunosuppressive therapy may be the other possible mechanisms of this keloid formation.



Figure 2a, b. After the second intralesional steroid injection

References

1. Straus SE, Schmader KE, Oxman MN. Varicella and herpes zoster. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI. Fitzpatrick's Dermatology in General Medicine. 6th edition. New York. McGraw-Hill, 2003; 2070-2085.
2. Huang CW, Tu ME, Wu YH, Lin YC. Isotopic response of fungal granuloma following facial herpes zoster infections- report of three cases. *Int J Dermatol* 2007; 46: 1141-1145. PMID: 17988332
3. Requena L, Kutzner H, Escalonilla P, Ortiz S, Schaller J, Rohwedder A. Cutaneous reactions at sites of herpes zoster scars: an expanded spectrum. *Br J Dermatol* 1998; 138: 161-168. PMID: 9536241
4. Homsy J, Katabira E, Kabatesi D, Mubiru F, Kwamya L, Tusaba C, Kasolo S, Mwebe D, Ssentamu L, Okello M, King R. Evaluating herbal medicine for the management of herpes zoster in human immunodeficiency virus-infected patients in Kampala, Uganda. *J Altern Complement Med* 1999; 5: 553-565. PMID: 10630349
5. Ruocco E, Baroni A, Tripodi Cutri F, Grimaldi Filioli F. Granuloma annulare in a site of healed herpes zoster: Wolf's isotopic response. *J Eur Acad Dermatol Venerol* 2003; 17: 686-688. PMID: 14761138
6. Alster TS, Tanzi EL. Hypertrophic scars and keloids: etiology and management. *Am J Clin Dermatol* 2003; 4: 235-243. PMID: 12680802
7. English RS, Shenefelt PD. Keloids and hypertrophic scars. *Dermatol Surg* 1999; 25: 631-638. PMID: 10491047
8. Wolf R, Brenner S, Ruocco V, Grimaldi Filioli F. Isotopic response. *Int J Dermatol* 1995; 34: 341-348. PMID: 7607796