Journal of Clinical & Experimental Dermatology Research

Sugiura and Sugiura, J Clin Exp Dermatol Res
2018, 9:6
DOI: 10.4172/2155-9554.1000474

Letter to Editor Open Access

Suppressed of UV Skin Radiation Changes (Erythema and Pigmentation) Around Site of Skin Injury: A Case Report

Keiji Sugiura* **and Mariko Sugiura**

Department of Environmental Dermatology & Allergology, Daiichi Clinic, Nakaku, Nagoya, Japan

***Corresponding author:** Keiji Sugiura, Department of Environmental Dermatology and Allergology, Daiichi Clinic, Nittochi Nagoya Bld., Nakaku, Nagoya, Japan, Tel: +81522040834; E-mail: ksugiura@daiichiclinic.jp

Received date: September 11, 2018; **Accepted date:** October 11, 2018; **Published date:** October 15, 2018

Abstract

We report the case of a 48-year-old male with sunburn that did not develop UV radiation skin changes around injuries on his right arm. After the injury healed, the skin color around the scar had a different, lighter colored pigmentation. We theorized that this phenomenon might be a rare case related to TGF-beta 1 secreted by keratinocytes and platelets and iNOS up-regulated by UV radiation.

Keywords: Skin; Sunburn; Injury; TGF-beta; UV; iNOS; Pigmentation

Introduction

Sunburn is caused by UV radiation to the skin. The minimum degree of sunburn presents as erythema. Basically, with erythema the skin structures are not damaged, although the skin injury does not always maintain normal skin structure. Sunburn inflammation and injury are not same. The determinant skin changes between sunburn (erythema) and skin injury include defect of the epidermis or dermal layer, bleeding, neutrophils and platelets infiltration, fibrin deposition and up-regulation of related-cytokines in injury. In the case of erythema (sunburn), some epidermal cells are damaged as apoptosis of keratinocytes (sunburn cells) [1], lymphocytes infiltration, and blood vessels enlargement.

Case

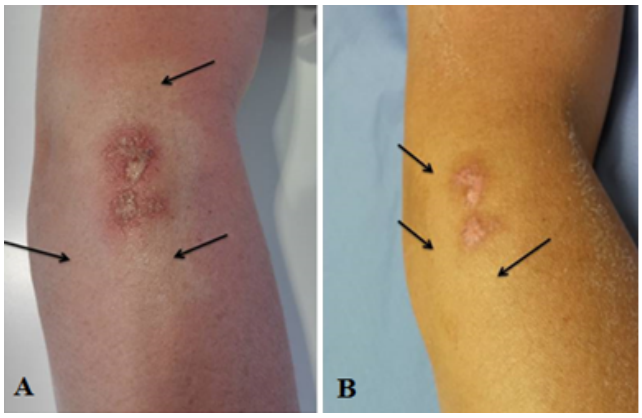

Figure 1: (A) Patient's right arm one day after receiving sunburn. There was no visible UV radiation skin change (erythema) around the injury; (B) Patient's right arm at 10 days after sunburn. The skin color around the scar was a different, lighter colored pigmentation.

We report the case of a 48-year-old male with sunburn who did not have UV radiation skin changes (erythema) around skin injuries (skin ulcer and erosion) on his right arm. The skin erosion and ulcer were caused by abrasion against a wall. The treatment for skin injury was kept clean by tap water without wound dressing. Three days after sustaining skin injuries, the patient was sunburned on his legs, arms and face during cycling. The skin around the injuries did not change in response to UV radiation effects (erythema; Figure 1A) after one day of sunburn; and only the skin close to the injury developed inflammation. Ten days after sunburn, his skin ulcer was improved, but the skin color around the injuries was different from the surrounding skin, with lighter colored pigmentation (Figure 1B).

Discussion

We hypothesized some reasons why the skin around the injuries did not change with sunburn. Inflammation associated with skin injury and UV radiation could be different from the pathological state, and some factors of inflammation by injury may play a preventive role against UV radiation skin change. Conversely, the immunosuppressive effects of UV radiation might suppress inflammation from injury, since there might not be pigmentation around the scar. Pigmentation after UV radiation has a close relation with melanocytes, keratinocytes and fibroblasts. In skin injuries (erosion and ulcer), there are not enough keratinocytes in these cells, and pigmentation could not develop. In this case, the reasons for these skin changes are not qualifying; we noticed an up-regulated cytokine in injury, transforming growth factor (TGF)-beta. TGF-beta has three isoforms, beta-1, beta-2 and beta-3. TGF-beta 1 is secreted by keratinocytes and platelets after injury [2], and it is an essential chemo-attractant factor for wound repair [3]. For hemostasis, mechanisms of coagulation act, and one of their actions is platelets agglutination. Platelets are accumulated at the site of the skin injury, and then platelet-derived TGF-beta1 is released for wound healing. Large amounts of TGF-beta1 possess immunosuppressive action and inhibition of melanocyte proliferation, T cell, B cell and NK cell [4-7].

From these findings, we consider one reason why the skin around the patient's injuries did not show sunburn changes was that TGF-beta 1 secreted by keratinocytes and platelets for injury healing suppressed UV skin radiation changes. Another key point is nitric oxide synthase

(NOS). NOS have three isoforms: inducible NOS (iNOS), neuronal NOS (nNOS), and endothelial NOS (eNOS). A mouse study reported that iNOS was required in a skin injury [8]. In addition, iNOS is up-regulated by UVB radiation and is an immunomodulatory mediator in sunburned skin [9]. iNOS is inhibited by TGF-beta [10]. TGF-beta in the skin wound could inhibit iNOS action in sunburn skin and injuries. In the case of simultaneous skin injury and sunburn at the same region, the action of improving injury could be dominant to that of sunburn to protect the body. It is interesting that green tea may be related to the skin difference shown in this case. This patient is Japanese, and he often drinks green tea. A previous interesting report [11] found that green tea down-regulated UVB radiation skin damage. Green tea may have promoted the patient's skin change.

References

1. Brash DE, Ziegler A, Jonason AS, Simon JA, Kunala S, et al. (1996) Sunlight and sunburn in human skin cancer: p53, apoptosis, and tumor promotion. J Investig Dermatol Symp Proc 1: 136-142.
2. Singer AJ, Clark RA (1999) Cutaneous wound healing. N Engl J Med 341: 738-746.
3. Mustoe TA, Pierce GF, Thomason A, Gramates P, Sporn MB, et al. (1987) Accelerated healing of incisional wounds in rats induced by transforming growth factor-β. Science 237: 1333-1336.
4. Kondo S (1999) The roles of keratinocyte-derived cytokines in the epidermis and their possible responses to UVA-irradiation. J Investig Dermatol Symp Proc 4: 177-183.
5. Kehrl JH, Roberts AB, Wakefield LM, Jakowlew S, Sporn MB, et al. (1986) Transforming growth factor beta is an important immunomodulatory protein for human B lymphocytes. J Immunol 137: 3855-3860.
6. Kehrl JH, Wakefield LM, Roberts AB, Jakowlew S, Alvarez-Mon M, et al. (1986) Production of transforming growth factor beta by human T lymphocytes and its potential role in the regulation of T cell growth. J Exp Med 163: 1037-1750.
7. Morikawa M, Derynck R, Miyazono K (2016) TGF-β and the TGF-β Family: Context-Dependent Roles in Cell and Tissue Physiology. Cold Spring Harb Perspect Biol 8: a021873.
8. Kitano T, Yamada H, Kida M, Okada Y, Saika S, et al. (2017) Impaired Healing of a Cutaneous Wound in an Inducible Nitric Oxide Synthase-Knockout Mouse. Dermatol Res Pract 2184040.
9. Seo SJ, Choi HG, Chung HJ, Hong CK (2002) Time course of expression of mRNA of inducible nitric oxide synthase and generation of nitric oxide by ultraviolet B in keratinocyte cell lines. Br J Dermatol 147: 655-662.
10. Chang DS, Seo SJ, Hong CK (2002) The effect of amniotic membrane extract on the expression of iNOS mRNA and generation of NO in HaCaT cell by ultraviolet B irradiation. Photodermatol Photoimmunol Photomed 18: 280-286.
11. Song XZ, Bi ZG, Xu AE (2006) Green tea polyphenol epigallocatechin-3-gallate inhibits the expression of nitric oxide synthase and generation of nitric oxide induced by ultraviolet B in HaCaT cells. Chin Med J 119: 282-287.