Unusual formation of keloids after each episode of recurrent herpes zoster in an HIV positive patient

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Abstract

Herpes Zoster (HZ) lesions are well known to heal with keloids. As immunity plays an important role in the development of abnormal scars and keloids, the latter is unusual in HIV where immunity is low. We report a rare case of recurrent HZ in an HIV-positive male where the lesions have healed with formation of keloids in both episodes. Within 50 days of last episode, he had an attack of herpes progenitalis.

Key words: HIV, keloid, recurrent Herpes Zoster

INTRODUCTION

Scars are formed by collagen produced by fibroblasts during the normal procedure of wound healing. Hypertrophic scars are formed when fibroblasts fail to stop producing collagen but remain restricted to original wound. When it overgrows the original wound, it is called keloid. The word keloid is derived from Greek word ‘chele’, meaning crab’s claw and suffix ‘oid’ meaning like. In most patients trauma remains the most important and singular provoking factor. Herpes Zoster (HZ) is caused by reactivation of Varicella Zoster virus, which stays latent in sensory nerve ganglion after causing varicella (chickenpox) as primary infection. HZ lesions are well known to heal with formation of keloids.[1,2]

Cellular immunity is more important than humoral immunity for preventing reactivation of latent herpes virus as well as for recurrence. HZ is often the first manifestation of HIV and it is regarded as marker of HIV in African countries.[3] Repeated HZ lesions are mostly described in immunocompromised patients and very rarely in immunocompetent hosts.[4]

Immunity plays vital role in the development of abnormal scars and keloids. So keloids are not common in HIV where immunity is low. We report an unusual presentation of recurrent HZ in an HIV-positive patient where keloids formed after each episode of zoster. He also had herpes progenitalis within 50 days of last episode of zoster.

CASE REPORT

A 31-year-old male presented with pain and vesicles on erythematous base since 3 days on left chest and back involving T5,6 dermatomes. He also had firm, irregular, hyperpigmented, solid lesions with overlying smooth shiny skin on chest, shoulders at region of left C4,5, T2 which appeared in last 6 months [Figure 1]. On questioning, he gave history of similar fluid filled lesions 1 year back at the site of latter lesions. ELISA test for HIV was positive. CD4 count was 273/µL. He was diagnosed as recurrent HZ (left T5,6) with formation of keloids at sites of old multidermatomal HZ lesions (left C4,5, T2). He was treated with tablet acyclovir 800 mg five times per day for 7 days.

On admission he had raised ESR (70 mm), diarrhea, plenty of pus cells in urine. His condition improved and was discharged on tenth day. He returned after 7 days with secondary infection at site of zoster lesion. Systemic antibiotic was started.
He returned after more than 35 days with pain, fever, and groups of vesicles on an erythematous base on the genitalia and right inner thigh [Figure 1]. Provisional diagnosis was of herpes progenitalis. He gave history of four to five similar episodes in the last year. Early keloids [Figure 3] were noted on the left chest (T5) at the place of last episode of HZ.

**DISCUSSION**

The mechanisms underlying the pathogenesis of keloids are not fully known. Both dominant and recessive modes of inheritance are known. HZ occurs 2–7 years after contracting HIV. So, it is certain that our patient had HIV 1 year back during his first attack of HZ. In our patient, keloids formed at sites of both episodes of HZ. HZ occurred in early stages of HIV when the immunity was decreasing (CD4 count 273/µL), but was not totally absent. The development of keloids in lesions old HZ lesion suggests that the related immunological process was still functioning.

Recent and past studies show that the active immune system[5] plays a vital role in formation of keloids. Eriguchi et al, reviewed records of surgical procedures done on HIV patients. They concluded that wound healing is poor in HIV and keloids are not formed.[6] In 2002, SK Goel and colleague[7] reported keloids on HZ scars in an HIV patient. They claimed it to be the first official report of its kind. Very recently Jose G. Castro and Luis Espinoza reported formation of keloid in a 65-year-old black Hispanic HIV positive man with CD4+ cell count of 343/µL, and HIV RNA level of 23,500 copies/mL.[8] However, keloids are more common in black Africans and a recent Nigerian study showed no significant difference in keloid formation in HIV and non-HIV patients.[9] But outside this single study on Negroid population, keloids have been rarely reported after HIV.

Both HZ and HSV can occur in an immunocompromised state. There is no direct relationship between the occurrence and recurrence of the two infections. Incidence of HZ and HSV are increased with HIV. In fact they are the most common cutaneous manifestation of IRIS (Immune Reconstitution Inflammatory Syndrome). But very few reports are there in the literature about their concurrent reactivation. According to our knowledge, most of them were in immunocompromised patients.[10] Hyun-Ho Park et al[10] opined that it might be due to difference in their rate of recurrence (HSV recurs many times while HZ occurs once in life time), age of recurrence (HSV occurs more in children and rate declines with age due to increasing immunity where as HZ occurs more in old age), reactivating cause (HSV is reactivated by trauma, stress but apparent provoking factors are less in HZ), and resident site...
of latency (HSV in neuronal cells where as HZ virus in satellite cells).

According to best of our knowledge, this is a very rare case report in the world literature, describing formation of keloids after each episode of Herpes Zoster in an HIV-positive individual.

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