

**UNUSUAL PRESENTATIONS OF KELOID**

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**ABSTRACT**

Keloid is a benign, hyper proliferative growth of dermal collagen that usually results from excessive tissue response to skin trauma with resultant scar tissue. The most common anatomical sites for keloid includes the chest, shoulders, earlobes, nape of the neck, upper arms, and cheeks. All forms of trauma in genetically predisposed individuals develops as keloid. The common causes are ear piercing, acne, inflammation, folliculitis, tattooing or burns and occasionally seems to occur spontaneously. We observed two cases of unusual presentations of keloid which developed from resolved varicella lesions and herpes zoster in a non-genetically predisposed individuals without any keloidal tendency. Hence we report the above cases for its rarity.

**Keywords:** Keloid, Herpes zoster, Varicella

**1.INTRODUCTION**

The term keloid, means 'crab claw', was first coined by Alibert in 1806, describes the lesion that expands from the original scar into the surrounding normal tissue.<sup>1</sup> Keloid is a benign condition, in which there is an excessive proliferation of dermal fibroblasts and abnormal production of dermal collagen as a result of impaired collagenase activity. Keloid may develop up to several years after minor injuries and persists usually for a longer period of time, and do not regress spontaneously.<sup>2</sup> The patients often present with severe itching, tenderness, pain, sleep disturbance, depression and esthetic concerns.<sup>3</sup> Keloids are 15 times as likely to occur in darker-skinned individuals because of genetic influences.<sup>4</sup> An incidence of 4.5% to 16% has been reported in black and Hispanic population.<sup>5</sup> Keloid may occur at any age. Varicella-zoster virus (VZV) causes two clinically distinct diseases. Primary infection results in varicella (chickenpox), is characterized by generalized vesicular rash with pleomorphic lesions and another form is herpes zoster (shingles) which results from reactivation of latent VZV from dorsal root ganglia, characterized by unilateral, grouped vesicles in a dermatomal pattern associated with radicular pain.<sup>6</sup> Reactivation may be triggered by trauma, sunburn, exhaustion, injection, elderly age and immunosuppression.<sup>7</sup> The common complications

are secondary bacterial infection, post herpetic neuralgia, allodynia, motor paralysis, pancreatitis, visual impairment, esophagitis, transverse myelitis, pneumonitis, scarring.<sup>8</sup> Keloid scar occurring at the site of resolved varicella and herpes zoster lesions has been rarely reported in the literature.

**Fig. 1: Reticulate pattern of Keloid**



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## CASE REPORTS

### CASE 1

A 55 year old male, agricultural laborer presented with 3 years history of multiple, raised linear scars. Lesions were gradually increased in size and appeared 1 month following an episode of varicella. Almost all the lesions were following a typical reticulate pattern involving the entire body and predominantly over the trunk (Fig. 1). Lesions were associated with severe itching and burning pain which disturbed the sleep at night. Patient was diagnosed as a case of pulmonary tuberculosis (TB) three and half years ago prior to the onset of varicella and had taken incomplete treatment. No history suggestive of keloid or hypertrophic scar in the past and among the family members.

### CASE 2

A 48-year-old female presented to us with the complaints of raised itchy, painful scars appeared after the healing of herpes zoster lesions over the left flank involving T<sub>8</sub>, T<sub>9</sub> dermatomes since 2 years (Fig. 2). She had not taken anti viral but used native topical medications which worsened the lesions resulted in infection and ulceration. With appropriate dressing and antibiotics, lesions healed after 2 months with scarring. Over a period of 6 months, she developed increasing in size of healed scars which was associated with severe pain, itching and hyperesthesia. There was no history suggestive of keloid in the past or in family members. Based on the clinical appearance, she was diagnosed as zosteriform keloid following herpes zoster.

Fig. 2: Keloid following Herpes zoster



## 2.DISCUSSION:

Keloid is a multifactorial disorder that involves both a genetic component and impairment of local mechano-physiological skin factors.<sup>9</sup> Most lesions reported from seborrhic areas suggesting the role of pilo-sebaceous unit and sebum production in the pathogenesis of keloid. Skin injury exposes the pilosebaceous unit to systemic circulation, and it triggers T-lymphocyte and proliferation of antigen-specific T-lymphocytes. This process continues as the keloid grows and further pilosebaceous units on the advancing border are disrupted.<sup>10</sup>

In case 1, keloid scars were observed over both seborrhic as well as non-seborrhic areas were progressively increasing in nature with persistent disease activity even after 3 years.

Generalized reticulate pattern of keloid following varicella has not been reported so far in the literature. Interestingly, patient had pulmonary tuberculosis and did not take proper treatment. The development of keloid in a patient with underlying tuberculosis suggests that the immunological mechanism remains active. Placik et al showed that the active immune system plays a vital role in formation of keloid.<sup>11</sup> Extensive nature of reticulate keloid in our patient was treated with compression stocking and intralesional steroids. Patient did not show much of improvement.

Ogawa reported that keloids are 15 times more common in people with darker skin suggesting increased activity of melanocyte stimulating hormone (MSH).<sup>9</sup> A study by Sharquie et al also observed that keloid occurs more in pigmented race.<sup>12</sup> Both our patients are dark-skinned individuals and they are more susceptible to develop keloid. In case 2, patient was not taking anti-viral and applied topical native medications. Healing of herpes zoster delayed and resulted in secondary infection and wound contamination. Because of the altered cellular response, there was an increased growth factor production like transforming growth factor (TGF- $\beta$ ) and platelet-derived growth factor (PDGF), which promoted increased proliferation of fibroblast and collagen synthesis and neovascularization and decreased collagen degradation resulted in keloid formation.<sup>13</sup> Patient was treated with intralesional steroid which showed transient regression followed by recurrence, probably could be due to persistence of foreign antigen within the keloid. Hence we report this case for its rarity.

## 3.CONCLUSION

These two cases represents keloids of rare presentation. Further studies are required for accessing the immunological factor and inflammatory mediators which involved in the pathogenesis of keloid in the above cases. This case report also highlights that keloid can occur as a complication of herpes zoster and varicella and effective treatment is necessary to prevent keloid formation.

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