A Rare Case of Isotopic Phenomenon - Herpes Coster, Lichen Striatus and PMLE

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1. Introduction

"Wolf's isotopic response" refers to the occurrence of a new dermatosis at the site of previously healed dermatosis. A number of factors including viral, neural, vascular, and immunologic factors have been implicated in the causation of this peculiar response but none has been proven conclusively.^[1]

The term isotopic response describes the occurrence of a new, unrelated disease that appears at the same location as a previously healed infection.^[2]

2. Presentation of Case

Presenting a case of isotopic phenomenon, which includes herpes zoster, lichen striatus and PMLE. A 36 years old female residing at Bandlaguda who is a cook by occupation, presented to the dermatology OPD with complaints of fluid filled lesions on limbs since 1 week which started appearing on the previously healed PMLE and lichen striatus lesions. She also complained of dragging pain in the left upper limb associated with constitutional symptoms and painful blisters. The patient had pruritus on the extensor aspect of the left forearm and hand since 1 week and the dragging pain on the same limb since 5 days. The pruritus was moderate in severity, more during night time i.e. there was diurnal variation and was gradual in onset and progressive in nature and relieved on scratching. The dragging pain was also gradual in onset, progressive in nature and unbearable. The patient is diabetic since 3 years and on medications but her blood glucose levels were above the normal levels. The patient also gave a history of being diagnosed for varicella zoster viral infection when she was 10 years of age. Surgical history was insignificant.

The patient had PMLE lesions on the arms and forearm 7 months back which were spontaneously resolving and recurring and now when the patient presented to the dermatology OPD, the lesions appeared to be healed lesions after topical steroid application for 1 month. The new herpes zoster lesions appeared at the site of healed PMLE lesions. PMLE lesions had seasonal variation, reoccurring in the summer season which was described as troublesome by the patient.

The patient had lichen striatus lesions on the arms and forearm 1 year back. They were gradual in onset and progressive in nature. She had moderate pruritus which was more during night in these lesions and initially pruritus was localised to the lesions but later it became generalised.

On cutaneous examination proper, lichenified linear shining plaques measuring 12*2cm were distributed on the lateral extensor aspect of the left arm. Grouped vesicular lesions were found on the same limb below the elbow on the extensor surface till the thumb and little finger. Few healing achromatic macules measuring 2*1mm size were found on the right arm. There was generalised scaling all over the upper limbs. Eyelids were hyperpigmented and scaly. Mucous membrane and appendages (hair, nails) were normal.

The patient was diagnosed to be suffering from herpes zoster intercostalis (C8, T1) and appropriate treatment was started.

The patient was started on valacyclovir 1000 mg TID for 5 days, diclofenac sodium & serratiopeptidase tablets and hydroxyzine hydrochloride tablets. The patient was also advised to use sunscreen and moisturiser. Patient had good improvement since strict sun protection advice was given.

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Lichen striatus lesions



Herpes zoster lesions



Healed PMLE lesions

3. Discussion

In 1955, Wyburn-Mason^[3] for the first time described the occurrence of a new skin disease at the site of another skin disease that had already healed. Such cases continued to mark their presence in literature till Wolf and Wolf^[4] in 1985 gave it a term; "isoloci response" (same locus). It was modified to "isotopic response" (same place) by Wolf et al.,^[5] and finally reframed as Wolf's isotopic response by Ruocco et al.^[2]

The dermatoses which are known to present in the site of previously healed disease include granulomatous reactions, malignant tumours, leukemic infiltrates, dermatoses secondary to immunologic dysfunction, infections, comedogenic reactions, and other miscellaneous conditions.^[1]

It is also essential that the second disease be a new and unrelated one. $^{\left[6\right] }$

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The most common preceding dermatosis is herpes zoster. Many dermatological entities can develop at the site of healed herpes zoster such as granulomatous processes, papulosquamous diseases, lymphomas, pseudo lymphomas, primary skin tumours, and metastatic deposits.^[7]

In most of the cases, initial dermatosis is herpes zoster, but the condition is also seen with herpes simplex virus, varicella, scrofuloderma, and also with thrombophlebitis.^[8,9,10] Although many consider isotopic responses a herpesspecific phenomenon, Wolf's definition included a wider range of initial dermatoses, which would have implications for the underlying pathophysiology.^[11]

Though the two dermatoses are not related in terms of their morphology, the seeds of second disease may be sown during the healing process of the first disease. This is because the healed skin may continue to show microscopic and physiologic changes for a long time after the initial insult and some of these changes may in fact be responsible for occurrence of new dermatosis.^[12]

The etiopathogenesis behind this response remains inconclusive. The neural hypothesis is the most accepted one. During herpes zoster, varicella zoster virus causes destruction of the A-delta and C nerve fibres. Besides, neurohumoral factors stimulate the development of the second dermatosis by releasing substance P, bradykinin, serotonin, vasoactive intestinal peptide, calcitonin generelated peptide, and α -melanocyte-stimulating hormone. These neuropeptides initiate the immunological cascade of the pathogenesis of the second disease. In addition to this, neurohumoral factors lead to unregulated activation of the immune system which contributes to the isotopic response. Allegue et al. proposed the role of tumour necrosis factor alpha (TNF-alpha) in the pathogenesis of this isotopic response. TNF-alpha is known to possess antiviral activity, and the levels are elevated in herpes zoster. Besides, TNFalpha is also elevated in psoriatic skin lesions. Therefore, it can be hypothesized that increased levels of TNF-alpha during the course of herpes zoster could serve as a triggering factor for the development of psoriasis over the healed sites.^[1] Immunologic hypothesis is relevant in this case as the patient is diabetic (immunosuppressive). Immunity protects us from any foreign antigens but on the other hand, if dysregulated can lead to tissue inflammation. A dermatosis affecting a certain body site may lead to changes in the regional immune system or formation of memory T cells which when triggered at a later date may give rise to a new dermatosis. The preferential occurrence of acyclovirinduced macular and papular lesions at the sites of previous herpes zoster may be due to the memory T cells, which may have appeared during the initial herpetic infection, and the accidental excessive activation of these cells by drugs could result in localized tissue injury.^[13] Intraepidermal memory T cells are abundant in such lesions. This is supported by the observation that some fixed drug eruptions initially appear at the site of previously traumatized skin.^[14]

Therapeutically, topical, intralesional and systemic corticosteroids, as well as acyclovir, have been tried with inconsistent results.^[12]

4. Conclusion

We are reporting a case of 3 clinical entities occurring in a short span, which had good improvement on treatment.

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