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LETTER TO THE EDITOR

Zosteriform Morphea Without History of Herpes Zoster Infection

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Morphea is a rare connective tissue disorder. Hardening and thickening of the skin due to an increased density of collagen are its characteristic features.[1] It has been classified into plaque or circumscribed, linear, generalized, morphea profunda (deep), pansclerotic and combined forms with wide clinical presentation.[2] Zosteriform morphea (ZM), which is described as a dermatomal distribution of lesions like herpes zoster clinically, is a rare entity. Most of them have been described on the same area of preceding herpes zoster infection as Wolf's isotopic response.[3,4] In this article, we report a case with usual morphea diagnosed histopathologically at unusual location without history of herpes zoster infection.

An 18-year-old healthy, male patient presented with complaints of hardness and discoloration of the skin at the right posteroinferior side of his trunk with a zosteriform distribution of $T_{10\text{-}12}$ dermatomes, over a period of two years. We detected no characteristic finding in his medical/family history and physical examination except indurated plaque that measured 9x6 cm in diameter with central hyperpigmentation among the xyphoid and umbilicus that extended to the lateral part of the trunk with atrophic lesions in a zosteriform distribution (Figure 1). We detected no history of herpes zoster or other skin lesions on the site of ZM, abnormal skin moisture, suggestive signs of other extracutaneous, musculoskeletal or systemic sclerosis. Serology for varicella-zoster virus tested negative for immunoglobulin M, but positive for immunoglobulin G. Anti-nuclear antibody, anti-human immunodeficiency virus, first-tier immunoglobulin M and immunoglobulin G enzyme immunoassays for Borrelia were negative. A skin biopsy revealed an interstitial lymphoplasmacytic infiltrate which surrounded eccrine coils and distributed among separated deep dermal thickened, hypocellular and swollen collagen bundles, and papillary dermal collagen elastic fibers (Figure 2). Laboratory studies including blood cell count, serum chemistry, and autoantibodies (antinuclear antibodies and anti-Scl-70 also called anti-topoisomerase 1) were all normal or negative. Clinical and pathological features were consistent with the diagnosis of morphea. A written informed consent was obtained from the patient.

Zosteriform term describes the dermatologic morphology attributing to the distribution of herpes zoster. Some diseases such as lichen planus, parakeratosis, common warts, fungal infections, nevus, and skin metastases have been described with this pattern.[3] Also, several types of cutaneous lesions such as granulomatous dermatitis, vasculitis and folliculitis, granuloma annulare, pseudolymphoma, keloid, sarcoidal granuloma, systemic lymphoma, leukemia cutis, lichenoid dermatitis such as lichen planus and sclerosis, cutaneous Rosai-Dorfman disease have been reported on the site of healed herpes zoster

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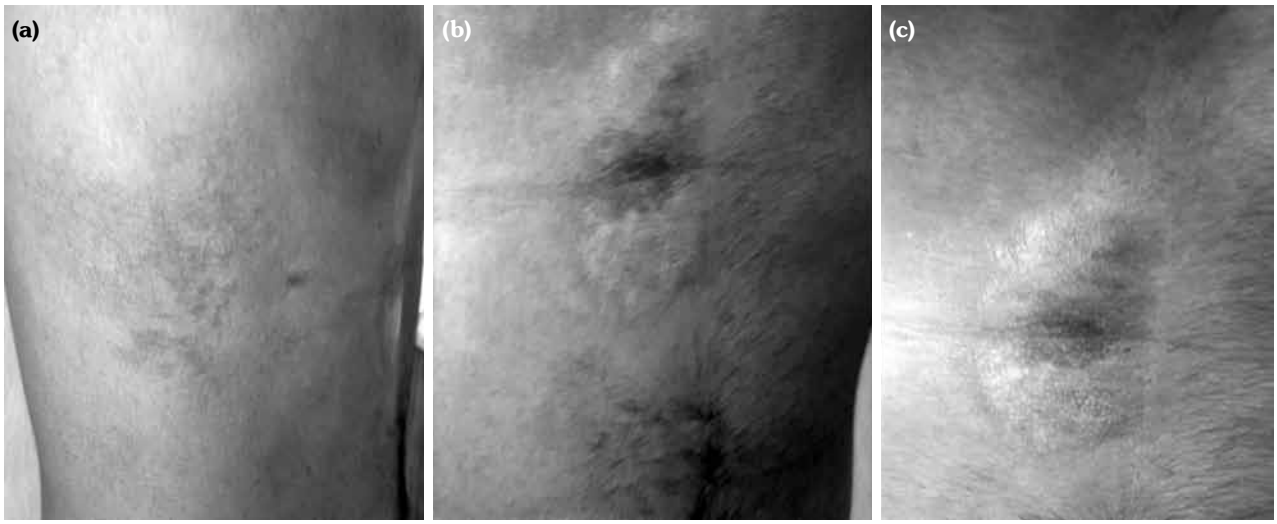

Figure 1. (a-c) Indurated plaque of zosteriform morphea with central hyperpigmentation.

lesions, and this reaction is called as Wolf's isotopic response.[5-7] Herpes zoster infections may cause atypical delayed-type hypersensitivity reaction to viral antigen or a tissue antigen alteration hypothetically.[4] A few cases of ZM localized on the site of improved herpes zoster infection have been reported previously.[3,4] Zoster sine herpete, which has a chronic radicular pain without rash due to varicella zoster virus (VZV) reactivation, should be ruled out from ZM.[7] Our patient did not have a radicular pain and rash on site of disease. However, VZV deoxyribonucleic acid was not performed. Clinically and pathologically, we evaluated this case as ZM. To our knowledge, only one case was reported as zosteriform pattern without evidence of herpes zoster like our case.[8]

Although trauma[9] and previous infection (Borrelia burgdorferi)[10] may incite morphea, the

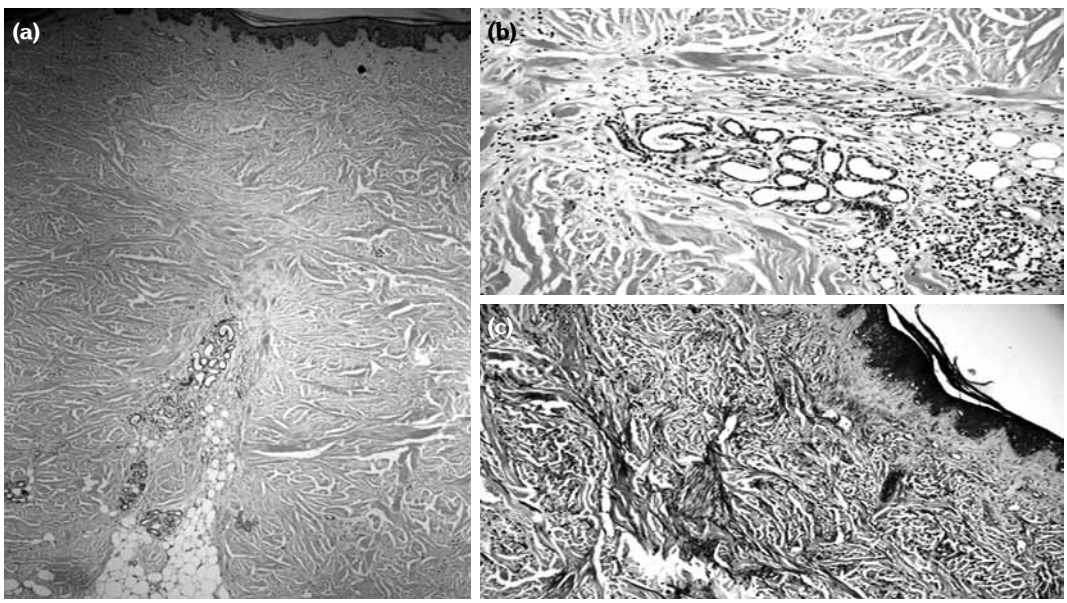

Figure 2. (a) There is an interstitial lymphoplasmacytic infiltrate distributed among deep dermal collagen bundles (H-E x 40). **(b)** Collagen bundles thickened, hypocellular and swollen, inflammatory infiltrates separate deep dermal collagen bundles, surround eccrine coils in the dermis (H-E x 200). **(c)** Papillary dermal collagen elastic fibers with Verhoeff-van Gieson stain (Verhoeff-van Gieson, x 200).

definitive etiology of morphea is not known. We do not know the reason of zosteriform distribution of morphea, but any neurogenic stimulation may be the cause of dermatomal distribution. Consequently, we presented this case with usual morphea diagnosed with histopathology and of which the location of morphea was unusual and worth to report.

Declaration of conflicting interests

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