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A Case of Wolf's Isotopic Response Presenting as Bullous Pemphigoid

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Dear Editor:

Wolf's isotopic response refers to a phenomenon in which a new skin disorder develops at the site of another, unrelated, and already healed skin disease. According to a report by Ruocco et al.[1] in 2014, there are approximately 200 cases of such a condition. Majority of primary infections includes infection by varicella zoster virus. In contrast the secondary skin lesion includes various cutaneous conditions such as granulomatous reaction, malignant tumor, dysimmune reaction, and morphea[2]. In this report the authors present a case of bullous pemphigoid (BP) with Wolf's isotopic response in a Korean female patient.

An 80-year-old female patient presented with an itchy erythematous patch, bullae, and ulceration on the right T3 dermatome. (Fig. 1) The bullous lesions had persisted for the last 2 months, and the patient had suffered from herpes zoster at the same site 6 months prior to this visit. Incisional biopsy of the affected lesion revealed subepithelial vesicles with dermal infiltration of lymphocytes, histiocytes, and eosinophils (Fig. 2A). Direct immunofluorescence with fluorescein isothiocyanate revealed linear deposition of immunoglobulin G in the basement membrane zone (Fig. 2B). With the final diagnosis of BP with Wolf's isotopic response, the patient was started on systemic steroid therapy and she showed clinical improvement. The patient has tapered oral methylprednisolone and is now free of new bulla with topical steroid and 1 gram of tetracycline and nicotinamide per day.

Recently Ruocco et al.[1] analyzed the previously reported cases of Wolf's isotopic response. Although numerous skin conditions have been identified as a result of Wolf's isotopic response, bullous disorders such as pemphigus vulgaris and BP were seldom seen. Up until now only one

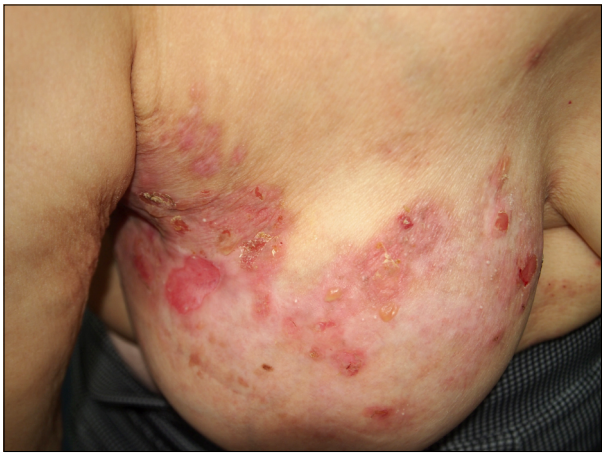

Fig. 1. Erythematous patch, bullae, and ulceration on right T3 dermatome.

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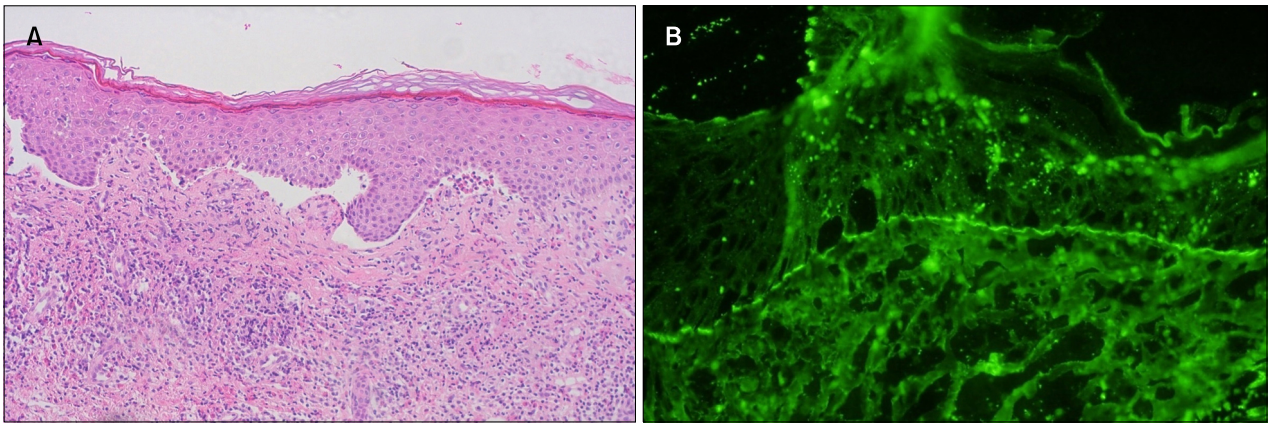

Fig. 2. (A) Subepithelial vesicles with dermal infiltration of lymphocytes, histiocytes, and eosinophils (H&E, x100). (B) Direct immunofluorescence reveal linear deposition of immunoglobulin G in basement membrane zone.

report has presented a case of pathologically confirmed BP at the site of previous herpes zoster[3]. In this report the patient experienced herpes zoster one month prior to the onset of BP. Distribution of the bulla was consistent with the area of herpes zoster.

Recent studies imply the possibility of connection between BP and nervous systems. It is traditionally known that patients with BP have a significantly increased risk for neurologic disorders such as dementia, stroke, Parkinson's disease, and multiple sclerosis. This fact is supported by the detection of neurological isoforms of both BP180 and BP230, the autoantigens responsible for occurrence of BP, in the central nervous system[4]. Furthermore, in one report the level of anti-BP180 antibodies in the serum was elevated in a patient with positive polymerase chain reaction assay of varicella zoster virus[5]. The level of anti-BP180 antibodies decreased after valaciclovir administration, and so were the blisters and erythematous patches which were confined to the C4 dermatome area.

Meanwhile there are studies suggesting that peripheral nervous systems may also be associated with BP. In one study biopsy of the skin previously infected with varicella zoster virus demonstrate a reduction in the dermal peripheral nerve network[4]. Unfortunately no study has yet succeeded in detecting the causative autoantigens in the dermal peripheral nerve fibers. Further studies to detect these autoantigens are required to establish a definite correlation between BP and varicella zoster virus infection.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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