SHOTA TAKASHIMA, MD
Department of Dermatology, Chitose City Hospital, Chitose, Japan

MITSUHITO OTA, MD, PhD
Department of Dermatology, Chitose City Hospital, Chitose, Japan

Herpes zoster triplex

A 77-year-old man presented with a 4-day history of painful eruptions on the left chest, right lower groin, and left thigh. He had been taking oral prednisolone 16 mg daily for interstitial pneumonia for 5 years. Ten days earlier, he had started to feel a stinging pain in these areas, but without eruptions.

Physical examination showed several grouped erythematous vesicles in the T2 dermatome of the left chest, L1 dermatome of the right groin, and L2 dermatome of the left upper anterior thigh (**Figure 1**).

Based on the presentation and a Tzanck smear of the lesions, a diagnosis of preherpetic neuralgia with herpes zoster triplex was made. The patient received intravenous acyclovir 750 mg/day for 7 days and continued to take the prednisolone. The lesions improved within 1 month, leaving scarring but no postherpetic neuralgia.

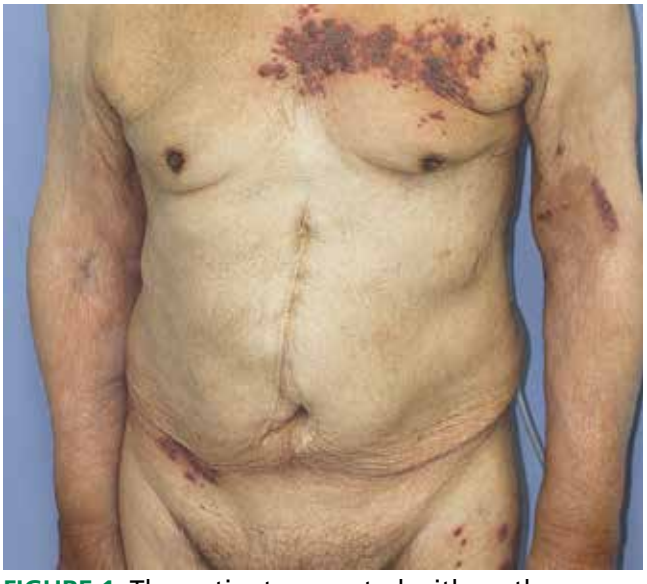

FIGURE 1. The patient presented with erythematous vesicles in the T2 dermatome of the left chest, the L1 dermatome of the right groin, and the L2 dermatome of the left upper anterior thigh in a band-like arrangement.

PREHERPETIC NEURALGIA

Herpes zoster usually occurs unilaterally in a single dermatome, with dermatomal pain appearing before the rash.[1] Preherpetic neuralgia may be misdiagnosed as myocardial infarction or renal colic, especially in a case of zoster sine herpete.

Making the diagnosis of preherpetic neuralgia in our patient was difficult because it occurred simultaneously in three dermatomes. At first, his symptoms were suspected of being a recurrence of his past illnesses, including aortic dissection, gallstones, and diverticulitis. Anti-varicella-zoster virus immunoglobulin (Ig) M antibody was not detected, and the IgG antibody titer did not increase.

It has been suggested that cellular immunity is more important than humoral immunity for the surveillance and control of reactivations of herpes viruses. Risk factors for reactivation are increasing age, cancer, acquired immunodeficiency syndrome, and immunosuppressive medications.[2,3] In addition, varicella-zoster virus can cause atypical lesions, including recurrent chickenpox, single-dermatomal herpes zoster with scattered rash, and herpes zoster in multiple dermatomes.[4]

Clinical suspicion for herpes zoster is important in the differential diagnosis of acute pain of uncertain origin, even if it occurs in multiple dermatomes in an immunocompromised patient. ■

doi:10.3949/ccjm.82a.14173

REFERENCES

1. **James WD, Berger TG, Elston DM**. Andrew's Diseases of the Skin. Clinical Dermatology, 10th ed. Philadelphia, PA: WB Saunders; 2006.
2. **Vu AQ, Radonich MA, Heald PW**. Herpes zoster in seven disparate dermatomes (zoster multiplex): report of a case and review of literature. J Am Acad Dermatol 1999; 40:868–869.
3. **Failla V, Jacques J, Castronovo C, Nikkels AF**. Herpes zoster in patients treated with biologicals. Dermatology 2012; 224:251–256.
4. **Kennedy PG, Steiner I**. A molecular and cellular model to explain the differences in reactivation from latency by herpes simplex and varicella-zoster viruses. Neuropathol Appl Neurobiol 1994; 20:368–374.

***ADDRESS**: Mitsuhito Ota, MD, PhD, Department of Dermatology, Chitose City Hospital, Hokkou 2, Chitose 066-0033, Japan; e-mail: ota@med.hokudai.ac.jp*