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NEUROLOGICAL PICTURE

Herpes zoster duplex bilateralis

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Herpes zoster (HZ), caused by reactivation of varicella zoster virus (VZV) from latency in a sensory ganglion, is almost always a condition involving a single dermatome.[1 2] Usually it occurs because of an age related decline in cellular immunity or immune compromised conditions. When spreading, it might involve one or two adjacent dermatomes or disseminate systemically (disseminated HZ).[3] The simultaneous reactivation of VZV from more than one ganglion is an extremely rare condition.[4]

Case report

A 64-year-old Arab woman was hospitalised with generalised weakness, urinary tract infection and a vesicular eruption on her back and thigh.

Four months previously she had been diagnosed with polymyositis associated antisynthetase syndrome, treated with 60 mg/day prednisone which was gradually tapered down to 20 mg/day by the time of presentation. She had type II diabetes and sustained a left basal ganglia stroke 2 years prior to her presentation.

On physical examination, she was afebrile, alert and fully oriented. She did not have organomegaly or lymphadenopathy without residual deficit from the stroke. She had a vesicular eruption, confined to dermatomes D8 on the left and L4 on the right (figs 1, 2). Laboratory tests, including complete blood count and basic biochemistry panel, were unremarkable, except for a C reactive protein level of 3.7. Diagnosis of HZ in these two dermatomes was established and treatment with intravenous acyclovir 10 mg/kg three times a day was initiated, followed by gradual improvement with crusting of the lesions. No recurrent zosteriform lesions were noticed during a follow-up period of 2 months.

Discussion

Following chicken pox, VZV establishes latent infection in peripheral sensory ganglia. Although the mechanisms of VZV reactivation from latency are unknown, its association with immunological decline suggests that an effective immune system maintains the viral genome in the latently infected cell and prevents viral replication and spread via retrograde axonal flow to the skin.[5] Although the latent viral genomes are present in many peripheral sensory ganglia,[6] HZ is usually confined not only to a single dermatome but also, and unlike reactivations of herpes simplex virus, to a single episode. Thus it seems that the biological context that supports the phenomenon of reactivation from a single ganglion under systemic immune compromised conditions also requires local factors: these may include the number of viral copies present in the tissue/cell or local trauma such as pressure on the nerve root or ganglion. The context that enables VZV reactivation seems to be so restrictive and dependent on local factors that simultaneous reactivation becomes an extremely rare phenomenon. While in the present case the mechanisms of bilateral VZV reactivation are unknown, it seems that the combined severe immunosuppression provided the required milieu to facilitate such a phenomenon. Whether it occurred independently in two separate ganglia or was caused by viral spread from one ganglion to another remains speculative.

Asaf Peretz, Johannes Nowatzky
Department of Internal Medicine, Hadassah University Hospital Mount Scopus, Jerusalem, Israel
Israel Steiner
Neurological Sciences Unit, Hadassah University Hospital Mount Scopus, Jerusalem, Israel

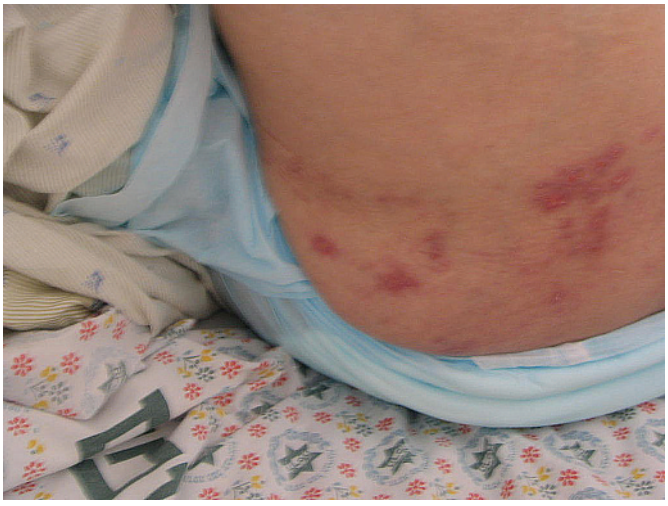

Figure 1 Zostiform vesicular eruption within the D8 dermatome on the left.

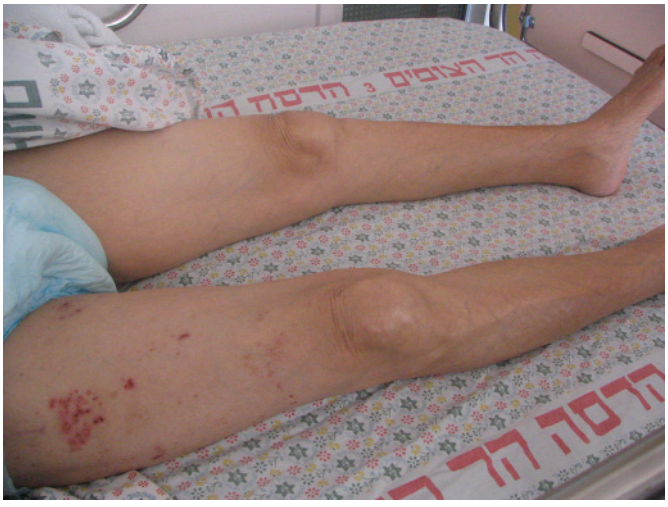

Figure 2 Zostiform vesicular eruption within L4 on the right.

Correspondence to: Dr Asaf Peretz, Department of Internal Medicine, Hadassah University Hospital Mount Scopus, Jerusalem 91240, Israel; asafp@bgu.ac.il

Informed consent was obtained for publication of figs 1 and 2.

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