Herpes zoster occurs most often in elderly and immunocompromised individuals, and rarely after surgery. We report 2 cases in which zoster developed in the contralateral dermatome distribution homologous to the surgical site. The mechanism by which unilateral surgery might affect homologous ganglia is likely to involve spinal cord pathways above the dermatomal level of surgical trauma.

We report, to our knowledge, the first cases of herpes zoster in a contralateral dermatome distribution homologous to sites of surgery weeks earlier.

**CASE 1**

A 55-year-old man who had undergone lumbar laminectomy 25 years earlier underwent successful cystoscopic removal of a right ureteral stone. A thoracolumbar magnetic resonance imaging scan revealed mild degenerative joint disease without spinal cord or nerve root compression. Three weeks later, he experienced the acute onset of severe left T12-L1 distribution pain that was not relieved by epidural injection of steroids. Zoster developed on the anterior surface of the abdomen just above the pubic ramus. Since treatment with 800 mg of oral acyclovir 5 times daily for 1 week, and 60 mg of oral prednisone daily for 5 days, he has been pain free.

**CASE 2**

A 63-year-old man underwent surgical correction of a Swan neck deformity of the left fifth and ring fingers. One month later, he developed right C8-T1 distribution, burning pain, and itching. After taking 500 mg of famciclovir 4 times daily for 7 days, both rash and pain resolved in less than 1 week.

Varicella zoster virus (VZV) causes chickenpox (varicella), becomes latent in cranial nerve, dorsal root, and autonomic ganglia, and reactivates decades later to produce shingles (zoster). Zoster is characterized by pain and vesicles on an erythematous base, usually limited to 2 or 3 dermatomes. The virus reactivates primarily in elderly and immunocompromised individuals. More than 100 years ago, classic studies correlated pathological changes of ganglionitis corresponding to ipsilateral rash.

The unique features of the cases described here reflect not only the close temporal relationship of surgery with the development of zoster, but also the development of zoster in the contralateral dermatome distribution homologous to the surgical site. The skin over the anterior surface of the abdomen and just above the pubic ramus is supplied by the L1 root, though skin resistance studies mapped the same area of skin to the T12 dermatome. Furthermore, the nerves supplying the ureter originate at T11 to T12 and L1. Thus, surgical trauma to either the skin or the ureter during cystoscopy could affect the T12 to L1 roots. It is un-
clear how local surgical trauma led to virus reactivation from ganglia on the opposite side in the same dermatome distribution since there are no defined anatomic pathways that connect ganglia even at the same level. Nevertheless, these cases are consistent with an earlier study showing that unilateral shingles can produce bilateral segmental damage to primary sensory neurons.7 Based on studies that demonstrated that nerve injury–induced tactile allodynia was mediated via ascending spinal dorsal column projections, and dependent on inputs to supraspinal sites,8 the mechanism by which unilateral surgery might affect homologous ganglia is likely to involve spinal cord pathways above the dermatomal level of surgical trauma. Such a notion is further supported by strong circumstantial evidence that argues against a peripheral mechanism (ie, via circulating factors) and in favor of a central mechanism; in particular, signaling via the system of commissural interneurons that is present in the spinal cord and brainstem.9 While our observations are not the first to describe an association between surgery and zoster, we are not aware of any literature that has shown that surgical trauma can trigger zoster in the contralateral dermatome distribution homologous to the surgical site.

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REFERENCES