An unusual facial ulcer

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A 69-year-old Caucasian man presented with a lesion on his forehead which “grew overnight” (*Figure 1*). It was located at the edge of a scar of a previously removed squamous cell carcinoma. The patient has had numerous squamous cell carcinomas and mild hyperlipidemia. *What is your diagnosis?*

Over the ensuing week, the patient developed two fresh ulcers on his forehead “overnight” (*Figure 2*). There was no erythema, flaking, pain, numbness, or tingling in the areas prior to the development of the ulcers. The two new lesions were tender. There was no known trauma. *Now, what is your diagnosis?*

DIAGNOSIS: Herpes zoster.

Herpes zoster is the reactivation of varicella zoster virus. It typically presents with a prodrome of pain, tingling, or tenderness, and then an eruption of grouped vesicles on an erythematous base occurs within a sensory dermatome. The most common complication is postherpetic neuralgia, which is a neuropathic pain syndrome that develops after the rash has resolved (1–3). It is typically described as burning, stabbing, or

Figure 1. Right frontolateral forehead. A 5-mm well-circumscribed superficial ulcer with a rolled pink border and surrounding telangiectasias at the medial edge of a scar. Superolateral to the lesion, there is a second scar from a previously treated nonmelanoma skin cancer.

Figure 2. The blue circle (A) is a superficial ulcer with an overlying hemorrhagic crust along the scalp fringe to the right of the midline. The red circle (B) is a superficial ulcer with a rolled border within the right eyebrow. The yellow circle indicates the primary lesion.

shocklike pain in the area where the rash was located (1, 4). Postherpetic neuralgia occurs in 14% to 20% of patients with herpes zoster (2).

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A vaccine for the prevention of herpes zoster and postherpetic neuralgia, Zostavax, was approved by the Food and Drug Administration in 2006 (5). It is a live virus vaccine composed of at least 19,400 plaque-forming units of the Oka/Merck strain of the varicella zoster virus. The potency of this vaccine is 14-fold higher than that used in children (2). Zostavax reduces the risk of developing herpes zoster by 51% and decreases the incidence of postherpetic neuralgia by 67% (3, 4); further, it decreases the burden of illness of herpes zoster by 61% and decreases the duration of postherpetic neuralgia by 57% (3, 4). Therefore, disease severity is decreased in patients who experience a herpes zoster outbreak despite vaccination (3, 4). Most recently, Zostavax was shown to decrease the risk of ophthalmic involvement, therefore decreasing the risk of serious vision-threatening sequelae (4). Zostavax is recommended by the Centers for Disease Control and Prevention for use in patients >60 years of age who are not immunocompromised (3, 4).

Our patient had received the Zostavax vaccine 2 weeks prior to his initial presentation. The patient is otherwise healthy and is not immunocompromised. To date, there are no reports of the Zostavax Oka/Merck varicella virus strain causing herpes zoster itself. However, there are reports of a herpes zoster–like reaction occurring at an average of 16 days after vaccination (4, 6). Wild-type varicella zoster virus DNA was isolated by polymerase chain reaction from the active lesions of the vaccinated patients with a herpes zoster–like reaction, confirming the reactions were likely not due to the vaccine (6). Thus, our

patient experienced a herpes zoster–like reaction from the vaccine given 2 weeks prior to the onset of his facial lesions.

The case highlights the importance of conducting a thorough history, including a vaccination history. It exemplifies how a vaccine-dampened outbreak of herpes zoster can clinically mimic nonmelanoma skin cancer. Therefore, truncated herpes zoster should be included in the differential diagnosis for nonmelanoma skin cancer for recently vaccinated individuals; a biopsy of the lesion is necessary for confirmation of disease.

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