

Case report

Herpes zoster–like reaction triggered by the recombinant vaccine against herpes zoster (Shingrix)

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Abstract

Zosteriform reactions triggered by the recombinant shingles vaccine (Shingrix) have been described in only two patients although many more cases that might be dermatomal seem to have been reported to regulatory agencies. It appears that these reactions may be caused by two different pathomechanisms: paradoxical reactivation of varicella zoster virus (VZV) or immune-mediated reactivity to already deposited virus or its antigens boosted by the vaccine. Our patient's cutaneous reaction favors the latter explanation because she developed quite a disseminated reaction that did not respond to oral valacyclovir. Apart from the interesting immunological underpinnings of the reactions, these reports suggest that physicians should test such lesions for the presence of VZV DNA to guide management. If viral DNA is absent and/or antivirals fail, a short-term course of oral corticosteroids should be considered.

Keywords: herpes zoster, vaccine, recombinant, adverse reaction, zosteriform, dermatomal, Shingrix

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Introduction

Shingrix (GSK) is a recombinant glycoprotein vaccine proven to be highly effective (97%) in preventing shingles and subsequent postherpetic neuralgia (1). It is recommended for immunocompetent adults age 50 and older, although efficacy is slightly reduced (91%) in those age 70 and above. The vaccine is administered in two intramuscular doses, 2 to 6 months apart (1).

Common side effects of Shingrix include localized reactions (pain, redness, and swelling at the injection site) and systemic adverse effects such as myalgia, fever, headache, shivering, fatigue, nausea, and stomach pain (2). Cutaneous side effects account for approximately 27% of all adverse reactions to Shingrix reported in EudraVigilance, the European database of suspected adverse drug reactions. As of July 6th, 2025, there were 24,338 reports of Shingrix-related adverse events (3).

Despite the relative frequency of cutaneous side effects, published literature includes only two case reports of dermatomal rash following Shingrix vaccination (4, 5). Here, we describe a third case of zosteriform eruption that could not be definitively classified as either true shingles or an immune-mediated reaction triggered by vaccination.

Case report

A 65-year-old Caucasian woman was referred for dermatomal skin rash, pain, and hyperesthesia persisting despite a full course of oral valacyclovir.

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Three weeks prior to evaluation, she received her first dose of intramuscular Shingrix (injected into the left upper arm). Five days post-vaccination, she developed lancinating pain on the right side of her trunk and right elbow. By day 7, a rash appeared on the right hemithorax, beginning at the midline. The rash consisted of multiple erythematous papules, two of which had clear vesicles. A dermatologist prescribed oral valacyclovir (1 g three times daily for 7 days). No varicella zoster virus (VZV) PCR testing was performed. Despite treatment, new lesions continued to emerge, and pain extended to unaffected areas (the right ear, right hemithorax, and right abdominal wall). She completed the valacyclovir course with worsening symptoms.

Her medical history was notable only for migraine headaches. She had chickenpox in childhood. At our evaluation (22 days post-vaccination, 17 days post-symptom onset), her primary complaints were persistent shooting pain, hyperesthesia, and rash. Examination revealed erythematous papules and macules in a dermatomal distribution (T1–T3) on the right trunk and arm (Fig. 1). No vesicles, crusts, atrophy, or scarring were observed.

After discussing the possible etiology, the patient consented to a skin biopsy. Histopathology showed superficial perivascular and focal lichenoid inflammatory infiltrate, and interface reaction with individual keratinocyte necrosis (Fig. 2).

Due to refractory pain and hyperesthesia (severely impacting daily function and sleep), she was started on oral prednisone (20 mg daily), which led to significant improvement in both skin lesions and symptoms. After 8 days, prednisone was discontinued, and she used topical mometasone furoate for an additional week. At her 4-month follow-up, she remained lesion-free with minimal neuropathic pain excellently controlled by several sessions of acupuncture. The patient chose not to receive the second dose of the vaccine.

Discussion

Shingrix is a recombinant adjuvanted subunit vaccine containing glycoprotein E (a VZV envelope component) and the AS01B adjuvant system (1). Glycoprotein E, a type 1 transmembrane protein, is critical for viral replication and host cell infection, eliciting both CD4⁺ T-cell and B-cell antibody responses (6). In individuals with latent VZV infection, vaccination boosts cellular and humoral immunity (1).

Cutaneous reactions (beyond injection-site effects) are rarely published. Bell et al. (7) described a patient with ulcerative colitis that developed a disseminated vesiculopustular eruption after both Shingrix doses. Thompson et al. (8) reported a woman with Crohn's disease that, 2 days post-second dose, developed a bullous eruption on the left axilla, arm, and chest wall. PCR was negative for herpes simplex virus (HSV)/VZV, suggesting bullous fixed drug eruption.

Only two prior cases of dermatomal rash post-Shingrix have been published. An immunocompetent German woman developed left V2 dermatomal vesicles (with scattered lesions on the back and abdomen) after the first dose. VZV DNA was PCR positive, confirming shingles with dissemination (4). Another woman developed right L3–L4 dermatomal vesicles 3 days post-vaccination (co-administered with flu vaccine). The rash resolved spontaneously without testing or treatment (5).

In our patient, the lack of response to valacyclovir and absence of VZV PCR testing complicate the diagnosis. Possible explanations include atypical shingles (dermatomal lesions with disseminated symptoms) or immune-mediated reaction from resident memory T-cells responding to vaccine antigen (glycoprotein E) or latent VZV antigens. Tissue-resident memory T-cells, which are known to persist for at least a year post-shingles (9), may react to vaccine-administered antigen (glycoprotein E) or a vaccine-boosted reaction of the cells to viral antigens previously deposited during latent viral shedding. Because VZV can be latently (asymptotically) shed in healthy subjects, it can be hypothesized that these virus-specific memory cells may be present in the skin even without preceding herpes zoster (10). Vaccine administration may then produce a zoster-like inflammation. Histological findings in the biopsy taken 3 weeks after the onset of the skin rash may be interpreted as a late stage of herpes zoster, its atypical form, or an immune reaction of the tissue-resident memory T cells spurred by the vaccine (11).

Within the Eudra Vigilance database reports, there were 724 cases designated as “vesicular rash” and “bullous dermatitis” (3). Many of the skin reactions were additionally described as “herpes zoster” or “vesicular rash with neuralgia.” Thus, these reactions may not be so rare.

Running title: Dermatomal reaction to recombinant zoster vaccine

Zosteriform eruptions post-Shingrix may not always indicate true shingles. Whenever possible, physicians should test lesions for VZV DNA (PCR) to guide management. If PCR is negative and/or antivirals fail, they should consider a short-term course of oral corticosteroids to alleviate the inflammation.

How should patients with this type of adverse reaction be counseled—which, apparently, is not so rare? If the reaction was simply caused by reactivation of the dormant virus, then a patient could be offered a prophylactic dose of oral valacyclovir or a similar agent for a week following vaccine administration. If it was an exuberant immune reaction to viral antigens triggered by the vaccine, then the second dose should be deferred. Alternatively, just before the time of the second dose, immunity against VZV (either humoral by enzyme-linked immunosorbent assay or fluorescent antibody to membrane antigen tests, or cellular by interferon-gamma enzyme-linked immunospot assay) may be checked. If sufficient, the patient may skip the dose. If not, the dose may be administered cautiously. Currently there is no evidence for the latter scenario. Because it seems that the reaction is not so rare, the manufacturer should be encouraged to organize a prospective clinical trial designed to answer these questions.

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Figure 1. Cutaneous lesions seen 17 days after the first signs appeared. Erythematous macules, papules and small plaques on the (A and C) right chest, (B) right back, and (D) right inner arm, corresponding to T1 to T3 dermatomes.

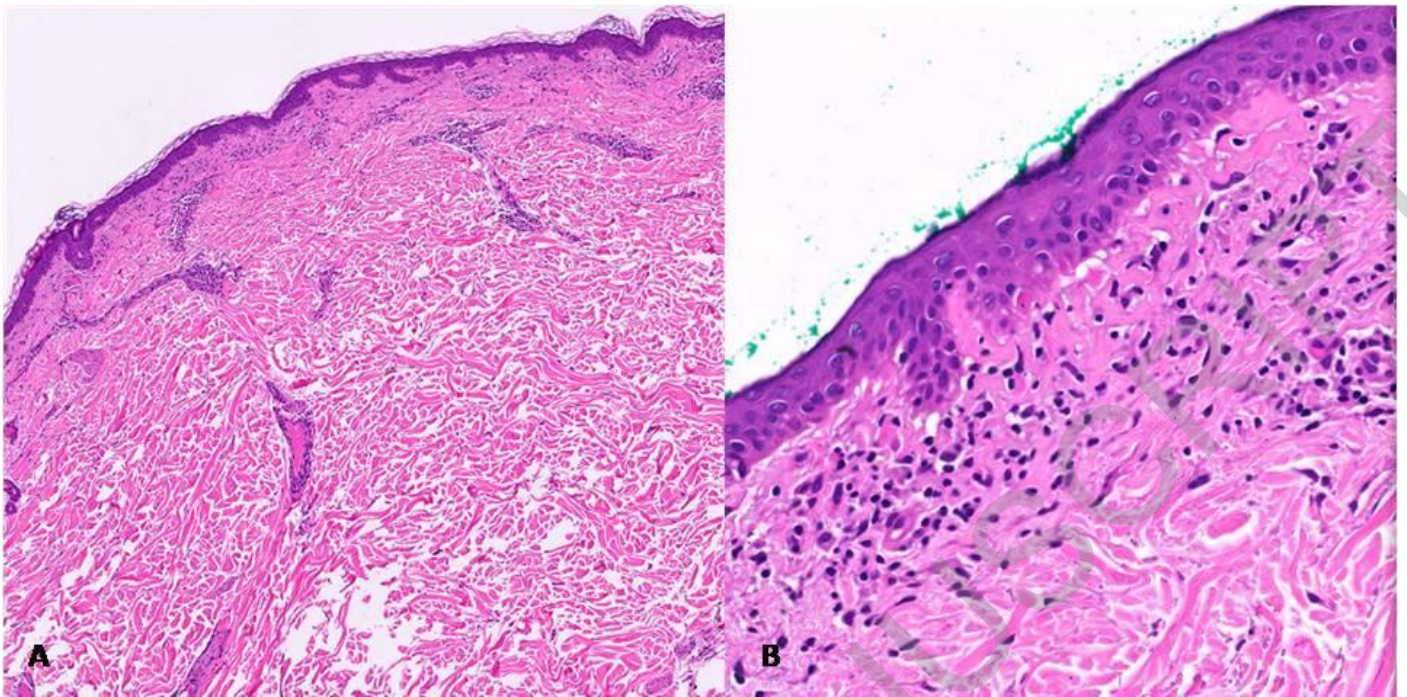


Figure 2. Histopathologic images of biopsy of one of the erythematous macules seen at (A) low and (B) high-power view; description in the text.