# Recurrent Dermatitis and Dermal Hypersensitivity Following a Jellyfish Sting: A Case Report and Review of Literature

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Abstract: Jellyfish envenomation often causes an immediate painful vesiculopapular eruption. Less commonly it can cause a type IV allergic hypersensitivity that manifests with delayed or recurrent cutaneous lesions at the primary site or distant from the primary site. These secondary reactivations may be related to high antijellyfish immunoglobulin levels, intracutaneously sequestered antigen, or cross-reacting venom. Immunomodulators such as pimecrolimus and tacrolimus and topical and intralesional corticosteroid therapy decrease this recurrent dermatitis. We report a case of a 9-year-old girl with a recurrent jellyfish dermatitis lasting more than 1 year after the initial envenomation. The dermatitis finally resolved after treatment with tacrolimus and intralesional triamcinolone acetonide therapy.

Jellyfish stings commonly produce an acute linear vesiculopapular eruption at the site of envenomation. This reaction is caused by nematocysts, toxin-releasing epithelial organelles found in jellyfish. Localized hyperhidrosis, lymphadenopathy, limb necrosis, gangrene, and contracture can also occur (1).

Unlike acute reactions to jellyfish stings, recurrent and delayed dermal reactions are rare. There are few reported cases demonstrating delayed and recurrent cutaneous reactions after jellyfish envenomation. These delayed reactions are believed to be due to an immunologic allergic-type mechanism. Our case is unique in that the delayed reaction recurred for more than 18 months after the initial jellyfish sting and that it was successfully treated with tacrolimus and intralesional triamcinolone acetonide injections. Through this case report and review of the literature

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we hope to increase awareness of the management of skin manifestations of jellyfish stings.

## **METHODS**

The English-language public literature was searched using PubMed.

# CASE REPORT

A 9-year-old girl presented to our department with red, itchy papules on the left lower leg (Fig. 1). The localized eruption first occurred after a jellyfish sting in South Carolina 1 year before presentation. When the patient was originally stung, she was treated with baking soda and vinegar. One week after the sting she developed a persistent red, itchy, inflamed plaque at the site of the sting. After failure to resolve with topical steroids, a skin biopsy was obtained (Fig. 2A,



Figure 1. Photograph of the recurrent dermatitis in the location of a jellyfish sting on the leg of a 9-year-old girl.

B) and histologic examination showed a superficial and deep dermal infiltrate composed of lymphocytes and many eosinophils with degranulation and early flame-figure formation. Periodic acid-Schiff (PAS) and acid-fast bacilli stains did not show the presence of any organism. A skin culture did not show acid-fast bacilli, mycobacteria, or fungus.

Initial management included oral cetirizine, a class II topical steroid ointment, for 3 weeks, which was transitioned to tacrolimus ointment for an additional 4 weeks, and the dermatitis resolved. After discontinuing tacrolimus, the red, itchy patch recurred and then resolved again with similar therapy. Four months after resolution, the irritation recurred, and intralesional triamcinolone acetonide therapy was initiated with two injections of 10 g/mL administered 3 weeks apart.

## DISCUSSION

Physicians commonly recognize acute cutaneous reactions from jellyfish envenomations, but they do not recognize recurrent cutaneous eruptions as easily. Pain is often appreciated during the acute event after the sting and is believed to be due to the toxic effect of the nematocysts, whereas delayed and recurrent reactions manifest with pruritus and are thought to be a delayed allergic reaction (1-4). Acute cutaneous findings are usually painful, linear, papular vesicular or urticarial reactions (1–3,5). Chronic cutaneous findings include localized fat atrophy, hyperhidrosis, vasospasm, hyperpigmentation, keloids, lichenification, lymphadenopathy, and scarring (1,2,6).

Few cases of recurrent eruptions and delayed eruptions have been reported after a single jellyfish envenomation (2,5,7,8). These recurrent eruptions,

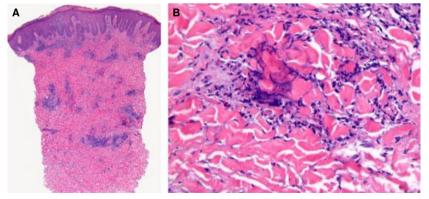


Figure 2. (A) Low-power view shows superficial and deep perivascular inflammation and epidermal hyperplasia (hematoxylin and eosin, 40x). (B) High-power view shows mixed inflammation with a prominent component of eosinophils. An early flame figure is seen with deposition of eosinophilic material on collgen bundles (hematoxylin and eosin,  $200\times$ ).

unlike the initial eruption, are believed to be due to an immunologic mechanism from high antijellyfish immunoglobulins, intracutaneously sequestered antigen, or cross-reacting venom (2,4).

The histology of recurrent cutaneous eruptions shows significant lymphocytic perivascular inflammatory infiltrates with eosinophils, similar to the histology in our patient. It is suggested that Langerhans cells and helper T-lymphocytes that elicit a type IV allergic reaction cause these histologic findings (9,10).

Identifying a specific jellyfish species that leads to recurrent dermatitis is difficult because they are rarely seen. The geographic location can aid in possible identification. Our patient was stung in South Carolina, where the most common types of jellyfish are the cannonball jelly (Stomolophus meleagris), mushroom jelly (Rhopilema verrilli), moon jelly (Aurelia aurita), lion's mane (Cyanea capillata), sea nettle (Chrysaora quinquecirrha), sea wasp (Chironex fleckeri), and Portuguese man-of-war (Physalia physalis), listed from the least to the most venomous. In the United States, most cases of recurrent cutaneous eruption after jellyfish stings occur off the Carolina beaches, with the most likely causative species being the P. physalis (11).

The goal of therapy for acute envenomation is to decrease any further nematocyst rupture. Vinegar has been shown to work for acute reactions caused by C. fleckeri or P. physalis, and baking soda works for C. quinquecirrha stings (12). Avoiding the use of fresh water is important because this activates nematocysts. Alcohol can also inactivate the toxin. The acute toxic phase has also been effectively treated using oral and topical steroids, although these interventions have not been equally effective at preventing or treating recurrent lesions.

Some reports show success with treating recurrent envenomation reactions using immunomodulators such as pimecrolimus (13) and tacrolimus (14). The effectiveness might be due to their ability to target T-lymphocytes. Pimecrolimus in combination with oral levocetirizine led to complete resolution of recurrent dermatitis after 45 days of treatment in one case report (13). Our patient developed a recurrent dermatitis and hypersensitivity reaction that was present for longer than 6 months and was responsive to topical steroids and tacrolimus therapy, with complete resolution after triamcinolone acetonide injections.

Further studies are needed to identify the exact pathogenesis of recurrent and delayed reactions. T-lymphocytes seem to play a major role and immunomodulators have been shown to be effective in treating these lesions. Physicians should be aware that not only do jellyfish stings produce an acute reaction, but lesions can also present as recurrent or delayed pruritic dermatitis-like reactions and immunomodulators and intralesional steroids work well in treating these lesions.

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