Abstract

Corals can elicit both toxic and allergic reactions upon contact with the skin. Clinical presentations vary depending on whether the reaction is acute, delayed, or chronic. Literature concerning cutaneous reactions to corals and other Cnidarians is scarce. Herein we report a case of delayed contact hypersensitivity reaction to coral and review the clinical and histopathological features of coral contact dermatitis.

Case synopsis

A 56-year-old man presented with a slightly pruritic, well-demarcated, erythematous, serpiginous plaque on the left anterior distal thigh (Figure 1). Two weeks prior, while snorkeling off the coast of Roatán, an island located in the Caribbean Sea, the patient’s left thigh accidentally brushed against a convoluted coral. Minutes after the incident, the site of contact became red, but was otherwise asymptomatic. The redness resolved over several hours time. Three days later a slightly pruritic, red plaque developed at the site of original trauma, which over the course of several subsequent days evolved into a well-delineated pattern similar in appearance to the coral with which he came into contact. The patient had not come into contact with coral prior to this incident.

A 3mm punch biopsy revealed a superficial and slightly deep perivascular lymphocytic dermatitis with focal spongiosis and some papillary dermal edema. No eosinophils were present and no exogenous fragments were seen, thus supporting a diagnosis of a delayed hypersensitivity reaction to coral (Figure 2).

Figure 1. A well-demarcated, erythematous, serpiginous plaque on the left anterior distal thigh.
The patient was prescribed a category II topical steroid to apply to the affected area twice daily. He never used the topical steroid, but the rash and associated pruritus resolved over the following two weeks.

**Discussion**

Corals are included in the large phylum, Cnidaria, along with the true jellyfish, box jellyfish, sea anemones, and hydra. Cnidarians are characterized by cnidocytes, specialized cells within the epidermis that contain organelles called cnideia. One type of cnideia, the nematocyst, contains a coiled filament that discharges upon contact with a foreign body. After the filament punctures the foreign body, the nematocyst releases its toxins including histamine, histamine liberators, quaternary ammonium compounds, proteins, 5-hydroxytryptamine, and catecholamines [1, 3]. Although the amount of toxin contained within each nematocyst is relatively small, a typical contact causes the discharge of thousands to millions of nematocysts leading to local and sometimes even systemic reactions [1].

Acute reactions to Cnidarian envenomation commonly result in mild to severe stinging and pruritic sensations with local erythema and edema. Vesiculation and necrosis are less common [1]. An acute reaction can occur immediately or within several hours of contact with a coral and typically resolves within several hours to days [2]. Histology is characterized by spongiosis with microvesicles, intracellular edema of the epidermis, and edema of the papillary dermis [3].

Delayed hypersensitivity reactions occur less commonly than acute reactions and are triggered by a sequestered antigenic component of the nematocyst [4]. A delayed reaction typically develops over several days to weeks. It is characterized by localized, firm, erythematous papules or plaques and sometimes later by erythematous lichenoid papules or plaques [5]. Histologic examination reveals parakeratosis, focal spongiosis, vacuolization of the epidermal basal layer, lymphocyte exocytosis, erythrocyte extravasation, wedge-shaped, subepidermal and perivascular lymphocytic infiltrates, and papillary dermal edema [3,4,6]. The histologic picture of more chronic lesions is characterized by acanthosis, parakeratosis, hypergranulosis, and a diffuse dense lymphocytic infiltrate in the upper dermis [3,4].

In some cases of delayed hypersensitivity to coral, histology shows atypical CD30+ lymphocytes and superficial, foreign-body granulomas with tuberculoid or sarcoidal features, although no foreign material can be found [3,4]. In these instances, it has been postulated that delayed hypersensitivity mechanisms and vascular damage may cause focal collagen degeneration and subsequent granuloma formation [4].

**References**