

Prolonged Hyperpigmentation Induced by Wild Parsnip Phytophotodermatitis

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Abstract

Wild parsnip (*Pastinaca sativa*) is an invasive Apiaceae species prevalent in prairies, fields, farmlands, and along roads and paths. Cutaneous contact with the sap of the plant, along with exposure of the impacted skin to ultraviolet light, leads to a chemical reaction that causes cellular damage in the epidermis. Acutely, the cellular damage manifests as tender and pruritic contact erythema with variable edema and bullae formation. This reaction is known as phytophotodermatitis. As the initial inflammatory response resolves, there is residual hyperpigmentation due to the melanocytic response to the parsnip sap that fades over the ensuing months. Although the acute and subacute stages of the cutaneous response to parsnip exposure are well documented, there is scant literature about the chronic effects. In this case report, we discuss long-term follow up of parsnip dermatitis, document the residual hyperpigmentation persisting over 4 years, and review treatment options.

1. Case Report

We previously reported the case of phytophotodermatitis in a 19-year-old woman caused by occupational exposure to wild parsnip (*Pastinaca sativa*) [1]. The rash distributed on the forearms was induced by contact with leaves and stems of this invasive plant during prairie restoration work, followed by ongoing sun exposure (FIG. 1). The linear and geometric configuration, corresponding to areas where photosensitizing sap from the plant contacted the skin, resulted in a tender and edematous eruption appearing about 24 hours after initial exposure. The rash was treated with topical steroid cream and the acute presentation improved during the next 2 weeks. The patient presented for follow-up for unrelated reasons 4.5 years later. Linear hyperpigmentation persisted in the areas previously affected by the acute photosensitivity eruption (FIG. 2). She had not pursued any treatments to mitigate the hyperpigmentation during this time, though reported occasional use of sunscreen. She reported that the patterned hyperpigmentation became accentuated with sun exposure during summers and faded only slightly during winters. Topical treatments for hyperpigmentation (including hydroquinone and tretinoin creams) were discussed but declined. Broad-spectrum sunscreen was recommended.

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FIG. 1. Acute phytophotodermatitis. Note linear edematous lesions with vesicles and deep violaceous erythema (Photo taken June 2016).



FIG. 2. Residual hyperpigmentation of the ventral forearm, 4.5 years later. Bronze linear patterning corresponding to areas of most severe initial inflammation (Photo taken December 2020).

2. Discussion

Phytophotodermatitis results from sequential exposure of skin to a photosensitizer followed by ultraviolet light. Furanocoumarins are photosensitizing agents present in many plants, including those in the Apiaceae (previously Umbelliferae; parsnip, celery, parsley), Rutaceae (citrus fruits including lime, lemon, grapefruit, orange) and Moraceae (fig) families. The cutaneous reaction reflects cellular damage upon exposure to ultraviolet A (320 nm - 400 nm), triggered directly (through interactions between the photosensitizer and target molecule) or indirectly (through reactive oxygen species) [1,2].

Hyperpigmentation results from several mechanisms, including melanocyte hypertrophy, increased melanosome density and melanocyte dendricity, and increased tyrosinase activity [1]. A recent liquid chromatography analysis of *P. salvi* seeds identified 13 furanocoumarins, including bergaoten, pimpinellin, methoxsalen, and psoralen [3]. Many of these compounds have known pathogenic implications for contact dermatitis as well as clinical utility in phototherapy.

Wild parsnip (*P. savita*) is a non-native plant species that has invaded native prairie grasslands throughout North America, Europe, and Australia. Originally introduced to the Americas in the 18th century during European colonization, the plant was cultivated as a root vegetable. Parsnip is a member of the Apiaceae family, which includes carrot and parsley, all of which contain furocoumarins. Over time, wild parsnip escaped cultivation and is now considered an invasive species as it outcompetes native plants. It is especially prevalent in prairies, along roadsides and railways, trails, and along disturbed edges of forests, fields, and farmlands.

Wild parsnip grows as a biennial or occasionally perennial. The stem grows from 2-5 feet high with a basal rosette of alternate, pinnately compound leaves. The flowers are yellow and arranged in a five-petal umbel clustered on slender stems [4]. The typical flowering period is late spring to summer (FIG. 3). The second-year plants die in the winter but each produce dozens of seeds which can be propagated through the environment and can remain viable in soil for up to four years [5].



FIG. 3. Invasion of *P. savita* in a field adjacent to a hiking trail. Note bright yellow flowers clustered as umbrels arising from long narrow stems.

In some U.S. states, wild parsnip is categorized as a Prohibited Noxious Weed, prohibiting the sale, transportation, or propagation of the plant [5]. Care must be taken to wear protective clothing when working around this plant outdoors. Ingesting these plants does not appear to be related to significant photosensitivity.

Treatment options for hyperpigmentation induced by wild parsnip are similar to treatment of other causes of postinflammatory hyperpigmentation [6]. Topical agents include hydroquinone, retinoids, and azelaic acid. Hydroquinone acts as a tyrosinase

inhibitor, preventing conversion of dihydroxyphenylalanine to melanin. Azelaic acid also inhibits this enzyme. Retinoids such as tretinoin reduce melanosome transfer and increase keratinocyte turnover. In addition, several topical botanical agents (including Kojic acid, soy, licorice, ascorbic acid, green tea, and nicotinamide) reduce hyperpigmentation through antioxidation as well as tyrosinase inhibition. Procedural treatments include chemical peels and laser treatments, though these carry some risk of worsening hyperpigmentation or causing mottled hypopigmentation [6]. Interestingly, available sources indicate that the hyperpigmentation from wild parsnip or furanocoumarins may last “up to 1-2 years” [4,7]. However, this case clearly highlights that the duration can extend much longer. Knowledge of the extended duration of hyperpigmentation is important for physicians, as the circumscribed pattern may simulate other causes such as physical trauma (burns, abuse, excoriations), primary dermatoses (linear lichen planus, morphea), congenital patterning (pigmentary demarcation lines), or drug reactions. Inquiring as to prior exposure to photosensitizing situations may avoid unnecessary work-up.

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