ORIGINAL ARTICLE

Phytophotodermatitis and/or Berloque Dermatitis: A Brief Review of the Topic in Light of Several Observed Cases

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SUMMARY

Phytophotodermatitis is a branch of dermatology encompassing phototoxic inflammatory reactions to certain chemicals found in some plants or fruits. These reactions are triggered by exposure to sunlight, causing inflammation and pigmentary sequelae. Many believe that Berloque dermatitis and phytophotodermatitis are the same condition, given their shared clinical and pathogenic similarities. However, others maintain that phytophotodermatitis is caused by plants or fruits, while Berloque dermatitis is caused by colognes or perfumes (especially those containing bergamot oil). We reviewed this topic in light of several cases observed at our center. We also provide a brief overview of current knowledge on the subject.

INTRODUCTION

In clinical practice, a considerable frequency of patients presenting with erythematous, bullous, and pigmented skin lesions has been observed. These manifestations, commonly diagnosed as Berloque dermatitis, occur with greater incidence during the beach season. However, they can currently be observed throughout the year, which justifies the need for this review.

Phytophotodermatosis (phyton: plant; phōs: light) encompasses an inflammatory cutaneous reaction, a form of contact dermatitis resulting from contact with various chemical products, such as furocoumarins and psoralens, which can be found in a variety of sources including plants and fruits. This is complemented by what is known as phytophotodermatitis.[1]

Historically, phytophotodermatitis dates back to ancient Egypt around 2000 BC, when vitiligo was treated with psoralens and sunlight. Later, in the 20th century, it regained importance among individuals who handled plants containing phototoxic substances such as psoralens and were exposed to ultraviolet light.[2] Finally, in 1942, Robert Klaver introduced the term phytophotodermatosis to unify the concepts that had been used up to that time.[3]

With regard to Berloque dermatitis, it is important to note that the word berloque originates from both the French berloque and the German berlock, terms that refer to a small charm, pendant, or amulet hanging from a chain. It was said that the lesions resembled these hanging amulets.

In 1916, Freund described hyperpigmented macules following sun exposure in areas of skin where cologne had previously been applied;[4] however, it was Rosenthal who, in 1925, coined the term to describe pendant-shaped pigmented streaks on the neck, face, arms, or trunk.[5] It is considered a subtype of phytophotodermatitis caused by various substances, whether artificial or natural, such as components of perfumes or cosmetics, in which bergamot essential oil was responsible for the lesional hyperpigmentation. It is a phototoxic reaction mediated by light activation of a psoralen (the active principle of furocoumarins), leading to the production of free radicals and a non-immunologically mediated inflammatory reaction. This differentiates it from photoallergic reactions, in which photosensitization to a substance occurs and a second exposure triggers a type IV immunological reaction.[6]

Although these differences exist, it may be stated that—except for the triggering agents and, for some authors, certain lesion locations—both processes are practically identical. it is acceptable to classify Berloque dermatitis simply as a subtype of phytophotodermatoses.

Adverse cutaneous reactions caused by plants constitute a group that ranges from simple irritations to severe allergies and include irritant contact dermatitis (due to physical or chemical agents), allergic contact dermatitis (as an immune reaction to plant allergens), phototoxicity (resulting from exposure to photosensitizers followed by ultraviolet light), and urticaria (immediate non-allergic reactions, such as histamine release from nettles, or allergic reactions to foods or plant products).[7]

Numerous plants, fruits, and substances have been mentioned as agents implicated in phototoxicity; the most frequently cited are listed below:

Citrus fruits: (grapefruits, limes, lemons, oranges) (Fig. 1A); in our setting, lemons in particular, which are squeezed into soups, ceviches, or other foods and are commonly followed by sun exposure. When these citrus substances are present in alcoholic beverages, the lesions tend to appear in the areas where the drinks may have spilled.[8–9]

Peppers: (Fig. 1B) are solanaceous plants that contain capsaicin and may be associated with intense cutaneous irritation.[10]

Bergamot: Citrus bergamia, commonly cultivated in tropical and subtropical regions.

Rue: (Fig. 1C) Ruta graveolens. Commonly known as rue, it has multiple uses as an analgesic, anti-inflammatory, antiandrogenic, antihyperlipidemic, and hypoglycemic agent. It contains psoralens and furanocoumarins such as 5- and 8-methoxypsoralens, which induce a phototoxic reaction when activated by sunlight.[11] Additionally, rue is used as an esoteric remedy by healers to treat the "evil eye," as reported by Córdoba and collaborators, in which a rue preparation was applied to the entire body except the head, and dermatitis developed in the uncovered areas.[12]

Celery: (Fig. 1D) Apium graveolens (celeriac, celery root, leaf celery), widely consumed for its nutritional properties, contains furocoumarins and psoralens, especially in the roots of the plant, which may cause phototoxic reactions. These reactions do not occur through ingestion or normal handling, but rather in individuals with massive exposure to this vegetable, such as supermarket workers, food industry laborers, or farmers.[13]

Carrots: Few cases of phototoxicity caused by carrots have been described. One report cites the case of a woman who used a decoction of wild carrots as moist compresses on her foot three times a day, with sun exposure during the procedure, resulting in inflammation with residual pigmentation. Another case report describes centrally located pigmented facial lesions in a patient who had used a sunscreen containing carrot extract.[14]

Byrne and colleagues reported the case of a 5-month-old infant with an erythematous eruption on the face, chest, and hands, accompanied by bullous lesions, who had eaten carrot and parsnip purée the previous day while being exposed outdoors to sunlight.[15]

Buttercup or Ranunculus: This is a highly irritating flowering plant of bright yellow color, used as livestock

forage, which contains the unsaturated lactone protoanemonin. This toxic substance is released when the plant is crushed, irritates the skin, and upon sun exposure produces photosensitivity, with inflammation, erythema, and blisters that last for weeks and leave residual pigmentation.[10]

Figs: (Fig. 2A) Surprisingly, the fig tree is also among the agents that produce photosensitivity, as the leaves and sap of the fig tree contain photoactive furocoumarins. This places the effect of this plant on the skin within the broad group of conditions that cause cutaneous irritation or burns.[16–17]

Mulberries: (Fig. 2B) belonging to the Moraceae family, produced by the mulberry tree. Phytophotodermatitis caused by skin contact with mulberries has been reported, although it is much less frequent than with citrus fruits. Lesions are located mainly on the arms due to contact with the plant and may occasionally produce inflammatory erythematous and bullous lesions, as in the case reported by Kim and collaborators.[18]

Fennel: Although fennel has gained importance as both a dermatological treatment (for acne, hyperpigmentation, anti-inflammatory effects, improvement of muscle tone, etc.) and an oncological agent (particularly in the treatment of melanoma through inhibition of nuclear factor kappa B [NF-$_K$B] and matrix metalloproteinase-9 [MMP-9]), the use of fennel seeds in dermatology presents a relevant limitation. Their application is associated with increased cutaneous photosensitivity, which heightens susceptibility to ultraviolet radiation–induced damage, including sunburn, phototoxic reactions, and photoaging processes.[19]

Figure 1. A: Lemon tree. B: Hot pepper. C: Rue. D: Celery
Photographs taken from the author's garden.

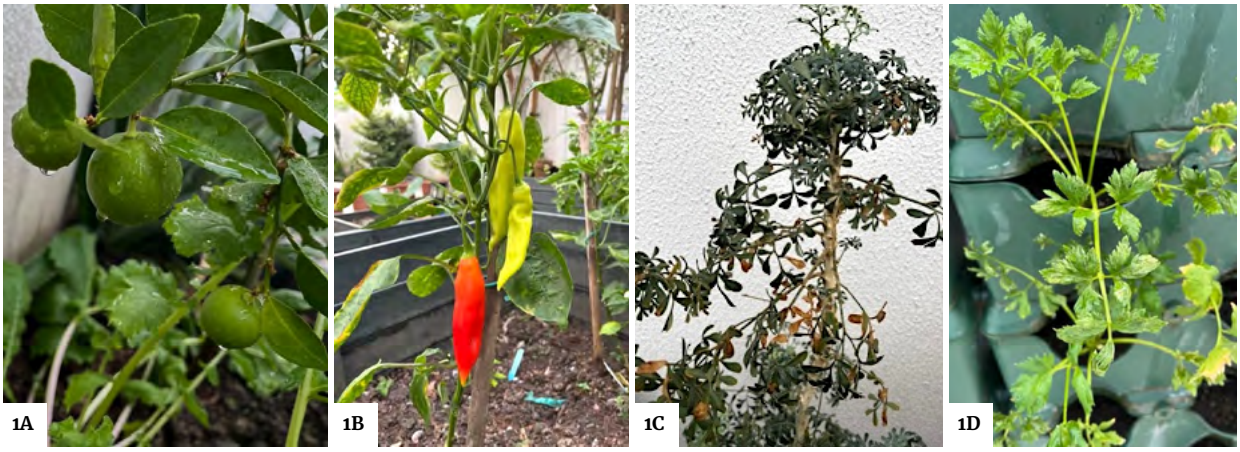

Figure 2. A: Fig tree. B: Mulberry tree. C: Sage D: Parsley
Photographs taken from the author's garden.

Giant hogweed: A plant of the genus Heracleum, which produces phototoxic cutaneous lesions resembling burns. These are triggered because its sap contains furocoumarins that, upon sun exposure, produce the aforementioned lesions. These painful lesions with large blisters leave residual hyperpigmented areas and sometimes prolonged photosensitivity. They are commonly seen in individuals who collect plants in agricultural environments, as in the patient reported by Jarmendi.[20]

Parthenium: Also known as congress grass or bitter weed, it is an annual herb native to the West Indies and Mexico but currently widespread worldwide. Exposure to this plant can cause phytophotodermatitis, producing erythema, blisters, and pigmentary changes. It contains psoralens in particular, which induce dermatitis upon exposure to UV light.[21–22]

Dill: Cutaneous reactions caused by this plant may present as allergic contact dermatitis or as phytophotodermatitis; in the latter case, they are related to the psoralens it contains and result in a burn-like reaction. In some studies, dill appears among the frequent agents involved in the development of this condition.[23]

Sage: (Fig. 2C) As occurs with other plants, the basic factor is the presence of psoralens, which are deposited on the skin and, upon sun exposure, provoke cutaneous inflammation.

Parsley: (Fig. 2D) This plant, for reasons similar to those explained in the previous cases, can cause phytophotodermatosis as a result of excessive handling, particularly during kitchen or restaurant work.[24]

Wild parsnip (cow parsnip, Heracleum lanatum or Indian celery) is a root vegetable similar to the carrot. Cow parsnip oil, present in all parts of the plant, contains furanocoumarins that cause phytophotodermatitis when exposed to sunlight, leading to residual hyperpigmentation due to psoralen-induced changes in melanocyte size and number, increased tyrosinase activity, and increased melanosome density. It should be noted that this pigmentation may persist for years.[25–26] Carlsen et al. published 19 cases of phytophotodermatosis in hospitalized children, 17 of which were related to contact with wild parsnip.[15–27]

Recently, cases of phytophotodermatosis induced by alcoholic beverages, such as the mojito—a liquor originating from the island of Cuba and composed of rum, lime, sugar, spearmint or mint, and soda—have been reported.[28]

Galván-Pérez del Pulgar et al. reported nine cases of hyperpigmentation associated with mojito preparation, with lesions located exclusively on the dorsum of the hands. They proposed the term "dorsal acropigmentation due to mojito preparation" to define an occupational dermatosis affecting the dorsum of the hands of bartenders who prepare mixed drinks such as mojitos that contain lime.[29]

Mexican beer, which is served with a slice of lime placed in the neck of the bottle, also poses a risk, since squeezing the lime may cause citrus juice to drip onto the skin.[30]

Similarly, the margarita cocktail contains lime juice or other related citrus fruits. Either through squeezing limes to prepare the drink or through contact with spilled beverages on the skin, always followed by sun exposure, this has led to the designation "margarita burn," which also falls within occupational dermatoses, as it is frequently observed in bartenders or similar occupations, as reported by Cochran and collaborators.[31]

Another preparation that has also been reported as a causal agent is the popular sangria, which among its multiple components also includes lime.[32]

Likewise, various activities—whether recreational, such as excursions or outdoor leisure activities; agricultural work; or professional activities such as bartending, in which citrus-containing beverages are handled—may be associated with a higher frequency of the disease. The use of herbal remedies should also be considered as a possible cause.[33]

As expected, the disorder is more frequent in warm and humid climates, where there is a greater abundance of

phytotoxic plants. Thus, phytophotodermatitis can be classified both as an occupational dermatitis (affecting military personnel, farmers, cooks, gardeners, waiters, etc.) and as a recreational dermatitis, which may resemble occupational exposure but occurs during leisure activities.[34]

Other triggering factors have been described, such as photodermatitis induced by citrus-based hand sanitizers, as reported by Lee et al.,[35] as well as two reported cases of bullous photodermatitis caused by babchi oil. Babchi or bakuchi (Psorylea corylifolia) is a plant found in India, China, and southern Africa, used in Ayurvedic medicine, particularly in the treatment of vitiligo.[36]

ETIOLOGY

Contact with photosensitizing substances found in certain plants or fruits, combined with exposure to sunlight, results in a cutaneous phototoxic reaction, leading to inflammatory skin reactions with edema, blisters, and other sequential manifestations.

Photodermatitis occurs when the skin comes into contact with psoralens or other furocoumarins and is subsequently exposed to UV light. These substances are generally found in plants such as those mentioned above and, less frequently, in other forms with the same underlying mechanism—that is, photosensitizing substances commonly found in seeds, flowers, or stems of various plants, most often belonging to the Rutaceae family, such as lemon, lime, and grapefruit, as well as celery, parsnip, and others.[22]

Furocoumarins are a type of organic compound synthesized by many plants as a natural chemical defense against predators such as fungi, bacteria, and insects.

It is noteworthy that furocoumarin levels vary among plants. Total furocoumarin concentration was highest at 23,215 ng/g in fresh parsley and 21,858 ng/g in whole grapefruits; 14,580 ng/g in lime juice; 9,151 ng/g in limes; 1,551 ng/g in lemon juice; 396 ng/g in celery root; 335 ng/g in parsnip; and 68 ng/g in carrot.[37]

Interestingly, prospective studies with follow-up periods of 20 to 26 years comparing individuals who consumed citrus fruits less than twice per week with those who had high consumption of furocoumarin-rich citrus have suggested a possible association with basal cell carcinoma, squamous cell carcinoma, and melanoma.[38–39] Supporting these studies, furocoumarins have been detected in the skin of animals after oral consumption of these products, which may help explain tumor development.

It is essential to understand that phytophotodermatitis is fundamentally a non-allergic contact dermatitis, meaning that this reaction is not mediated by the immune system but is directly due to the toxic action of photosensitizing substances on skin cells. The intensity of the reaction depends on factors such as the concentration of photosensitizers, the duration and intensity of sun exposure, and individual susceptibility.[40]

In summary, the pathophysiology of phytophotodermatitis involves a two-step process. First, the skin is exposed to photosensitizing substances present in plants through direct contact. Once these compounds penetrate the skin, they remain latent until activated by exposure to sunlight, particularly UVA radiation (320–380 nm). After exposure, a photochemical reaction occurs, leading to the formation of reactive oxygen species and subsequent damage to skin cells, thereby triggering the characteristic symptoms of phytophotodermatitis.[41]

CLINICAL FEATURES

Symptoms are varied but fairly characteristic and usually appear 24–48 hours after exposure. The condition begins with persistent erythema, which may range from moderate to severe, often presenting as linear streak-like lesions and sometimes complicated by the presence of vesicles and/or blisters in more intense cases. It is important to clarify that this is not a sunburn but a phototoxic reaction. These lesions are accompanied by skin edema, desquamation, and subsequent post-inflammatory hyperpigmentation. Pruritus is usually absent or may be mild during the healing and desquamation phase.

In summary, the symptoms of the disease include a burning or stinging sensation similar to sunburn, pain, and subsequently pruritus.[10]

Acute skin lesions include erythema, edema, pseudopapules, vesicles, and bullae, followed by hyperpigmentation, which may be long-lasting.[10–17]

The distribution of lesions is variable and often adopts unusual shapes. They may be linear, as described in the cases reported by Dreher and Evans involving the cheek and hands due to lemon exposure[42] or may vary according to the mechanism of exposure. Splash-like patterns caused by citrus droplets can be observed, as well as linear patterns following exposure to plants such as wild parsnip, or drip-shaped lesions resulting from the downward flow of the causative liquid over the skin.

The timing of lesion onset is also variable. Although the classic onset is 24–48 hours after exposure, it may occur earlier in cases involving fig tree or giant hemlock exposure. The latter can be particularly dangerous in children who use its hollow stems as makeshift telescopes, leading to severe periocular lesions.[43]

Below, we present some of the cases observed at our center, in which the clinical characteristics are clearly fulfilled, both in terms of lesion types (erythema, inflammation, pigmentation, bullae), anatomical distribution (face and perioral region, hands, trunk, and extremities), and lesion morphology (plaques, linear, splashed, dripping patterns, etc.) (Figs. 3–12).

Figure 3. Patients with hand lesions showing erythema, inflammation, and residual pigmentation.

Figure 4. Same type of lesion, but with the presence of blisters.

Figure 5. Splash-pattern pigmentation, likely due to lemon droplets.

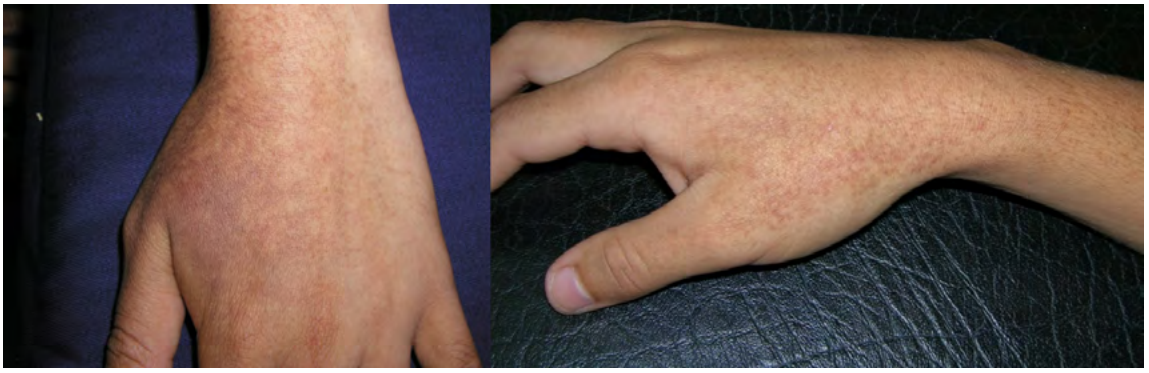

Figure 6. Facial and perioral lesions.

Figure 7: A and B: Linear facial and palpebral lesion, with dermoscopy showing pigmentation and scaling.
C: Linear facial lesion and pigmentary involvement of the fingers.

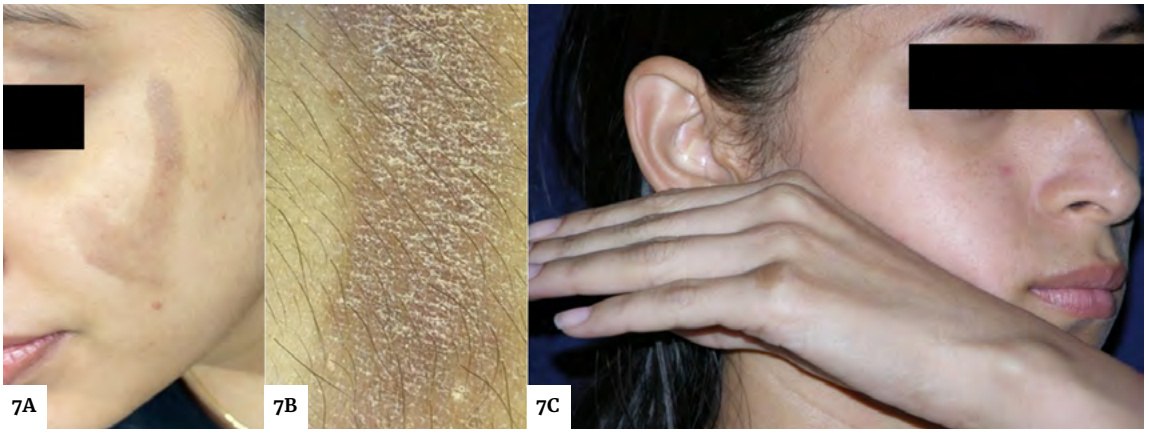

Figure 8: A: Digitiform pigmented lesions on the back.
B and C: Hand lesions and punctate pigmentation on the chest and abdomen.

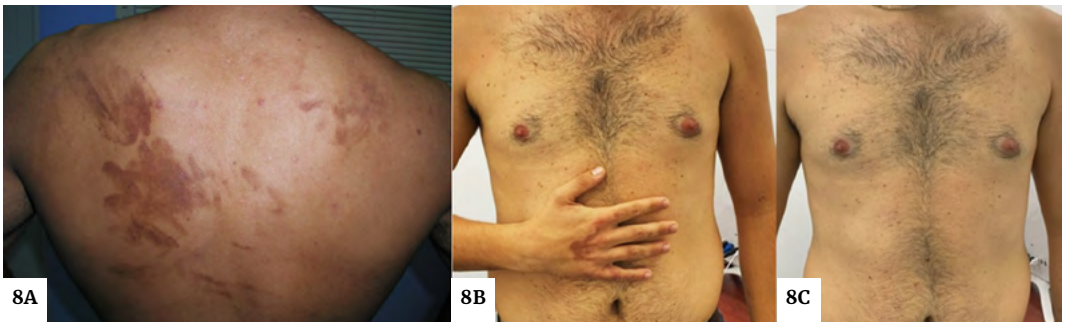

Figure 9: Inflammatory drip-like lesions.

Figure 10: Pigmented drip-like lesions.

Figure 11: Varied lesions in different locations.

Figure 12: Phytophotodermatosis lesions in children.

DIFFERENTIAL DIAGNOSIS

In some cases, phytophotodermatitis is confused with other conditions such as allergic contact dermatitis, cellulitis, or fungal infections.[44] Several publications suggest that physical abuse or child maltreatment should be considered in the differential diagnosis, probably due to the bruise-like lesions that may sometimes be observed in this condition.[45]

Other diagnoses that may or should be considered include photosensitivity disorders such as solar urticaria or porphyria; infections such as tinea or herpes simplex; drug-induced allergic reactions such as erythema multiforme;[41] jellyfish stings; burns; impetigo; bullous dermatoses; among others.[17]

TREATMENT

In the acute phase, treatment includes wet dressings with Burrow's solution or potassium permanganate, topical or systemic corticosteroids when warranted by the severity of the condition, as well as anti-inflammatory agents and analgesics. Prevention involves educating the patient about the underlying causes, avoiding sun exposure for at least two days following contact with the causative agent, and washing the hands immediately after contact or exposure.[46]

CONCLUSIONS

Phytophotodermatosis constitutes a relatively frequent condition in our setting. Although in some cases its diagnosis is straightforward, it is often immediately attributed to contact with lime. However, the existence of multiple etiological agents is now recognized, as well as the importance of the time of onset after contact, and the type and distribution of cutaneous lesions, in order to establish an accurate diagnosis and appropriate management.

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