CLINICAL IMAGES

Phytophotodermatitis

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A five year old boy, accompanied by his concerned mother, presented to the local emergency department fast track with a well-demarcated, erythematous skin rash in a drip-mark configuration on his shoulders, upper arms, trunk, and back (Fig. 1 and Fig. 2). The mother noticed the rash the same day as arrival to the emergency department. The child had just spent the weekend with his father and the mother was aware that the child had been playing outdoors. The mother reported no known environmental exposures. The mother denied patient history of fever, pruritus, lesions on palms or soles, mucosal lesions, recent bug bites, or others in the household with a similar rash.

The father was then contacted to provide further history. He revealed that two days prior to appearance of the rash he attempted to rid his son of head lice by pouring a homemade solution of lemon juice and hydrogen peroxide over his son's head. The patient then proceeded to play outside without a t-shirt or sun protection and had not rinsed the solution from his scalp or body prior to the sun exposure.

FIGURE 1:

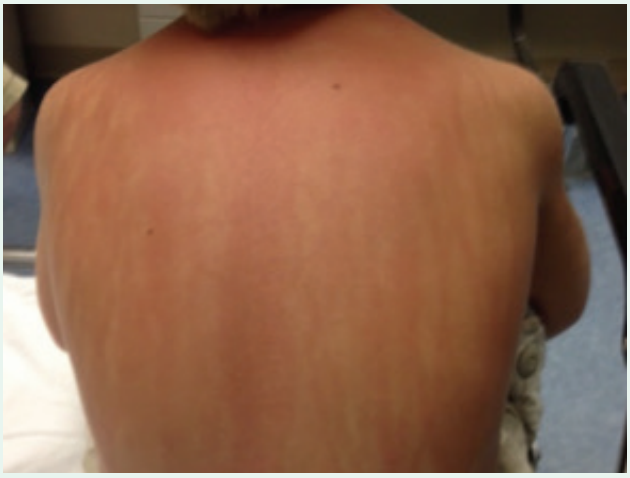
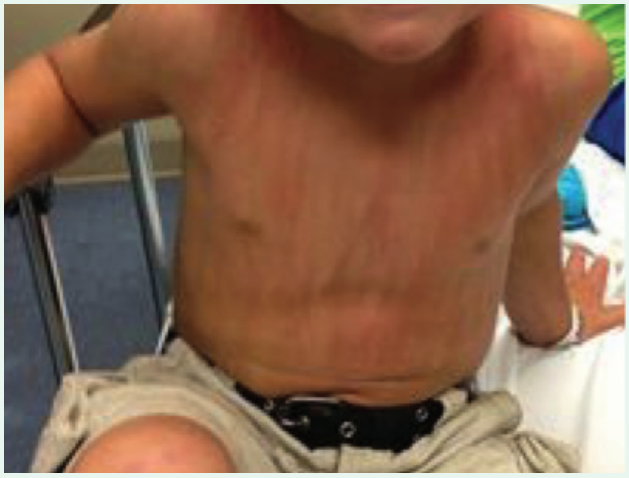


FIGURE 2:



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QUESTIONS

1. What is the diagnosis?

    a. Folliculitis
    b. Dermatitis Herpetiformis
    c. Phytophotodermatitis
    d. Atopic Dermatitis
    e. Rhus Dermatitis

2. What is the mechanism of the rash?

    a. Inflamed and infected hair follicles
    b. Autoimmune
    c. Contact with photo-reactive compounds
    d. Dietary exposure
    e. Poison ivy exposure

3. What is the recommended treatment for this patient?

    a. Conservative, symptomatic care, wet dressings
    b. Oral Prednisone
    c. Gluten-free diet
    d. Bleaching agents
    e. Oral antibiotics

See page 56 for answers and discussion.

ANSWERS

1. What is the diagnosis?

Answer C: Phytophotodermatitis

Explanation: The patient had skin exposure to a citrus solution when his parent was attempting to rid him of lice. He subsequently had ultraviolet light exposure while playing outdoors without a t-shirt on or sun protection. The other answers are incorrect because: Folliculitis is a bacterial infection localized to hair follicles which presents with pustular papules.[11] Dermatitis herpetiformis is a skin manifestation of Celiac Disease.[12] It is a pruritic skin eruption with papules or vesicles most often localized to the elbows, knees, back and buttocks.[12] Atopic dermatitis is a pruritic, erythematous skin rash localized most commonly to the antecubital and popliteal fossa.[13] It often presents with scaling patches and may result in post-inflammatory pigmentation changes.[14] Rhus dermatitis should be distinguished from a blistering presentation of phytophotodermatitis. It is not limited to sun-exposed areas of the body and may continue to worsen over the course of one or two weeks. However, phytophotodermatitis will peak in intensity in 48-72 hours and classically leaves behind a well-demarcated bizarre pattern of post-inflammatory hyperpigmentation.[4, 7, 15]

2. What is the mechanism of the rash?

Answer C: Contact with photo-reactive compounds

Explanation: Phytophotodermatitis is caused by contact with photoreactive compounds in certain plants that cause inflammation of the skin when exposed to UVA radiation.[1]

3. What is the recommended treatment for this patient?

Answer A: Conservative, symptomatic care, wet dressings

Explanation: Management depends on the area of skin involvement as well as the intensity. In mild cases, the treatment is conservative, symptomatic care with moist dressings.[7] If the dermatitis is severe, bullous and involving more than 30% of total body surface area, the patient may need burn center evaluation.[7] Topical or oral corticosteroids may help with patient discomfort by decreasing the inflammatory response.[7] Sunscreen with physical blockers of UVA radiation such as zinc oxide and titanium dioxide are recommended to prevent subsequent epithelial damage.[10] Bleaching agents such as hydroquinone are not indicated in this case as the patient has no cosmetic concerns regarding post-inflammatory hyperpigmentation. Antibiotics may be indicated if the clinical presentation involved vesicles/bullae that became secondarily impetiginized.

DISCUSSION

Phytophotodermatitis is a non-immunologic inflammatory skin reaction which develops following cutaneous contact with photoactive compounds in certain plants called furocoumarins in conjunction with sunlight exposure.[1] The furocoumarins, most commonly psoralens and angelicins, found in various plants and plant extracts form phototoxic combinations when exposed to ultraviolet-A (UVA) radiation.[2, 3] The phototoxic cutaneous reaction occurs commonly after contact with citrus fruit juices, celery, and rue.1

Phytophotodermatitis dates as far back as 2000 BC when the practice of rubbing juices from Ammi majus on the skin was encouraged for patients suffering from vitiligo, an autoimmune disease of skin depigmentation.[1] In 1400 BC, Psoralea corylifolia was boiled and used on these patients to induce the skin dermatitis and subsequent hyperpigmentation, from which the term "psoralen" was named.[1] However, it was not until 1942 that the combination of plants and sunlight as a cause of the phototoxic reaction was recognized and termed "phytophotodermatitis" by Robert Klaber, MD.[1] The plants that are largely responsible for the sun-induced skin rash include: the Umbelliferae family, which includes celery, parsley, and parsnips; the Rutacea family, which includes lemons and limes; and the Moraceae family which includes figs. In current times, the skin condition is common in children during the summer months when psoralens are in greatest numbers.[4] It is also common in beachgoers, florists, agricultural workers, and gardeners all of which have both exposure to plants and sunlight.[5]

In the acute phase of phytophotodermatitis, an oxygen-dependent cutaneous injury occurs.[1] The cutaneous insult involves psoralens on the skin surface that react with UVA radiation and initiate keratinocyte apoptosis. This leads to edema and desquamation of the epidermis.[1, 3, 6, 7] An oxygen-independent reaction occurs as UVA radiation induces covalent binding between these plant light-activating chemicals and the DNA of skin cells.[2, 7] Approximately 12 to 36 hours after exposure to the light activating chemicals in the plants, well-demarcated erythema with or without blisters may appear on the skin. A burning sensation in the affected area may also be present. Patients may also note edema, pain or pruritis.[8] The skin manifestations are highly variable and have been reported in some patients to mimic a partial thickness burn.[2] The reaction peaks in intensity at 48 to 72 hours after initial exposure.[4, 7] A post-inflammatory hyperpigmentation remains as the healing process begins and can persist for months. The pattern of dermatitis gives important clues to the diagnosis, as it often appears as a "drip mark" or "streak mark" configuration, or as an unusual sunburn pattern such as a handprint.[4, 6] The most common location for phytophotodermatitis is the dorsum of the hand,

as it is a body surface with both psoralen contact and sun exposure. The cutaneous upper lip is another commonly affected area of the phototoxic rash that may manifest due to biting into a psoralens-containing fruit or drinking fruit juice.[9]

The diagnosis of phytophotodermatitis is clinical and relies heavily on a thorough history and a high index of suspicion from the provider. It can be difficult to diagnose because it is often mistaken for atopic dermatitis, chemical burns, melasma, or even child abuse.[7,9] Management of the dermatitis depends on the area of skin involvement as well as the intensity. In mild cases, the treatment is conservative, symptomatic care with moist dressings.[7] If the dermatitis is severe, involving more than 30% of total body surface area, patient transfer to a burn care center may be considered.[7] Topical corticosteroids may be used to help diminish patient discomfort and minimize post-inflammatory hyperpigmentation. Oral corticosteroids can be implemented to control the cutaneous inflammation for patients with very severe cases.[7] Making the diagnosis is important both for patient education and prevention measures, as prevention is key for this disease process.[5] An early diagnosis allows for early initiation of topical corticosteroid therapy to diminish the extent of the hyperpigmentation which may be a cosmetic concern for the affected individuals.[5] Sunscreens containing physical blockers of UVA radiation such as zinc oxide and titanium dioxide are recommended to prevent further epithelial damage and inflammation. Bleaching agents such as hydroquinone are only indicated for patients with cosmetic concerns about hyperpigmentation, as pigment changes will likely resolve over the several-month healing process.[10]

In summary, phytophotodermatitis is a phototoxic skin reaction to cutaneous contact with psoralens in plants and plant extracts such as lime or lemon juice, and subsequent exposure to UVA radiation. The patient may present with an unusual distribution pattern, as this patient did with sunburn appearing drip marks distributed on his trunk. It is a clinical diagnosis requiring a high index of suspicion by the provider. Depending on the extent of the cutaneous damage, management of phytophotodermatitis may involve conservative care with cooling agents and topical corticosteroids or, in extreme cases may require admission to a burn center. This patient was treated conservatively because of his mild, asymptomatic clinical presentation. Daily sunscreen application should be encouraged to minimize additional epithelial damage from UV light exposure in the following weeks. Patient education is important with this skin condition as prevention measures may diminish the chance of a subsequent episode. Referral to a dermatologist or burn center is typically only indicated in severe cases.

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