genesis of DSP is a complex combination of genetic and environmental triggers. It is inherited as an autosomal trait and may be triggered by stimuli including sunlight and artificial UV light, viral infections, immunosuppressive conditions (hematologic malignancies, organ transplant, or autoimmune disease), and immunosuppressant therapies. The role of immunosuppressant therapy is supported by the observation in at least 1 case report of remission of DSP on withdrawal of immunosuppressant therapy. To our knowledge, there are no reports of DSP arising in patients receiving etanercept, other tumor necrosis factor (TNF) antagonists, or biological agents of other classes.

The mechanism by which immunosuppressant therapy induces DSP is not clear. It may cause the phenotypic expression of the disease in a genetically predisposed patient to manifest by either directly triggering the expression of abnormal clones of epidermal cells or by disrupting the growth of the epidermis and promoting clonal cell proliferation. Alternatively, immunosuppressant therapy might reduce immune surveillance and allow the proliferation of the abnormal clones. This theory is supported by the observation that T cells are important in the prevention of porokeratotic clonal growth. Etanercept is a recombinant TNF receptor fusion protein that binds to and inhibits TNF. Tumor necrosis factor is important in inflammatory, apoptotic, and cellular proliferation pathways. Etanercept, by inhibiting TNF, results in a decrease in the number of T cells, which are important in immune surveillance and in preventing the abnormal keratinocyte clonal proliferation that occurs in DSP.

This case is of interest because DSP was noted after etanercept treatment was introduced, and we suggest that the immunosuppressant effects of etanercept contributed to DSP development. As more patients are treated with biological immunosuppressant therapy, DSP may become more frequently reported as a common adverse effect of these therapies.

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Mexican Beer Dermatitis: A Unique Variant of Lime Phytophotodermatitis Attributable to Contemporary Beer-Drinking Practices

The phenomenon of phytophotodermatitis (PPD) induced by lime juice has been recognized for centuries. Limes contain varying concentrations of phototoxic coumarin compounds, including bergapten, 5-methoxypsoralen, xanthotoxin, and limettin. These chemicals absorb light in the UV-A range. Contact with lime juice or rind results in these phototoxic compounds being transferred to the skin, where their interaction with UV light results in phototoxic reactions of varying severity. Owing to the external causes of lime PPD, affected patients may exhibit bizarre patterns of phototoxic effects reflecting the pattern of cutaneous exposure to lime-derived phototoxins. Frequently, affected patients will have linear lesions that may mimic signs of allergic contact dermatitis, child abuse, cellulitis, impetigo, erythema migrans, and even jellyfish evenomation.

Report of Cases. Recently, I have seen a number of cases of lime PPD in which all of these patients were exposed to lime after drinking a popular Mexican beer. Mexican beers, including Corona (Grupo Modelo SA De CV, Mexico City, Mexico) and Dos Equis (Cerveceria Cuauhtemoc Montezuma, Monterrey, Mexico), are currently among the most popular imported beers in the United States. The beverage is customarily served with a lime slice or wedge in the mouth of the bottle. The drinker presses the lime into the bottle, then holds his or her thumb over the mouth of the bottle while inverting the bottle. This maneuver causes the lime to float upwards to the bottom of the inverted bottle, where it remains as the person drinks the beer. However, when the beer bottle is inverted, the carbonation in the beer frequently causes a mixture of beer and lime juice to spray from the bottle despite the drinker’s attempt to seal the bottle mouth with the thumb. This common drinking practice may result in lime juice being sprayed over a wide area of skin, especially in a patient who is shirtless by a beach or pool.

Case 1. A 32-year-old woman was seen for a linear eruption consisting of erythema and hyperpigmentation over the chest, abdomen, and legs (Figure 1). The eruption...
The present cases illustrate that exposure to lime and its associated preparations containing lime juice may cause substantial concern for these individuals. Furthermore, affected patients will typically deny direct exposure to lime juice or trees, and the history of lime exposure will not be elicited unless they are asked specifically whether they have been drinking Mexican beer. Therefore, prompt recognition of this unique phototoxic reaction and an understanding of its cause will help physicians counsel patients to avoid repeated episodes.

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Toxic Effects of Fluorouracil Cream Ingestion on Dogs and Cats

Report of a Case. A 63-year-old man with a history of squamous cell carcinoma and actinic keratoses was seen for an initial visit for dermatologic evaluation. Physical examination revealed multiple actinic keratoses diffusely distributed across his face. Field treatment with fluorouracil, 5%, cream was recommended. The patient declined because when he had previously been treated with 5-fluorouracil, 5%, cream, his 2-kg Yorkshire Terrier had bitten the tube and shortly thereafter began vomiting and seizing. The Animal Poison Control Center (APCC) told him that fluorouracil cream is extremely toxic to dogs, and they recommended that he immediately take his animal to a local veterinary emergency care facility. At the facility, his dog became comatose and died several hours later. He feared he might accidentally expose another of his dogs to the medication.

Comment. We were unaware of the danger of fluorouracil ingestion in dogs and contacted the main APCC in the United States, located in Urbana, Illinois (www.aspca.org; telephone number, 217-337-9721). The veterinarian at the APCC informed us that fluorouracil, 5%, cream was extremely toxic to both dogs and cats (Eric Duyer, MS, VMD, DABT, DABVT, telephone communication, April 30, 2010). The 2010 edition of the Physicians’ Desk Reference does not list this effect. The 50% lethal oral dose in rats is reported as 100 mg/kg; in mice, 500 mg/kg. However, the minimal lethal oral dose in dogs is only 20 mg/kg. In a 2-kg dog, therefore, a potential lethal dose might be only about 0.8 g of cream, which could easily be ingested via a puncture bite through the tube. According to Albretsen, chewing of the small, 25-g tube is the usual method of ingestion by dogs.

Figure 2. Hyperpigmentation in a linear splash pattern (A) and a finger swipe pattern (B) on the neck of patient 2, who was exposed to lime while drinking Mexican beer.