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COVER QUIZLET

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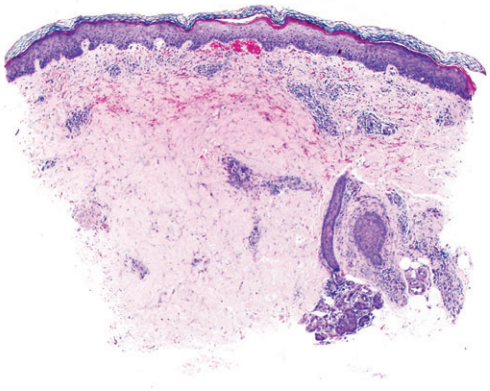
Figures 1 and 2 are depicted on the journal cover.



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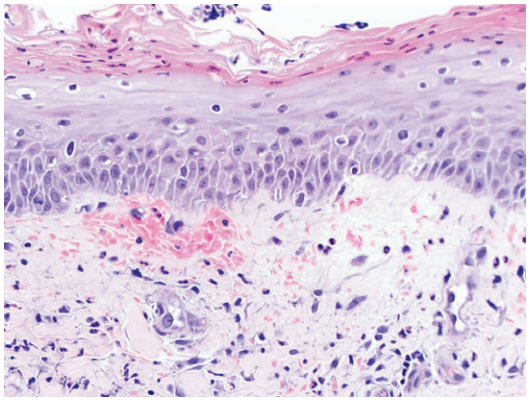

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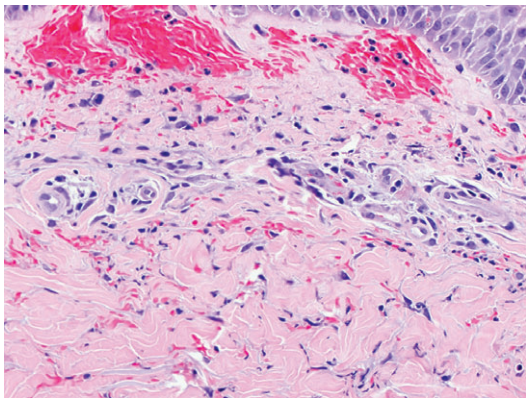

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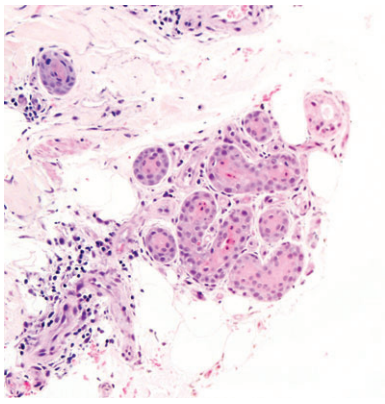

FIGURE 6.

FIGURE 7.

Your diagnosis?

Discussion follows on page 996

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Purpuric Agave Dermatitis

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1 | PURPURIC AGAVE DERMATITIS

A 53 year old man presented with a 4 day history of a rapidly progressive, painful purpuric eruption on the lower extremities. The lesions started as red to purple papules, some in a linear array, on the anterior and medial lower extremities several hours after working outside his house clearing foliage. The posterior legs were completely spared. Over the ensuing days, the lesions continued to progress, coalescing into large confluent purpuric plaques with surrounding purpuric papules and admixed multi-loculated hemorrhagic bullae (Figure 1). There were no lesions elsewhere on his body. He denied fever, chills, malaise, arthralgia, abdominal pain, hematuria, or other systemic symptoms. He had no significant medical history and took no medications.

He presented to his primary care physician who prescribed a short course of oral prednisone and referred him to dermatology. There were no laboratory abnormalities, and the patient's vital signs were within normal limits. When he saw dermatology the following day, a punch biopsy was performed and topical clobetasol ointment was added to his regimen. Histopathologic examination showed a perivascular and peri-adnexal neutrophilic inflammatory infiltrate with mild leukocytoclasia, scattered eosinophils, and numerous extravasated erythrocytes (Figures 2, 3). There was spongiosis and

FIGURE 1 Clinical appearance of purpuric agave dermatitis

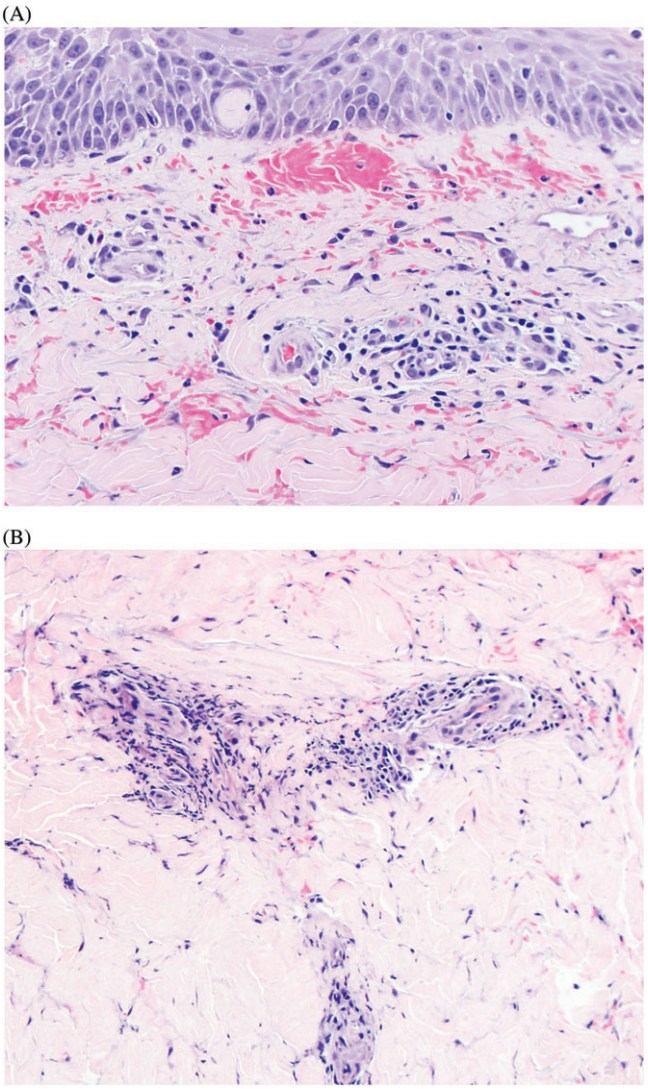

FIGURE 2 Histopathologic findings in purpuric agave dermatitis including a perivascular and interstitial infiltrate of neutrophils and lymphocytes with dermal hemorrhage. Leukocytoclasia is present around blood vessels. H&E 100x

FIGURE 3 Low power view of histopathology showing prominent purpura with a perivascular and periadnexal inflammatory infiltrate. H&E 20x

FIGURE 4 Epidermal changes in purpuric agave dermatitis with diffuse spongiosis and parakeratosis. Scattered single dyskeratotic keratinocytes are present in the basal and spinous layers. H&E 100x

FIGURE 5 Trace fibrin deposition in vessels was present, but was not a prominent feature in this case. H&E 200x.

FIGURE 6 Syringosquamous metaplasia. H&E 200x.

FIGURE 7 *Agave americana* plant.

parakeratosis of the epidermis, with scattered necrotic keratinocytes (Figure 4). Some subtle vascular fibrin deposition in dermal blood vessel walls was noted (Figure 5), suggesting a diagnosis of evolving leukocytoclastic vasculitis. Prominent syringosquamous metaplasia was also present (Figures 3 and 6). Polarized microscopy was negative for polarizable foreign material. Gram, PAS-D, and Fite stains were all negative for an infectious etiology. Follow up history provided by the patient included the fact that just prior to symptom onset, he had been trimming overgrown agave plants in his yard with a chainsaw during which fragments of the sticky plant material splattered forcefully onto his exposed legs. With that additional clinical history, the correct diagnosis of purpuric agave dermatitis was rendered. The patient completed a short course of oral prednisone in combination with topical clobetasol ointment twice per day to the affected area. With this regimen, his eruption completely cleared over the ensuing month.

2 | DISCUSSION

Agave americana (Figure 7) is a species of succulent flowering plant in the Asparagaceae family that is native to hot and arid regions in Mexico and the Southwestern United States. Other agave species can be found in tropical areas of South America. These plants have various industrial usages, including being used to make rope, coarse cloth, alcoholic beverages, and sweeteners. It is also used as a decorative plant. The sap of the agave plant contains several potential noxious substances including calcium oxalate crystals, acrid oils, agave gum, and saponins. These substances have been previously reported to cause irritant and allergic contact dermatitis.[1–3] Rarely, a purpuric variant has been reported. Purpuric agave dermatitis was first described by Ricks et al. in 1999, and was originally characterized histopathologically as a leukocytoclastic vasculitis, with perivascular neutrophils, fibrinoid degeneration, and karyorrhexis.[4] We found only one additional case of purpuric agave dermatitis reported in the English literature,[5] as well as two in Spanish, and one in French.[6–8] All reported purpuric cases have been associated with the use of a chainsaw, resulting in agave particles being propelled toward the skin with high velocity. This high velocity likely allows for dermal penetration and appears to distinguish the purpuric variant from simple irritant contact dermatitis from agave. Purpuric papules and plaques appear in a pattern correlating to the skin exposed to the plant's sap during trimming. One of the reported cases described systemic symptoms of fever, malaise, and myalgias associated with the purpuric eruption.[5]

The mechanism of purpuric agave dermatitis is not fully understood. One proposed mechanism is embedding of oxalic acid crystals into the dermis and surrounding dermal blood vessels, resulting in oxalic acid toxicity and subsequent vascular damage.[5] Others have proposed direct vascular damage due to trauma when the plant particles are forcefully embedded into the skin, or vascular toxicity triggered by saponins deposited near superficial blood vessels.[6] The commonly associated burning sensation experienced when the sap comes into contact with skin has been attributed to the sharply pointed crystals of calcium oxalate.[3]

The diagnosis of purpuric agave dermatitis can be made on the basis of history and clinical morphology, though biopsy can be helpful in confirming the diagnosis. Histopathologically, lesions demonstrate a perivascular infiltrate of neutrophils, neutrophilic debris, and extravasated red blood cells, with or without fibrin deposition in vessel walls, suggestive of an early or low-grade leukocytoclastic vasculitis. There is evidence of irritant contact dermatitis in the epidermis with spongiois, parakeratosis and variable keratinocyte necrosis. Without clinical information, a diagnosis of a systemic leukocytoclastic vasculitis may be rendered and lead to an unnecessary workup in search of a systemic inflammatory condition. In our case, fibrin deposition in vessel walls was only minimally present and there was prominent syringosquamous metaplasia, which may be a clue in distinguishing the vasculitic changes in agave purpuric dermatitis from a systemic leukocytoclastic vasculitis.

Given the rarity of this entity, the optimal treatment regimen is unknown. Oral corticosteroids can be considered depending on the severity of the purpuric eruption and the presence of systemic symptoms. Our patient responded quickly to a short course of oral prednisone and topical clobetasol.

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