Leukocytoclastic vasculitis sparing a tattoo with halo effect

The Harvard community has made this article openly available. Please share how this access benefits you. Your story matters

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Published Version</td>
<td>doi:10.1016/j.jdcr.2015.06.002</td>
</tr>
<tr>
<td>Citable link</td>
<td><a href="http://nrs.harvard.edu/urn-3:HUL.InstRepos:26859999">http://nrs.harvard.edu/urn-3:HUL.InstRepos:26859999</a></td>
</tr>
<tr>
<td>Terms of Use</td>
<td>This article was downloaded from Harvard University’s DASH repository, and is made available under the terms and conditions applicable to Other Posted Material, as set forth at <a href="http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#LAA">http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#LAA</a></td>
</tr>
</tbody>
</table>
Leukocytoclastic vasculitis sparing a tattoo with halo effect

Chen (Amy) Chen, BA,a Anar Mikailov, MD,b Beverly Faulkner-Jones, MD, PhD,c and Peggy A. Wu, MD, MPHb
Boston, Massachusetts

Key words: leukocytoclastic vasculitis; palpable purpura; reverse Koebner phenomenon; sparing phenomenon; Wolf’s isotopic nonresponse.

INTRODUCTION
The phenomenon of cutaneous diseases sparing sites of previous trauma has been described, although there are few case reports that illustrate this rare phenomenon. We report an unusual case of leukocytoclastic vasculitis (LCV) that completely spared tattooed skin with an additional halo effect around the tattoo.

CASE REPORT
A man in his 40s with recent intravenous drug use and hepatitis C was admitted to the medical intensive care unit with progressive respiratory failure in the setting of multifocal pneumonia, an epidural abscess, tricuspid valve endocarditis, oxacillin-sensitive Staphylococcus aureus bacteremia, and acute hepatorenal failure. Laboratory values on admission were notable for a white blood cell count of 20 K/μL, a hemoglobin level of 11.3 g/dL, international normalized ratio of 1.7, platelets of 30 K/μL, creatinine level of 6 mg/dL, and total bilirubin of 17.2 mg/dL. During his hospital stay, he received multiple antibiotics (levofloxacin, ceftriaxone, piperacillin-tazobactam, and nafcillin) and additional new medications (octreotide, pantoprazole, albumin, furosemide, lactulose). The patient had no known history of drug allergies. On day 5 postadmission, nonretiform, palpable purpura (Fig 1) developed consisting of well-defined, non-blanching, coalescing, 2- to 3-mm bright red to purple macules and papules primarily involving the legs. No splinter hemorrhages, stellate lesions, ulcerations, or blisters were noted. No additional symptoms were elicited, as the patient was intubated. Interestingly, the eruption exhibited a “sparing phenomenon,” with a 2- to 3-cm halo of clinically normal skin around his tattoos (Fig 2, A and B). Laboratory data showed a hepatitis C viral load of 323,000 IU/mL. Cryoglobulins, antinuclear antibody, anti-neutrophil cytoplasmic antibodies, anti-mitochondrial antibody, and anti–smooth muscle antibody test results were all normal. A skin biopsy found a superficial perivascular neutrophilic infiltrate with sparse leukocytoclasia, red blood cell extravasation, and reactive vascular changes with complement C3 deposition, consistent with LCV (Fig 3). The etiology was assumed to be secondary to nafcillin, given the known association between penicillins and LCV1 and the appearance of the rash 5 days after nafcillin was initiated. As a result, his nafcillin was discontinued and vancomycin started instead. By day 14 postadmission, the vasculitis was almost fully resolved without any further interventions.

DISCUSSION
LCV is an acute neutrophilic small vessel vasculitis resulting from vascular deposition of circulating immune complexes and subsequent activation of the complement cascade. This activation results in neutrophil chemotaxis and microvascular injury.2 Clinically, LCV is characterized by palpable purpura, although less common findings can include urticarial plaques, vesicles, bullae, and pustules. LCV can be secondary to a variety of agents including medications, chemicals, infections, or underlying systemic disease; however, up to 60% of cases are idiopathic.2
Here we report, to our knowledge, an unusual case of LCV with sparing of tattooed skin to produce a “halo.” The phenomenon of one skin disease sparing the site of a previous skin condition has been recognized since 1981, when Cochran and Wilkin reported a case of a drug rash failing to appear in a previously irradiated site on a 12-year-old girl. Since then, various related terms have been used to describe this phenomenon, including the reverse Koebner phenomenon (nonappearance or disappearance of skin lesions at sites of trauma) and Wolf’s isotopic nonresponse (absence of a cutaneous eruption at the site of another unrelated and already healed skin disease). Although the exact mechanisms and pathophysiology behind these sparing phenomena are unknown, current hypotheses suggest that alterations in the local structure or immunologic microenvironment at these sites may play a role.

To date, few articles report on sparing phenomenon in association with LCV. In 2011, Yadav et al reported a case of reverse Koebner phenomenon in a bandage-covered area in a patient with cutaneous small vessel immune complex vasculitis. The authors suggested that the sparing phenomenon may have been a result of diminished blood flow to the dermis secondary to mechanical pressure from the bandage and consequent failure of immune complex deposition. In 2014, Pinal-Fernandez and Solans-Laque reported a case of relative sparing of a purpuric rash over tattooed skin, although the purpuric rash still extended several centimeters into the area demarcated by the tattoo. The authors proposed several etiologic explanations for the sparing phenomenon over tattooed skin. These included the use of *Hamamelis virginiana*, a component of tattoo ink with known anti–tumor necrosis factor and anti–reactive oxygen species properties that may have had an inhibitory effect on the development of vasculitis, the potential of the tattoo dye to act as a photoprotective factor against light penetration into the dermis, and microvascular changes caused by tattooing.

**Fig 1.** Extensive nonretiform palpable purpura on bilateral lower extremities.

**Fig 2.** Palpable purpura exhibiting a “sparing phenomenon” with a 2- to 3-cm halo around a tattoo on the (A) right anterior thigh and (B) right anterior leg.
In this case, purpura not only spared the multiple tattoos but exhibited a unique halo effect of 2 to 3 cm of clinically normal skin surrounding the tattoos. The term halo is extrapolated from its use in the halo nevus where local immunologic changes account for perinevoid vitiligo. In the case of LCV, the halo effect could reflect local immunologic changes of the dermis caused by tattooing that inhibit neutrophil migration and subsequent cytokine cascade. The patient’s tattoos were self-administered 20 years previously using ink made from burned plastic ashes, Colgate toothpaste, and Alberto VO5 shampoo. Fluoride in animal and human studies has been found to affect immune function through observed effects on T regulatory cells and neutrophils. Given the important role of neutrophil migration in effecting an immune response, we hypothesize that the introduction of fluoride into the dermis through toothpaste may account for some degree of the observed halo effect. Distortion or destruction of postcapillary venules as a consequence of tattooing may also have a contributory effect. Further studies are required to elucidate the pathogenesis behind this novel phenomenon of LCV sparing a tattoo with halo effect.

REFERENCES