# IgA vasculitis triggered by alcohol consumption in a 51-year-old man: A case report

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## INTRODUCTION

IgA vasculitis, formerly known as Henoch-Schönlein purpura, is a leukocytoclastic vasculitis characterized by the deposition of IgA-dominant immune complexes in small blood vessel walls.[1] IgA vasculitis is most often idiopathic but has been associated with infectious agents, malignancies, and drug reactions.[1] While rare, there have been documented cases reporting alcohol consumption as a trigger for IgA vasculitis in adults.[2-5]

Here, we describe a case of recurrent IgA vasculitis triggered by alcohol and summarize the current literature about the theorized etiology and pathophysiology.

## CASE REPORT

An otherwise healthy 51-year-old man presented to the dermatologist for a petechial rash on both lower extremities and groin area. The patient noted that the night before the exanthem developed, he had consumed beverages containing distilled alcohol. The associated symptoms included both ankle swelling and pain. The patient was taking tramadol, as needed, for back pain. The review of systems was negative for other signs of systemic disease.

On cutaneous examination, multiple nonblanchable, dark-red macules, consistent with petechiae, were scattered on both lower extremities, suprapubic area, penis, and scrotum. Areas of necrotic ulceration were present on the left distal extremity (Fig 1, *A*). There was also marked edema of both ankles.

Punch biopsies were taken from the affected areas. An histologic evaluation demonstrated a neutrophilic dermatitis with the obliteration of small dermal vessels, erythrocyte extravasation, and deposition of fibrin and nuclear dust (Fig 2). Direct immunofluorescence studies demonstrated granular, perivascular IgA deposition.

A presumptive diagnosis of IgA vasculitis was made. Laboratory studies revealed normal urinalysis, coagulation studies, and inflammatory markers. Antinuclear antibodies, antineutrophil cytoplasmic antibodies, and antiphospholipid antibodies were not detected. The dilute Russell viper venom time was prolonged (>45 seconds). A course of oral prednisone led to the improvement of the petechiae and both ankle edema within 2 weeks.

Three months later, the patient returned due to a recurrence of the eruption with new-onset pruritus (Fig 1, *B*). His history was significant for a recent tattoo on the right lower extremity (1 week prior) and drinking tequila with friends the night before the flare. A biopsy again demonstrated IgA vasculitis. Oral colchicine was started after this recurrence, and the lesions improved with topical glucocorticoids.

One month later, the patient's exanthem flared again, this time occurring the day after a celebration at which he drank cognac. The patient noted that he could drink beer without a problem, but every time he drank distilled alcoholic beverages, he had a recurrence. A biopsy again demonstrated IgA

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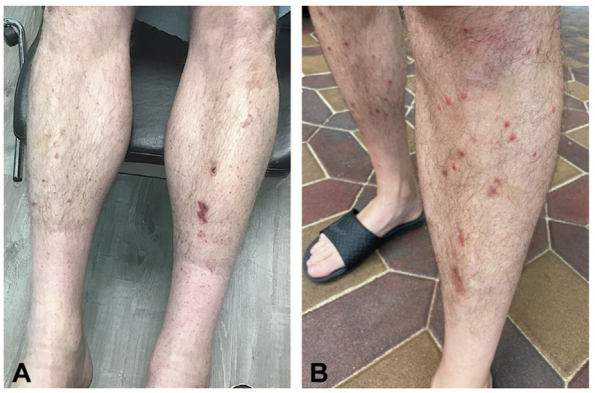


**Fig 1. A,** The patient first presented with dark-red macules scattered on both lower extremities, with areas of necrotic ulceration on the left distal extremity. Edema of both ankles can be visualized. **B,** The patient presented 3 months later due to a recurrence of the petechial exanthem on both lower extremities with new-onset pruritus.

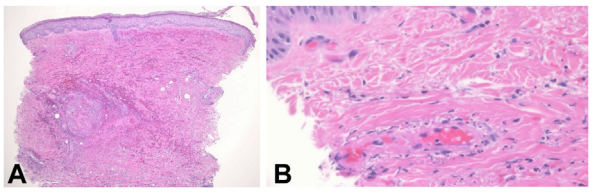


**Fig 2. A,** Scanning magnification shows diffuse perivascular inflammation and dermal hemorrhage (hematoxylin-eosin stain; original magnifications: ×40). **B,** A small, superficial vessel with leukocytoclasia and intramural fibrin deposition (hematoxylin-eosin stain; original magnifications: ×400).

vasculitis. A workup for paraneoplastic causes was performed, including laboratory work, a colonoscopy, and an endoscopy, which were negative. At this time, the patient was warned that the consumption of distilled alcoholic beverages was the likely cause of his vasculitis and that he should refrain from this in the future to prevent recurrence.

## DISCUSSION

Only 4 biopsy-proven cases of alcohol as a trigger for IgA vasculitis in adults have been reported.[2-5] In one of these cases, the patient only developed the exanthem secondary to the consumption of alcohol with hops.[5] Another case reported that the vasculitis flared with the consumption of wine and vinegar.[4] One report found beer to be the trigger.[2] In another case, the vasculitis developed when the patient drank beer, wine, or distilled alcoholic beverages.[3] In this case report, our patient only developed the vasculitis after drinking distilled alcoholic beverages. This refers to forms of liquor and contains higher alcohol content than beer and wine which do not trigger the patient.

The etiology and pathophysiology of alcohol-induced vasculitis are unclear.[5] Chua et al[2] reported a case that suggested the role of regulatory T lymphocytes in the development of high circulating IgA levels followed by IgA and C3 deposition in the skin. Abuabara et al[3] documented a case that suggested immune-complex deposition mediated by IgA as a cause of alcohol-triggered vasculitis.

It is possible that in a genetically susceptible person, alcohol or its metabolites may cause a dysregulated inflammatory response, leading to IgA-mediated immune-complex deposition in small blood vessel walls and cutaneous vasculitis. The identification of alcohol-induced vasculitis is important for the appropriate management and the avoidance of the trigger in susceptible persons, and a comprehensive workup should be done when it is suspected.

### Conflicts of interest

None disclosed.

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