

# Hydrochlorothiazide Induced Vasculitis

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## Introduction

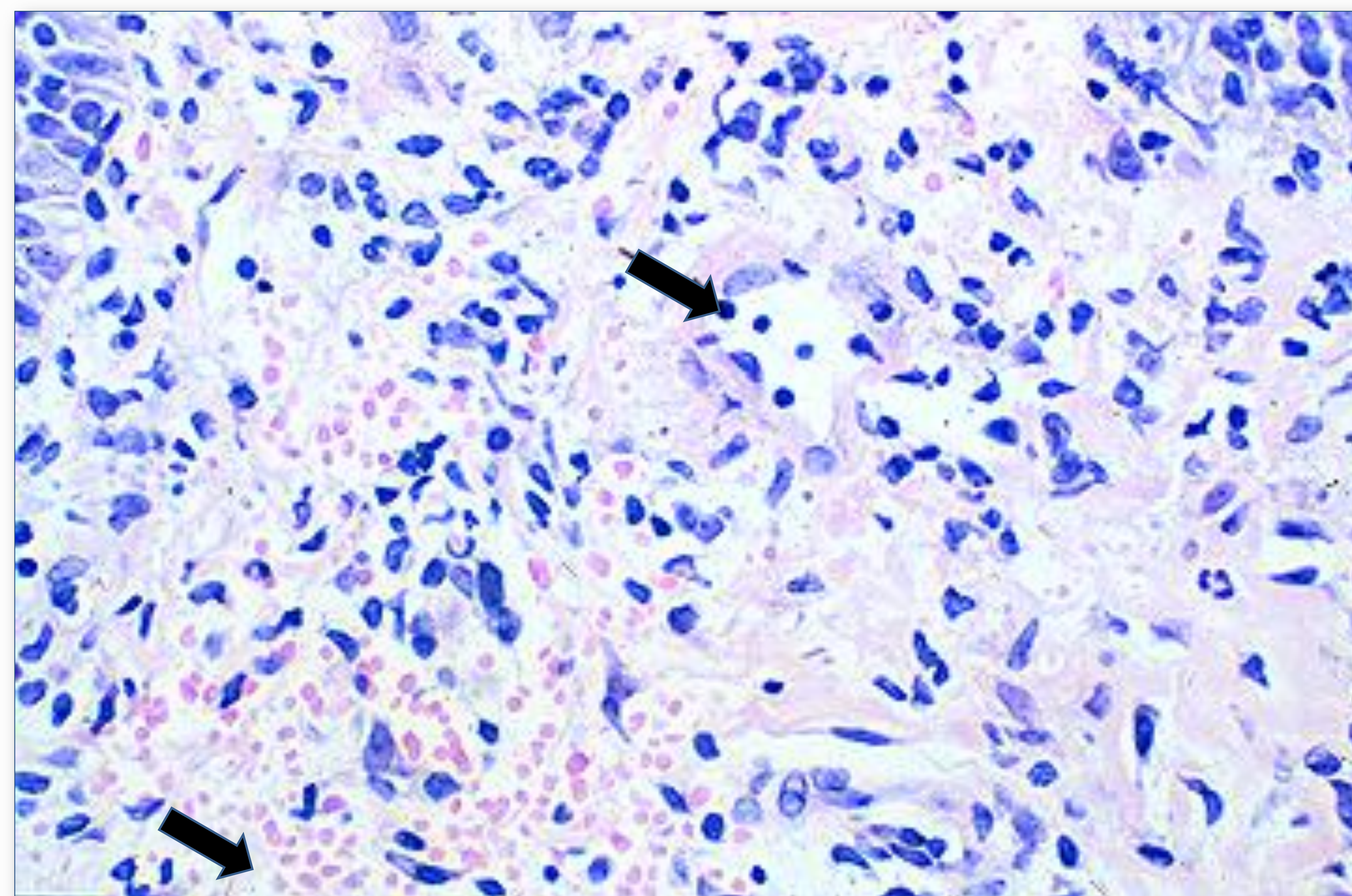
- Cutaneous leukocytoclastic angiitis, or hypersensitivity vasculitis, can result from treatment by many commonly prescribed medications
- Patients present with various manifestations, which may delay diagnosis and appropriate treatment

## Case Presentation

- History: 34 year-old previously healthy Hispanic female was transferred from an outlying hospital for concerns of progressive rash of two weeks duration with lower extremity edema, severe pruritus, arthralgia, abdominal pain
- Past medical history: newly diagnosed with hypertension, treated with Hydrochlorothiazide for one month
- Laboratory: UA, CBC, ANCA, ESR, Complement, RAF, ANA Panel, Cryoglobulin, HIV, Hepatitis studies were normal
- Cutaneous Biopsy: Inflammatory infiltration of lymphocytes and neutrophils, extravasation of erythrocytes, fibrin deposition in the walls of the small blood vessels with no evidence of IgA antibodies by direct immunofluorescence
- Treatment: discontinued Hydrochlorothiazide, began high dose IV steroid therapy with transition to oral taper
- Follow up: patient revealed prolonged recovery with continued hyperpigmented lesions and peripheral neuropathy



Palpable Purpura with Hemorrhagic Bullae



Punch Biopsy:  
Extravasation of lymphocytes and erythrocytes.

## Discussion

- Hypersensitivity vasculitis is a clinical syndrome due to immune complex deposition in capillaries, venules, and arterioles<sup>1</sup>
- Clinical symptoms are most commonly palpable purpura, but also includes arthralgia, myalgia, fever
- Latent period typically 7-10 days after exposure to offending agent
- Diagnostic Criteria<sup>2</sup>
  - Age > 16
  - Offending agent
  - Palpable purpura
  - Maculopapular rash
  - Biopsy with neutrophils around arterioles/venules
- Common insulting medications include penicillin, cephalosporins, loop diuretics, and thiazide diuretics
- These agents function as a hapten to stimulate an immune response<sup>3</sup>
- Infections such as hepatitis and HIV have also been associated
- Although an infrequent adverse reaction to a regularly prescribed medication, providers must be aware of this risk to provide appropriate treatment and prevent long term sequelae

## References

1. Tosca N, Stratigos JD. Possible pathogenetic mechanisms in allergic cutaneous vasculitis. *Int J Dermatol* 1988;27(5):291-6.
2. Calabrese LH, Michel BA, Bloch DA, et al. The American College of Rheumatology 1990 criteria for the classification of hypersensitivity vasculitis. *Arthritis Rheum* 1990;33(8):1108-13.
3. Calabrese LH, Duna GF. Drug-induced vasculitis. *Curr Opin Rheumatol* 1996;8(1):34-40.