Ceftriaxone-induced leucocytoclastic vasculitis

Daniela Andrea Haehn,¹ Archi Patel,² George Youngberg,² Alexei Gonzalez-Estrada³

¹Mayo Clinic Hospital
Jacksonville, Jacksonville,
Florida, USA

²Department of Pathology,
East Tennessee State University
James H Quillen College
of Medicine, Johnson City,
Tennessee, USA

³Division of Allergy and Clinical
Immunology, Department of
Medicine, East Tennessee State
University James H Quillen
College of Medicine, Johnson
City, Tennessee, USA

Correspondence to

Dr Alexei Gonzalez-Estrada,
gonzalez.alexei@mayo.edu

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DESCRIPTION

A 35-year-old man without history of drug allergy and a medical history of essential hypertension, intravenous drug abuse, and chronic hepatitis C with low viral load presented for evaluation with a 5-day rash in lower extremities associated with crimping joint pain. The patient had been on ceftriaxone for the last 14 days due to a methicillin-sensitive *Staphylococcus aureus* tricuspid endocarditis. Patient referred that the rash started as blisters on his ankles that then became purpuric and progressed upwards to involve his lower extremities. On physical examination, multiple non-blanching purpuric lesions limited to lower extremities were noted (figure 1A). Our clinical suspicion was leucocytoclastic vasculitis diagnosis. Skin biopsy was performed showing extravasation of erythrocytes, perivascular neutrophil degeneration and necrosis of the vessels (figure 1B). A diagnosis of leucocytoclastic vasculitis (LCV) was made. The patient was treated with systemic corticosteroids; ceftriaxone was discontinued, and daptomycin was initiated with improvement of the LCV.

LCV is a small vessel of the skin with immune complex that spares internal organs generally secondary to drug exposure or infection.¹ Half of the cases of LCV have been associated with chronic infections such as hepatitis B or C virus, infective endocarditis, infective shunts and HIV.² Histological findings in skin biopsy include vascular and perivascular deposition of neutrophils of the vessels walls, nuclear dust and extravasated erythrocytes.³ The diagnosis of LCV is suggested by clinical manifestation and a history of initiation of a new drug or infection.¹

Figure 1 (A) Multiple non-blanching purpuric lesions limited to the lower extremities. (B) Skin biopsy of a purpuric lesion showing extravasation of erythrocytes, perivascular neutrophil degeneration and necrosis of the vessels.

Learning points

- ▶ Leucocytoclastic vasculitis (LCV) most commonly occurs secondary to drugs but can be also secondary to chronic infectious diseases such as hepatitis B or C virus, infective endocarditis and HIV.
- ▶ Many medications are commonly implicated causes of LCV, but penicillins, cephalosporins, sulphonamides, phenytoin and allopurinol are the most common agents.

Non-blanching palpable purpura is the most common manifestation, but other skin lesions such as maculopapular rash, bullae, papules, plaques, nodules, ulcers and livedo reticularis can also be present. Skin biopsy confirms the diagnosis.³ Management of LCV is directed to treat the underlying cause. An isolated case of LCV by a known trigger should be treated with discontinuation of the trigger.³ Chronic, recurrent or severely symptomatic LCV should be treated with azathioprine, mycophenolate mofetil, methotrexate or rituximab should be started.³

There is a documented association between hepatitis C, infective endocarditis and initiation of a new drug. We believe that the causative agent in this case was the ceftriaxone since discontinuation of the aetiological agent improved the rash. Unfortunately, the patient was a lost to follow-up.

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REFERENCES

- 1 Cakiter AU, Kucuk OS, Ozkaya DB, et al. Demographic characteristics, aetiology, and assessment of treatment options in leukocytoclastic vasculitis. *Postepy Dermatol Alergol* 2017;2:104–9.
- 2 Blanco R, Martínez-Taboada VM, Rodríguez-Valverde V, et al. Cutaneous vasculitis in children and adults: associated diseases and etiologic factors in 303 patients. *Medicine* 1998;77:403–18.
- 3 Goeser MR, Lanioz V, Wetter DA. A practical approach to the diagnosis, evaluation, and management of cutaneous small-vessel vasculitis. *Am J Clin Dermatol* 2014;15:299–306.

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