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Drug-Induced Cutaneous Vasculitis: A Report of Three Cases

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Introduction & Objectives:

Drug-induced cutaneous vasculitis is a hypersensitivity reaction affecting small dermal vessels. Many drugs have been implicated. We report three cases triggered by different pharmacological agents..

Materials & Methods:

Case1 An 84-year-old woman with diabetes, hypothyroidism, and hypertension developed palpable purpura and necrotic ulceration on the lower limbs 10 days after starting lercanidipine. She had no fever or infectious signs. Labs showed eosinophilia (1300/mm^3), elevated ESR, negative CRP, and acute renal failure. Skin biopsy from an older lesion showed dermal lymphohistiocytic infiltrates. Discontinuation of lercanidipine led to near-complete lesion regression and rapid renal function improvement within two weeks.

Case2 A 44-year-old man, hospitalized for an ankle fracture, developed palpable purpura on his upper and lower limbs seven days after starting cefotaxime and ciprofloxacin. He had no systemic symptoms. Lab tests revealed neutrophilic leukocytosis and elevated CRP; ANCA, ANA, and serologies were negative. Skin biopsy showed dermal fibrosis with a lymphohistiocytic infiltrate. Stopping the antibiotics led to rapid clinical resolution.

Case3 A 23-year-old woman with Crohn’s disease developed palpable purpura on her limbs, with arthralgia, one week after her first adalimumab injection. No renal or visceral involvement was found. Lesions regressed within a week after stopping the medication.

Results:

Drug-induced cutaneous vasculitis is typically a leukocytoclastic small-vessel vasculitis caused by delayed hypersensitivity to medications. Onset varies from days to weeks after exposure. Clinically, it presents mainly as palpable purpura, sometimes with systemic symptoms, but can mimic primary vasculitis or infections, complicating diagnosis.

In our cases, the temporal relationship with drug exposure, absence of major systemic involvement, and rapid resolution after drug withdrawal supported a drug-induced etiology. The implicated agents—lercanidipine, cephalosporins, fluoroquinolones, adalimumab—illustrate the wide pharmacologic range of potential triggers.

Histopathology can assist diagnosis but is ideally performed on fresh lesions (<48h), as older lesions may lose characteristic vascular changes, limiting diagnostic value.

Conclusion:

In any recent-onset cutaneous vasculitis, drug-induced causes should be systematically considered and assessed through clinical evolution after withdrawal of the suspected agent.