**CLINICAL VIGNETTE**

# Atypical Presentation of UTI in Elderly – Cutaneous Small Vessel Vasculitis

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***Case***

An 86-year-old Hispanic female with history of hypertension, hyperlipidemia, prediabetes, urinary incontinence, and osteoporosis presented to clinic with 1 week of non-pruritic, non-painful rash in legs. She denied any new medications, recent illnesses, coughs, dysuria, hematuria, diarrhea, joint pain, oral ulcers, or fever. Patient is not a drinker or smoker. She does not have any known allergies. Her only medications are daily amlodipine 2.5mg and aspirin 81 mg. On exam, her vital signs are normal. She has erythematous non-blanching mascules and patches without palpable component in distal thighs and lower legs. Basic blood tests along with urinalysis and culture were ordered. Urinalysis returned with WBC clumps and 3+ leukocyte esterase and she was empirically treated for urinary tract infection with cephalexin. The next day she was seen by Dermatology and culture confirmed pan-sensitive e coli. Two 3mm punch biopsies were obtained for immunofluorescence. A few days later the rash resolved. Biopsy showed "one to two C3 highlighting small dermal blood vessels." IgG, IgM, IgA stains were negative.

***Discussion***

Vasculitis is inflammation of blood vessel walls. Sometimes direct invasion (infection) of microorganisms can cause vasculitis (for example, syphilis aortitis). According to 2012 CHCC (International Chapel Hill Consensus Conference) nomenclature system,[1] *non*infectious vasculitis can be categorized into large vessel, medium vessel, and small vessel vasculitis as well as variable vessel vasculitis, single-organ vasculitis (for example, skin-isolated vasculitis), vasculitis associated with systemic diseases (for example, lupus), and vasculitis associated with probable etiology (for example, hepatitis). Among these, cutaneous small vessel vasculitis has also been called hypersensitivity angiitis, cutaneous leukocytoclastic angiitis, and serum sickness. By definition, cutaneous small vessel vasculitis (CSVV) is a single-organ, skin-isolated vasculitis without systemic vasculitis or glomerulonephritis.[2] Histologically, it is characterized by a leucocytoclastic (neutrophilic infiltration) vasculitis.

Causes of CSVV includes infections such as hepatitis B or C virus and chronic bacteremias (eg, infective endocarditis) as well as medications such as penicillins, cephalosporins, sulfonamides including loop and thiazide diuretics), and allopurinol. CSVV usually presents as nonblanching palpable purpura or petechiae, beginning 7 to 10 days after the inciting event. The diagnosis of CSVV is supported by clinical findings and the history. Cutaneous lesions suspicious for vasculitis must be biopsied. Since cutaneous vasculitis may evolve quickly, it is important to perform the biopsy within the first 24 to 48 hours after the onset of cutaneous lesions.

It is possible that this patient's biopsy showed only C3, which is evidence for complement activation but did not show neutrophils because the biopsy was done a week after the lesion appeared. The clinical challenge is the same cutaneous presentation can be seen in multiple types of vasculitides. In addition to looking for an occult infection that can be diagnosed with relatively easy workup as in this case, a clinician must consider workup for other systemic causes and associated conditions when a patient's biopsy shows vasculitis. In this patient, c-ANCAs, p-ANCA, PR3 (proteinase-3 antibody), MPO (myeloperoxidase antibody), hepatitis serologies, rheumatoid factor, dsDNA, Sm/RNP antibodies, SSA/SSB antibodies, CH 50 as well as C3 and C4 levels, HIV were unremarkable.

Once the diagnosis is made, the management is expectant. A prospective, randomized controlled trial showed no therapeutic effect of colchicine.[3] The inciting drug should be discontinued and the infection should be treated. The lesions tend to disappear within days to a few weeks.

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