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Case Report

When the skin speaks: cutaneous vasculitis complicating a *Klebsiella* urinary tract infection

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ABSTRACT

Cutaneous small-vessel vasculitis (CSVV) occurs due to leukocytoclastic vasculitis of dermal post-capillary venules. It can be triggered by infections, autoimmune conditions, or medications. Our case report highlights a rare association between CSVV and a urinary tract infection caused by *Klebsiella pneumoniae* in an immunocompetent individual. A 31-year-old female with no prior known comorbidities presented with painful, pruritic lesions over both lower limbs and right arm preceded by gastrointestinal symptoms. On examination, there were erythematous macules, crusted plaques and haemorrhagic vesicles noted. A skin biopsy confirmed CSVV. Autoimmune markers were negative, while complement C3 levels were reduced. Urine culture grew *Klebsiella pneumoniae*, suggesting a post-infectious etiology. The patient was treated with antibiotics and steroids resulting in marked clinical improvement. Infections are common triggers of CSVV, however *Klebsiella pneumoniae* is an uncommon cause, particularly in immunocompetent individuals. This case underlines the importance of considering uncommon etiologies in the differential diagnosis of CSVV and emphasizes that prompt identification and treatment of the underlying infection can lead to complete resolution. This case contributes to the limited literature on *Klebsiella pneumoniae*-associated CSVV in immunocompetent individuals and underscores the value of biopsy, infectious work-up, and early therapy in achieving good outcomes.

Keywords: *Klebsiella pneumonia*, Leukocytoclastic vasculitis, UTI, Immunocompetent

INTRODUCTION

Cutaneous small-vessel vasculitis (CSVV) is a skin-limited variant of systemic vasculitis caused by leukocytoclastic vasculitis of postcapillary venules.[1,2] It can be idiopathic or secondary to infection, medication, connective tissue disease, or malignancy among other causes. The most common clinical presentation is of symmetrically distributed palpable purpura of the lower extremities.[3]

Histopathology is the gold standard for diagnosis with features appearing 24-48 hours after lesions emerge.[4] Our case report presents an unusual case of a post infectious biopsy proven CSVV which responded well to steroids and antibiotics in an otherwise healthy female.

CASE REPORT

A 31-year-old female, with no prior known comorbidities, presented with a history of painful and itchy lesions on her bilateral lower limbs since 10 days. Similar lesions also appeared on her right upper limb. The lesions initially appeared as erythematous macules but soon progressed into vesicles and bullae, which eventually crusted over. She also reported fever and joint pain for the past two days prior to hospitalisation. A week prior to onset of her skin symptoms, the patient had experienced abdominal pain and vomiting, which had resolved after treatment. On general examination, both lower limbs were edematous with multiple crusted plaques on the dorsum of both lower limbs with some tender erythematous macules and a few hemorrhagic vesicles scattered across the area. The right

arm also showed a few similar lesions. No lymphadenopathy was noted. Laboratory investigations revealed elevated inflammatory markers. Dermatology consultation was done and a skin biopsy was performed. The pathology report showed features consistent with CSVV. To rule out autoimmune causes, an ANA (antinuclear antibody) profile and anti-neutrophil cytoplasmic antibody (ANCA) was sent, which came back negative. Complement C3 was found to be low, while C4 levels were normal, suggesting a possible immune response. On further evaluation, she was also diagnosed with an underlying urinary tract infection, and her urine culture grew *Klebsiella pneumoniae*, a possible trigger for her immune response. A rheumatology consultation was sought, and a diagnosis of probable post-infectious vasculitis was made.

The patient was started on an appropriate course of antibiotics and systemic steroids were initiated to manage the vasculitis. Over the following days, the patient showed marked improvement in her general condition, and her skin lesions began to heal. She was discharged and is on regular follow up.

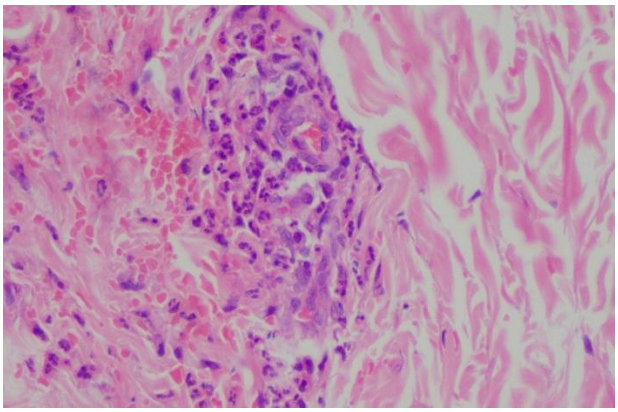

Figure 1: Extravasation of RBCs seen in leukocytoclastic vasculitis.

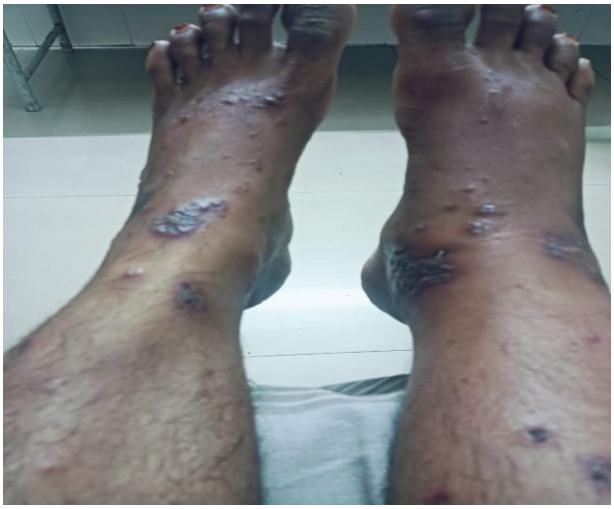

Figure 2: Skin lesions noted at the time of onset of symptoms.

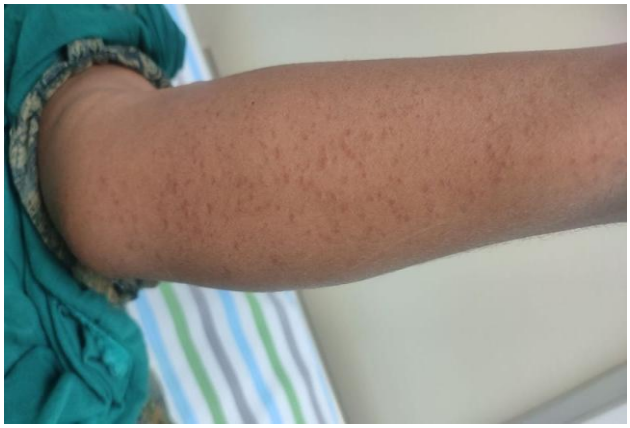

Figure 3: Erythematous maculopapular lesions over right upper limb.

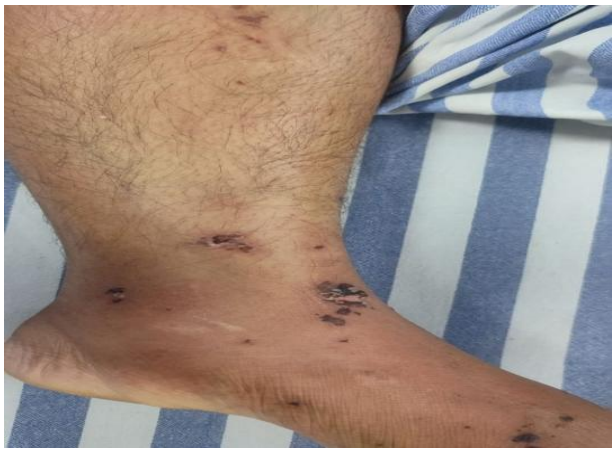

Figure 4: Healing skin lesions after initiation of treatment.

DISCUSSION

CSVV, is an immune complex-mediated inflammation of post-capillary venules in the dermis. Among the identified causes, infections are well known triggers. In a retrospective study by Sais et al it was found that infections were responsible for approximately 15-20% of vasculitis cases. They cause type III hypersensitivity reaction with immune complex deposition and complement activation finally leading to leukocytoclasia.[5] Most common bacterial pathogens are gram positive cocci like *Staphylococcus aureus* and *Streptococcus pyogenes* but our patient was diagnosed with a urinary tract infection caused by *Klebsiella pneumoniae*, a pathogen not commonly associated with CSVV.[6-8] *Klebsiella pneumonia* is a gram negative bacilli with virulence factors like lipopolysaccharide (LPS) which can serve as a antigenic stimuli for triggering immune responses.[9] While *Klebsiella pneumonia* is a frequent cause of UTI and can lead to severe complications such as pyelonephritis and bacteraemia, its association with cutaneous vasculitis is rare and seen typically in the setting of underlying comorbidities like diabetes, chronic kidney disease or any immunocompromised state.[10] In this case, the patient had

no known comorbidities, making the occurrence of *Klebsiella*-associated CSVV particularly notable. Cutaneous symptoms in post-infectious vasculitis appear 7-14 days after the triggering event, aligning well with the onset in this case.[11] In our case, the negative ANA and ANCA panels ruled out autoimmune vasculitis and supports the diagnosis of isolated post-infectious cutaneous vasculitis, a diagnosis of exclusion. Biopsy is crucial for confirming the diagnosis, showing characteristic findings of fibrinoid necrosis, neutrophilic infiltration, and extravasated red blood cells.[12] The treatment primarily focuses on antibiotic therapy to address the underlying infection. In our patient's case, the *Klebsiella pneumoniae* infection was successfully treated with appropriate antibiotics, leading to a resolution of both the infection and the vasculitis. Systemic corticosteroids may be used in more severe cases to manage the inflammatory response and control the symptoms of vasculitis. The prognosis is generally favourable when the underlying infection is treated promptly, as seen in our case.[13,14]

CONCLUSION

The case illustrates that even localised uncomplicated infections caused by *Klebsiella pneumoniae*, can trigger vasculitis even in patients with no prior comorbidities or immune deficiencies. Early diagnosis with skin biopsy and treatment of the underlying infection is crucial for management.

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