

BRIEF ARTICLE

A Palmoplantar Puzzle: Murine Typhus Involving the Palms and Soles

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ABSTRACT

Murine typhus, also known as “flea-borne” or “endemic” typhus, is a zoonotic infection caused by *Rickettsia typhi*. It is characterized by fever, headache, and a non-pruritic maculopapular rash that typically spreads centrifugally across the trunk while sparing the palms and soles. We present the case of a 51-year-old female with serologically confirmed murine typhus, who exhibited atypical cutaneous manifestations, including a purpuric rash involving her palms and soles. This case highlights how palmoplantar involvement, although rare, should not exclude murine typhus from diagnostic consideration. Dermatologists should be aware of atypical presentations of murine typhus and start empiric treatment with doxycycline while awaiting serologic confirmation.

INTRODUCTION

Murine typhus is a zoonotic infection caused by the intracellular gram-negative bacterium *Rickettsia typhi*. It is primarily transmitted via inoculation of infective flea feces into bite wounds. Although prevalent in tropical and sub-tropical regions, including the southern United States, murine typhus often presents with nonspecific symptoms, leading to frequent misdiagnosis.^{1,2} Following an incubation period of 7 to 14 days, the disease is classically characterized by the abrupt onset of fever, headache, and rash.^{3,4} The rash, which is observed in 20% to 80% of patients, typically appears one week after fever onset as a non-pruritic, maculopapular eruption that spreads centrifugally across the trunk while sparing the palms and soles.

Without timely diagnosis and treatment, murine typhus can lead to severe or even fatal complications including respiratory failure, disseminated intravascular coagulation, septic shock, and hemophagocytic syndrome.²⁻⁴ We describe the case of a 51-year-old female with serologically confirmed murine typhus. She exhibited non-classic symptoms, including a purpuric rash involving her palms and soles.

CASE REPORT

A 51-year-old female was admitted to the hospital with a 2-week history of acute-onset fever, myalgias, arthralgias, diarrhea, and rash. She reported living alone with a dog, but denied any sick contacts, high-risk sexual behavior, recent travel, new medications, or

history of autoimmune disease. On physical examination, the patient appeared fatigued and ill, with asymptomatic, purpuric macules diffusely involving her trunk and extremities, including the palms and soles (**Figures 1 and 2**). She was found to have a low-grade fever (100.3°F) and tachycardia with a lactic acidosis of 4.17 mmol/L. Laboratory investigations revealed anemia with a hemoglobin of 9.0 g/dL, thrombocytopenia with a platelet count of 46,000 per mm³, multiple electrolyte derangements, acute kidney injury with a creatinine of 3.27 mg/dL, hypoalbuminemia of 2.1 g/dL, and transaminitis (aspartate transaminase 134 U/L, alanine transaminase 105 U/L).

Punch biopsy of the left thigh showed an intraepidermal neutrophilic pustule, as well as a superficial perivascular infiltrate with neutrophils, leukocytoclasia, fibrin deposition within small vessels, and red blood cell extravasation, consistent with leukocytoclastic vasculitis (**Figure 3**). Direct immunofluorescence was positive for perivascular IgG and C3.

Given the clinical signs and symptoms, a rickettsial infection was strongly suspected, and empiric treatment with doxycycline was initiated while awaiting the results of Karius testing, which detects microbial cell-free DNA of distinct bacteria, fungi, protozoa, and DNA viruses in a patient's serum. Within 4 days, the patient exhibited marked improvement in both her laboratory abnormalities and systemic symptoms, leading to prompt recovery and eventual discharge after completing a 7-day course of doxycycline. After several weeks, Karius test results confirmed the presence of *R. typhi*, thus establishing the diagnosis of murine typhus.

DISCUSSION

Murine typhus, also known as “flea-borne” or “endemic” typhus, is transmitted when flea bite wounds become inoculated with flea feces.^{1,2} Following infection of cutaneous phagocytic cells, *R. typhi* are transported through the lymphatic system to the bloodstream, where they replicate and disseminate.⁵ Phagocytosis by human endothelial cells surrounding small- and medium-sized vessels results in endothelial cell injury, which manifests as a lymphohistiocytic or, more rarely, a neutrophilic vasculitis.^{5–8} *Rickettsia* vasculitis may occur in any organ, and can present as a rash, interstitial pneumonia, disseminated intravascular coagulation, meningoencephalitis, or even multi-organ failure.^{5,6,9}

Epidemiologic surveys on murine typhus highlight the risk of outbreaks due to its changing ecology and underestimated incidence.⁴ Although classically associated with a rat-flea-rat cycle, urban and suburban expansion has facilitated a peri-domestic animal cycle propagated by cats, dogs, opossums, and their fleas.¹⁰ The disease features nonspecific symptoms, including myalgias, anorexia, chills, and malaise, making the diagnosis challenging. The usefulness of the classic triad of fever, rash, and headache in diagnosing murine typhus is limited, as it may be present in only one-third of patients.⁴ Furthermore, morphology and distribution of the rash are variable. While classically described as a non-pruritic macular or maculopapular eruption spreading centrifugally across the trunk while sparing the palms and soles, our patient presented with palpable purpura involving the palms and soles.^{2,3} Clinicians should be aware that palmoplantar involvement is possible, albeit present in less than 3% of murine typhus cases.⁹ This is in contrast to other rickettsial infections, including Rocky Mountain spotted fever and Mediterranean

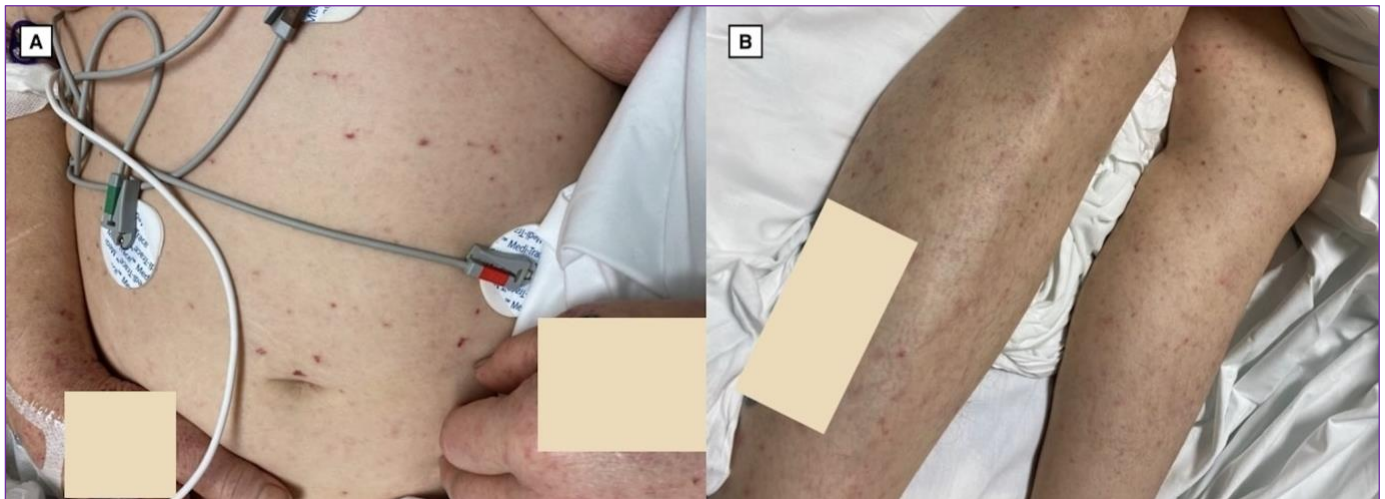


Figure 1. Purpuric macules and papules on the trunk (A) and lower extremities (B).



Figure 2. Purpuric macules on the soles (A) and palms (B).

spotted fever, which feature involvement of the palms and soles.¹ Similarly, a purpuric eruption and eschar at the site of inoculation are atypical cutaneous manifestations of murine typhus that are more commonly

associated with other rickettsial infections.^{11,12}

The nonspecific clinical presentation and evolving geographical setting of murine

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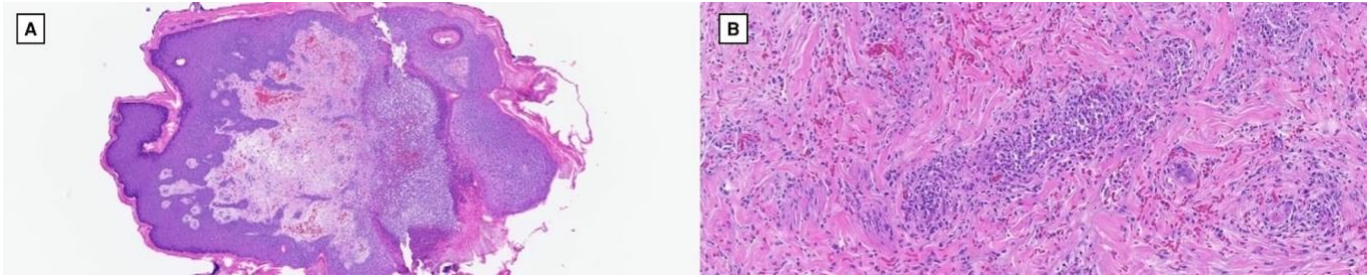


Figure 3. (A- H&E, 20X) Intraepidermal neutrophilic pustule with a primarily neutrophilic dermal infiltrate. (B- H&E, 200X) Perivascular neutrophils, leukocytoclasia, fibrin deposition within vessel walls, and red blood cell extravasation.

typhus pose a barrier to prompt patient care. A thorough social history may help identify risk factors, such as exposure to flea-infested animals. Empiric treatment with doxycycline for 5-7 days should be started while awaiting the results of serologic testing.⁹ The risk of outbreak can be minimized with involvement of the local health department.³

CONCLUSION

Murine typhus poses a diagnostic challenge due to its evolving ecology and clinical presentation, which may vary from classic teaching. Clinicians should be aware of the potential for cases to arise in new localities and that palmoplantar involvement does not preclude murine typhus from diagnostic consideration.

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