Crusted scabies triggering autonomic dysreflexia in a patient with spinal cord injury

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INTRODUCTION
Crusted scabies, also referred to as Norwegian scabies, is caused by an infestation with the Sarcoptes scabiei mite. Unlike classic scabies, in which a human host may be infected with 10 to 15 mites, an aberrant host response in crusted scabies leads to colonization with millions of mites. This condition is most often observed in patients with decreased sensory function and those who are immunocompromised, such as the elderly and those with underlying HIV, human T-lymphotropic virus 1 infection, and malignancy. Clinically, patients present with generalized thick, hyperkeratotic, psoriasiform, or eczematous-like plaques, most notable on acral surfaces. Pruritus may be absent. Systemically, generalized lymphadenopathy, peripheral eosinophilia, elevated immunoglobulin E levels, sleep disturbances, and secondary superinfections with Staphylococcus aureus and Streptococcus pyogenes are reported. Here, we present a rare case of untreated crusted scabies triggering autonomic dysreflexia in a patient with underlying spinal cord injury.

CASE PRESENTATION
A 45-year-old quadriplegic man with history of a complete C7 American Spinal Injury Association grade C spinal cord injury 15 years before initially presented to the emergency department with a several-day history of labile blood pressures with intermittent systolic blood pressures as high as 180 mm Hg and as low as 67 mm Hg, diaphoresis, and abdominal spasms. At baseline, the patient had advanced neurogenic bladder and urinary retention, for which he had had a suprapubic catheter placed years before, and gastroparesis secondary to neurogenic bowel. At this admission, he had exacerbation of these symptoms with increased urinary sediments, causing frequent catheter clogging, and a recent episode of stercoral colitis secondary to fecal impaction. Laboratory evaluation was notable for mild eosinophilia. The patient was admitted for acute management of his autonomic dysreflexia symptoms. During his admission, dermatology was consulted to evaluate a pruritic rash involving his hands, chest, back, groin, and upper and lower extremities. When further history was obtained, it was discovered that the rash began 10 months before his current admission and he had been treated with clobetasol 0.05% ointment because previous biopsy was suggestive of dyshidrotic eczema. On examination, the patient was diaphoretic and photophobic and appeared distressed. His skin examination revealed thick crusted plaques with caked-on scale, particularly in the interweb spaces, and scattered lichenified erythematous plaques with fine scale on the upper and lower extremities (Fig 1). Microscopic examination and skin scraping of the interweb spaces with mineral oil revealed live mites, supporting our suspicion of crusted scabies. On review of the patient’s initial skin biopsy from 10 months before, there was prominent eosinophilic spongiosis and a mite was also noted within the hyperkeratotic stratum corneum (Fig 2). The patient was treated with oral ivermectin 200 μg/kg (on days 1, 8, 15, and 22) and concomitant permethrin 5% cream applied to the entire body and left overnight (on days 1 and 15).

At 2-month follow-up, repeated scabies preparation result was negative and the patient was no longer pruritic. Review of neurology notes at his
3-month follow-up revealed that his neurogenic bowel and bladder symptoms were back to baseline, his blood pressure spikes had resolved, and he no longer required oral clonidine to control autonomic dysreflexia symptoms after successful treatment of the scabies.

**DISCUSSION**

Intermittent attacks of autonomic dysreflexia, or “sympathetic storm,” are well reported in patients with spinal cord injury at or above T6 spinal levels because of a decentralized spinal cord with interruption of descending modulation of thoracolumbar sympathetic preganglionic neurons above that level. Acutely, patients present with hypotension and bradycardia, but autonomic imbalance and dysregulation of splanchnic circulation lead to sympathetic overactivity, hypertension, and a baroreceptor-reflex-mediated bradycardia. Compensatory parasympathetic activity above the level of spinal injury could also cause secondary diaphoresis, rhinorrhea, and flushing. Patients can experience up to 40 attacks daily; thus, swift identification of triggers is vital because devastating cardiopysiological effects and even fatal complications can occur. Autonomic dysreflexia is well established in response to a number of noxious visceral and somatic stimuli below the level of injury, including urinary tract infections, deep vein thrombosis, constrictive clothing, pregnancy, orchitis, hemorrhoids, and surgical interventions. Cutaneous triggers of autonomic dysreflexia in spinal cord injury patients include skin infections, ulceration, and ingrown toenails. However, to our knowledge this is the first reported case in which scabies infestation has been implicated; this is supported by the resolution of the patient’s rash, peripheral eosinophilia, photophobia, bowel and bladder dysregulation, and labile blood pressures, with successful treatment of the infestation.
In addition to autonomic dysreflexia, scabies has been reported to trigger numerous other systemic diseases. Nine cases of scabies causing leukocytoclastic vasculitis have been reported. Scabies infestation has also been associated with exacerbations of chronic obstructive pulmonary disease and bullous pemphigoid. In all these disorders, it is hypothesized that the presence of the scabies mite causes a hypersensitivity-like reaction and immunoresponse to an unknown antigen in individuals affected. Recently, there also have been increased data to support that patients with a history of treated scabies are at a long-term higher risk for other systemic diseases, such as autoimmune disease or stroke. A recent population-based cohort study revealed that compared with matched controls, patients with a history of scabies showed an increased risk of developing hypersensitivity vasculitis, dermatomyositis, polyarteritis nodosa, systemic lupus erythematosus, psoriasis, myasthenia gravis, type 1 diabetes mellitus, pernicious anemia, and rheumatoid arthritis in a 7-year period. Similarly, another population-based study showed an elevated risk of stroke in patients with a history of scabies during a 7-year follow-up period; however, patients with scabies also had a higher incidence of metabolic syndrome, which is a well-established risk factor for stroke alone. More research is needed to elucidate the pathogenesis to better counsel patients with scabies, like the patient described in our case, about their imminent and long-term risks.

In conclusion, we report this case to highlight an unusual infection triggering autonomic dysreflexia and the need to swiftly identify crusted scabies from a public health and neurologic standpoint.

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