Our results are in agreement with a more recent study by Johnston et al[1] in Utah that found that although relative humidity was increased in homes with ECs, levels were still below the critical equilibrium humidity required for HDM survival. They argued that continuous humidity monitoring in their study was more accurate than single measurements and likely accounted for their opposing findings compared with previous studies. Limitations of our study include its retrospective nature. Also, other important factors that can affect HDM and mold sensitization, including water damage and housing characteristics, such as age of home and mattress, amount and type of furniture, type of flooring, and occupant density, were not evaluated.[1,7,11] In conclusion, ECs increase the relative humidity in the home, but this does not increase HDM or mold sensitization in pediatric patients with atopy residing in Colorado. These results indicate that providers and patients in arid or semiarid climates who choose to use an EC do not need to be concerned about developing HDM or mold sensitization or this contributing to their atopic disease.

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A case of neuropathic pain in monoclonal mast cell activation syndrome

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Neuropathic pain is an uncommon symptom associated with mast cell disorders but can significantly impact quality of life. Mast cells are known to be gatekeepers of nociception and play an important role in the development of neuropathic pain in the central and peripheral nervous system.[1] Although musculoskeletal pain has been associated with mast cell disorders,[2] neuropathic pain associated with mast cell disorders and its management has not been well described in the literature. We present a patient with neuropathic pain associated with monoclonal mast cell activation syndrome (MMAS) and review the agents used in his treatment.

The patient was a 49-year-old male with a past medical history significant for allergic rhinitis on allergen immunotherapy, who presented with a history of spontaneous flushing episodes for 4 years, 2 associated with loss of consciousness. He underwent extensive evaluations ruling out cardiac, neurologic, or endocrine etiologies of these episodes. After an episode of anaphylaxis after an allergen immunotherapy injection (serum tryptase 28.6 ng/mL at 1 hour after symptom onset), he had persistently elevated baseline serum tryptase (17.6 ng/mL) 1 week later. Repeat serum tryptase 1 month later remained elevated at 14.4 ng/mL, which prompted further evaluation for an underlying mast cell disorder.[2] Physical exam was without evidence of concerning skin lesions such as urticaria pigmentosa. Bone marrow biopsy revealed no mast cell aggregates or abnormal morphology but was positive for the KIT D816V mutation, leading to the diagnosis of MMAS, as no other criteria for systemic mastocytosis were met.[3] No other studies evaluating mast cell mediators in the blood, urine, or cerebrospinal fluid were performed.

The patient's flushing episodes worsened, increasing in frequency to 10 times daily. Concurrent with the increased frequency of flushing, he began complaining of pain in his wrists and forearms, progressing to his lower extremities over 1 year. Pain was described as severe burning, tingling, and numbness, becoming so debilitating he became wheelchair-bound. Prior to the onset of pain, he did not receive any vaccines nor were there any changes to his medications or daily activities. Treatment regimens for flushing included H_1 and H_2 antihistamines (fexofenadine 180 mg twice daily, ranitidine 150 mg twice daily), a leukotriene antagonist (montelukast 10 mg daily), cromolyn sodium 200 mg orally 4 times daily, and aspirin 81 mg daily for refractory flushing. Flushing improved on this regimen; a short trial of omalizumab had no effect.

Despite control of the flushing, his pain worsened and became increasingly difficult to treat, prompting a neurology referral. On evaluation he showed no evidence of monoclonal gammopathy, chronic infections, autoimmune disease, metabolic, toxic, or genetic etiologies of pain. Skin biopsy revealed normal numbers of epidermal nerve fibers; however, autonomic function testing revealed abnormal heart rate response to deep breathing, abnormal blood pressure response to Valsalva maneuver, and abnormal quantitative sudomotor axon reflex testing in the proximal and distal leg. Based on his clinical presentation and test results, the patient was diagnosed with painful small-fiber neuropathy and pandysautonomia.

Several medication trials for the treatment of his pain over the course of 6 months (Table 1), including nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, a selective norepinephrine reuptake inhibitor, gamma-aminobutyric acid analogs, opioids, and topical pain medications, were ineffective in improving his pain. He further underwent a trial of high-dose intravenous gammaglobulin[4] for 3 months without improvement in pain, and repeat autonomic testing showed worsening pandysautonomia.

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Table 1
Medications Used to Treat Neuropathic Pain in Our Patient Prior to Trial of Minocycline

Drug class	Medication(s)
NSAID	Ibuprofen, naproxen, aspirin, celecoxib
TCA	Amitriptyline, nortriptyline
SNRI	Duloxetine
GABA analogs	Pregabalin, gabapentin
Opioids	Tramadol, oxycodone
Topical pain medication	Topical lidocaine, topical capsaicin

Abbreviations: GABA, gamma-aminobutyric acid; NSAID, nonsteroidal anti-inflammatory drug; SNRI, selective norepinephrine reuptake inhibitor; TCA, tricyclic antidepressant.

Minocycline was tried as an alternative therapy for the patient's refractory pain. After 3 months of minocycline 100 mg twice daily, he reported subjective improvement of his pain symptoms, with objective improvement on autonomic function testing. Cardiovagal function normalized with autonomic function testing showing a normal heart rate response to deep breathing and normal blood pressure response to Valsalva maneuver, and quantitative sudomotor axon reflex testing was normal in his proximal and distal leg. At the time of the minocycline trial, the patient continued to take gabapentin, duloxetine, nortriptyline, and celecoxib. It is unclear whether the improvement was due to a combination effect of these medications with minocycline or due to minocycline alone.

There is increasing interest in the off-label use of minocycline as an alternative option for the treatment of pain and autonomic neuropathy. Studies have shown that minocycline is useful in preventing muscular pain hypersensitivity in human subjects[5] as well as attenuating bone cancer pain[6] and the development of diabetic neuropathy in rats.[7] In a randomized, placebo-controlled study, minocycline was found to improve peripheral and autonomic neuropathy associated with type 2 diabetes.[8]

Several mechanisms have been proposed to link neuropathic pain with mast cells. Mast cells in the central and peripheral nervous system are involved in neuroinflammation and the development of neuropathic pain,[9] especially those in close proximity to nociceptive C-fibers.[1] Mast cells release mediators including histamine, serotonin, interleukin-1β, tumor necrosis factor-α, and interleukin-6, which can induce hyperalgesia, and mast cell–derived nerve growth factor can lower the threshold of nociceptor firing.[1] Mast cells also release substance P, which increases release of pronociceptive mediators, including leukotrienes and prostaglandins.[1]

The exact mechanism by which minocycline affects the pain pathway and autonomic nervous system has not fully been elucidated, but is being studied in murine models.[6,7] Minocycline reduces inflammatory cytokines and inhibits activation of microglia,[10] cells involved in the pain pathway. Minocycline has also been shown to inhibit spinal cord Notch signaling in rats with effects on the development of diabetic neuropathy.[7]

Our case demonstrates the successful use of minocycline as an alternative agent in the treatment of refractory neuropathic pain in the setting of underlying MMAS after treatment failure with typical pain medications. The case also highlights the potential challenges in managing neuropathic pain associated with MMAS, which will likely require a multidisciplinary approach to refine medication treatment options to improve quality of life in these medically complex patients. Although the case shows the promise of minocycline as an alternative pain agent, further studies will be necessary to validate its widespread use in patients with neuropathic pain. Further research is also needed to define the mechanisms involved in the development of neuropathic pain in patients with mast cell disorders.

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Face-to-face anti-tobacco intervention lowers cotinine level in asthmatic children

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In this study we measured urine cotinine levels to assess the effectiveness of "face-to-face" intervention. The secondary end point was to assess the effect of anti-tobacco parental interventions on lung function in children with asthma.

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Patients were recruited to each study group until the number of parents or guardians of 70 children in each intervention group was reached. Inclusion criteria were children aged 6 to 18 years, with perennial asthma, with parents/guardians living in the same household and confirmed smokers. An environmental tobacco smoke exposure scale was created for the study based on data from the questionnaire (Table 1). Exposure to environmental tobacco smoke (ETS) was evaluated with a questionnaire addressed to parents/guardians and cotinine measurements in children's urine (based on