

MS Consult

Bladder Complications in MS

Q My patient has multiple sclerosis and complains of feeling weaker, but denies urinary symptoms. Why have I been told to check for urinary tract infection and not just administer steroids?

Bladder complications are extremely common in patients living with multiple sclerosis (MS), occurring in around 80% of this population.¹ These complications—which include urinary urgency, failure to fully empty the bladder, incontinence, and difficulty getting to a toilet in time—can increase risk for urinary tract infection (UTI). And because many patients with MS also have sensory problems (eg, neurogenic bladder), they do not always present with the hallmark UTI symptoms of burning or pain with urination.

Often, presenting symptoms include generalized weakness, increased spasticity, or intensified neurologic issues. These can lead patients to believe they are having a relapse, when in fact, a UTI is causing a pseudoexacerbation of their baseline neurologic issues. In addition, frequent nocturia can disrupt sleep and further contribute to MS-related fatigue. Patients may self-induce dehydration by limiting their daytime fluid intake in an effort to avoid bathroom visits.¹

In partnership with urology colleagues, you can help mitigate bladder complications in patients with MS; this can entail use of medication or interventions such as in-and-out or straight catheterization, timed voids, Botox, or pelvic floor physical therapy. Behavior modifications—ie, minimizing caffeine intake, limiting alcohol consumption, and stopping fluids early in the evening—can also be beneficial.^{1,2}

Before initiating bladder medication, it

is important to review potential adverse effects with the patient. It's also crucial to ensure that patients are fully emptying their bladders before starting anticholinergic medications, as these can worsen retention.

Which treatment should you choose? Insurance companies tend to prefer generic, older-generation anticholinergics, but bear in mind that these can cause or contribute to cognitive issues (which many patients with MS already have).³ Another medication, such as mirabegron, may be preferable; it's less likely than anticholinergics to cause dry mouth, which may help with compliance. Also, be aware that anticholinergics can cause blurred vision, which might lead patients to believe they are having optic neuritis or another MS-related visual change.⁴

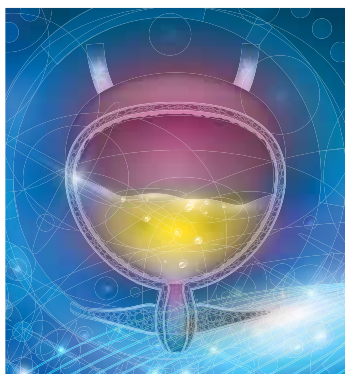
That said, it is possible for patients to have a relapse and a UTI simultaneously. Due to potential adverse effects, it is essential to balance the risks and benefits of steroid therapy. Steroids could worsen an untreated infection and may not be appropriate for the patient's symptoms or chief complaint.

Addressing bladder symptoms can not only help prevent UTIs but can also improve skin integrity, sleep quality, independence, and overall quality of life. A thorough exam and history-taking can alleviate secondary and tertiary urinary complications, as well as avoid unnecessary use of corticosteroids. —DRB


REFERENCES

1. Sheehan J. Coping with MS bladder dysfunction. www.everydayhealth.com/multiple-sclerosis/symptoms/coping-with-bladder-dysfunction/. Accessed November 18, 2017.
2. Mayo Clinic. Bladder control: medications for urinary problems. www.mayoclinic.org/diseases-conditions/urinary-incontinence/in-depth/bladder-control-problems/art-20044220. Accessed November 18, 2017.
3. Staskin DR, Zoltan E. Anticholinergics and central nervous

MS Consult is edited by **Colleen J. Harris, MN, NP, MSCN**, Nurse Practitioner/Manager of the Multiple Sclerosis Clinic at Foothills Medical Centre in Calgary, Alberta, Canada, and **Bryan Walker, MHS, PA-C**, who is in the Department of Neurology, Division of MS and Neuroimmunology, at Duke University Medical Center in Durham, North Carolina. This month's responses were authored by **Denise R. Bruen, MSN, APRN-BC, MSCN**, who is with the University of Virginia in Charlottesville, and **Maureen A. Mealy, BSN, MSCN**, who is Neuromyelitis Optica Clinical Research Program Manager, Senior Research Nurse of the Transverse Myelitis & Multiple Sclerosis Centers, and PhD candidate at Johns Hopkins School of Nursing in Baltimore.



Credit: Tefi / Shutterstock



International Organization of Multiple Sclerosis Nurses (IOMSN) is the first and only international organization focusing solely on the needs and goals of nurses involved with the care, education, research, and advocacy for multiple sclerosis and related autoimmune disorders of the central nervous system. For more information on IOMSN, visit www.iomsn.org.

system effects: are we confused? *Rev Urol.* 2007;9(4):191-196.

- Geller EJ, Crane AK, Wells EC, et al. Effect of anticholinergic use for the treatment of overactive bladder on cognitive function in post-menopausal women. *Clin Drug Investig.* 2012;32(10):697-705.

Diagnosing & Treating Neuromyelitis Optica Spectrum Disorder

Q How do you know if a neurologic symptom is due to a relapse of neuromyelitis optica spectrum disorder? And how should a confirmed

relapse be treated?

Neuromyelitis optica spectrum disorder (NMOSD) is a severe, relapsing autoimmune disease of the central nervous system (CNS) that targets the optic nerves and spinal cord, leading to blindness and paralysis.^{1,2} Whereas multiple sclerosis (MS) is characterized by demyelination, NMOSD is associated with astrocytic damage and tissue necrosis.³ Because longitudinally extensive inflammatory lesions are typical with NMOSD, permanent CNS damage is common with each relapse.⁴

Health care providers first need to determine whether a patient with NMOSD who presents with new or worsening symptoms is having a relapse. A relapse is caused by a breach of the blood-brain barrier by the peripheral immune system, which leads to inflammation and damage to the CNS.⁵ This causes neurologic symptoms that depend on the anatomic location. Once damage has occurred, symptoms may result either from a new relapse in the same location as a previous inflammatory event or from a pseudorelapse.⁶

Pseudorelapses are triggered by a systemic metabolic imbalance; they exacerbate symptoms from previous CNS damage. Differentiating between a true relapse and a pseudorelapse can be a diagnostic challenge for even the most seasoned of health care providers. Kessler et al retrospectively examined which clinical factors can distinguish relapses from pseudorelapses.⁶ Their find-

ings suggest that while clinical examination alone may be effective in events involving vision loss, MRI may be necessary when signs and symptoms are attributable to a spinal cord lesion.

In fact, they found that the degree of clinical worsening in patients with spinal cord symptoms caused by a pseudorelapse was similar to that of a true relapse. The most common causes of pseudorelapse included infection, dysautonomia, metabolic abnormalities, and changes to medication regimens. Interestingly, the presence of infection did not rule out a relapse, as patients experiencing relapses were equally likely as those with pseudorelapse to have a urinary tract infection. The authors concluded, based on their data, that an MRI is warranted to verify a relapse in patients who experience worsening of symptoms localized to the spinal cord but is not necessary to rule out a pseudorelapse of optic neuritis if visual acuity is reduced compared to baseline.⁶

In contrast to MS, a progressive phase is not believed to be associated with NMOSD.⁷ Instead, accrual of disability occurs with each relapse. The majority of patients with NMOSD do not return to baseline following an untreated relapse, making it especially important that patients receive adequate acute treatment to mitigate the damage.⁸

Currently, there are no medications approved by the FDA for the acute or preventive treatment of NMOSD. However, off-label use of immunotherapies, including rituximab, mycophenolate mofetil, azathioprine, prednisone, methotrexate, tocilizumab, and mitoxantrone, have been studied for relapse prevention.² In addition, there are three ongoing phase III trials investigating eculizumab (C5 complement inhibitor), inebilizumab (CD19 monoclonal antibody), and SA237 (IL6R blocker); results from these studies could potentially widen the landscape of immunotherapy use in NMOSD.²

Less investigation into appropriate acute treatment of new relapses has been conducted, however, leaving clinicians and patients uncertain about how to manage a new inflammatory event. Traditionally, firstline

treatment for acute NMOSD relapses has been the same as for MS relapses—high-dose methylprednisolone. However, due to the severity of NMOSD relapses and the relative lack of response to steroids alone, methylprednisolone is commonly followed by plasma exchange (PLEX).²

Most data to guide clinical decision-making suggest that patients with NMOSD relapses recover better when PLEX is added to steroid treatment. Abboud et al found that 65% of patients who received both PLEX and methylprednisolone recovered to their prerelapse baseline, compared to 35% of those who received methylprednisolone alone.⁹ These findings were supported by a larger retrospective investigation by Kleiter et al, which found improved recovery with treatment escalation in their cohort.⁸ These data support the recommendation to use PLEX as an adjunct therapy in acute relapses—particularly in relapses with severe presentations.

Because diagnosis and treatment of relapses involve many factors, ranging from accrual of disability, long-term immu-

notherapy decisions, and medical costs, diligence in provider decision-making is essential when caring for patients with NMOSD. —MAM **CR**

REFERENCES

1. Wingerchuk DM, Hogancamp WF, O'Brien PC, Weinshenker BG. The clinical course of neuromyelitis optica (Devic's syndrome). *Neurology*. 1999;53(5):1107-1114.
2. Kessler RA, Mealy MA, Levy M. Treatment of neuromyelitis optica spectrum disorder: acute, preventive, and symptomatic. *Curr Treat Options Neurol*. 2016;18(1):2.
3. Popescu BF, Lucchinetti CF. Immunopathology: autoimmune glial diseases and differentiation from multiple sclerosis. *Handb Clin Neurol*. 2016;133:95-106.
4. Jarius S, Ruprecht K, Wildemann B, et al. Contrasting disease patterns in seropositive and seronegative neuromyelitis optica: a multicentre study of 175 patients. *J Neuroinflammation*. 2012;9:14.
5. Orman G, Wang KY, Pekcevik Y, et al. Enhancing brain lesions during acute optic neuritis and/or longitudinally extensive transverse myelitis may portend a higher relapse rate in neuromyelitis optica spectrum disorders. *Am J Neuroradiol*. 2017;38(5):949-953.
6. Kessler RA, Mealy MA, Levy M. Early indicators of relapses vs pseudorelapses in neuromyelitis optica spectrum disorder. *Neurol Neuroimmunol Neuroinflamm*. 2016;3(5):e269.
7. Wingerchuk DM, Pittock SJ, Lucchinetti CF, et al. A secondary progressive clinical course is uncommon in neuromyelitis optica. *Neurology*. 2007;68(8):603-605.
8. Kleiter I, Gahlen A, Borisow N, et al. Neuromyelitis optica: evaluation of 871 attacks and 1,153 treatment courses. *Ann Neurol*. 2016;79(2):206-216.
9. Abboud H, Petrak A, Mealy M, et al. Treatment of acute relapses in neuromyelitis optica: steroids alone versus steroids plus plasma exchange. *Mult Scler*. 2016;22(2):185-192.