Overactive Bladder Medications in Geriatrics—Risks and Realities

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KEYWORDS

• Overactive bladder • Anticholinergic • Beta-3 agonist • Dementia • Geriatric • Falls

KEY POINTS

- Anticholinergic medications have been associated with increased risks of dementia, falls and fractures, and all-cause mortality. Beta-3 agonists have not been associated with these adverse events.
- Anticholinergics vary in blood-brain barrier penetration and receptor affinity. As a result, different formulations have different side effect profiles.
- Beta-3 agonists should be used preferentially in the management of geriatric overactive bladder patients.

BACKGROUND

Overactive bladder (OAB) is a common condition with overall prevalence believed to be as high as 27% in men and 43% in women.[1] When further stratified according to patient age, prevalence is even higher among older patients with up to 51% of women above the age of 65 endorsing OAB symptoms.[2] Multiple societies have published guidelines in the management of OAB including the American Urological Association (AUA),[1] European Association of Urology (EAU),[3] and the Canadian Urological Association.[4] Common to each society guideline is a general recommendation for treatment progression in a linear format starting with the least potential for harm. In general, this progression begins with behavioral modifications (timed voiding, fluid restriction, avoidance of irritants, bladder retraining, etc), and pelvic floor exercises. Beyond these measures, patients are often treated pharmacologically with either an anticholinergic or beta-3 receptor agonist medication.

Medication therapy has traditionally been thought of as *second-line therapy* due to its less invasive nature when compared to procedural interventions (sacral neuromodulation, intradetrusor onabotulinum toxin, and tibial nerve stimulation). More recently

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Abbreviations	
AUA	American Urological Association
OAB	overactive bladder

there has been a shift from this line of thinking in favor of a more individualized approach to management. For example, the most recent AUA guidelines published in 2024 eliminated the traditional *step therapy* approach in favor of a shared decision-making approach. There are many factors influencing this shift including a recent preference to decrease prescribing of anticholinergic medication due to concerns for adverse events. Among the elderly population these adverse events are potentiated by decreased rates of drug metabolism, changes in the blood-brain-barrier, and polypharmacy increasing the risk for drug-to-drug interactions and/or increased overall anticholinergic burden.[5,6] Beta-3 agonists (mirabegron and vibegron) are alternative medications for OAB with a lesser side effect profile; however, limitations with insurance coverage and cost often hinder regular use of these medications.[7] The purpose of this article is to highlight the risks and benefits associated with prescription OAB medication use in the elderly population.

RISKS ASSOCIATED WITH OVERACTIVE BLADDER MEDICATION

Since oxybutynin was first approved in 1975, anticholinergics have been a mainstay of OAB treatment.[8] These medications have all displayed efficacy in clinical trials with regard to return to continence and reduction of daily incontinence episodes. Furthermore, all of the anticholinergics used for this indication have similar efficacy. The major difference lies in the tolerability of the various medications.[9] These medications exert their effect by blocking the muscarinic receptors in the bladder leading to a reduction in detrusor contraction. In all likelihood they also act to decrease abnormal urgency sensation as well by blocking cholinergic receptors involved in afferent signaling from the bladder. Adverse effects resulting from blockade of muscarinic receptors present elsewhere throughout the body and include dry mouth, dry eyes, and constipation as well as cognitive issues possibly including dementia.[7] Dry mouth represents the most frequently encountered adverse event with the most recent Cochrane review identifying a relative risk of 3.50 compared to placebo.[10] Despite being the most common side effect, dry mouth is less frequently associated with treatment discontinuation. A study by Akino and colleagues identified constipation as the most bothersome side effect in the general OAB population.[11] This bother in the general population is not generalizable to the elderly population. A study by Decalf and colleagues identified that among the elderly population, the highest level of concern was associated with the potential loss of cognitive function.[12] Given the differences in drug metabolism, rates of polypharmacy, and rates of adverse events and patient preferences with regard to adverse events, it becomes apparent that treating the elderly patient with OAB requires careful deliberation.

COGNITIVE IMPAIRMENT AND DEMENTIA

Acetylcholine is well known as the primary neurotransmitter involved in pathways relative to cognition and memory. Indeed, acetylcholinesterase inhibitors are primary medications used to improve cognition in a variety of conditions.[13] As stated previously, anticholinergic medications exert their effect by blocking acetylcholine from binding to its receptor. Relevant to the treatment of OAB are the M2 and M3 receptors

within the urinary bladder. Within the nervous system M1 to M5 receptors are all present but of particular concern to memory, learning, and attention are the M1 (primarily), M2, and M4 receptors in the hippocampus and neurocortices.[14] Blockade of these receptors with anticholinergic drug use has proven to not only impact memory but also structurally alter the brain. A 2016 study by Risacher and colleagues demonstrated impaired performance on memory testing as well as increased brain atrophy among patients receiving anticholinergic compared to those not receiving an anticholinergic. Furthermore, this study identified a hazard ratio of 2.47 for development of major cognitive impairment in the anticholinergic population.[15] Patients in this study were required to be on medication with medium to high anticholinergic action, which included (but was not limited to) the commonly used OAB medications.

Since the publication of the 2016 study highlighted above multiple large observational studies also identified an increased risk of dementia associated with anticholinergic use. Coupland and colleagues evaluated patients 55 and older and found an overall odds ratio for dementia of 1.49 for those with highest anticholinergic exposure. They also evaluated specifically for patients receiving an anticholinergic prescription for OAB with an odds ratio of 1.65 for dementia.[16] Richardson and colleagues identified an odds ratio of 1.11 for all anticholinergics and again found a stronger association with medications used for urinary conditions.[17] Finally, a 2020 meta-analysis found that any anticholinergic use was associated with dementia with an odds ratio of 1.20; however, based on the data available at this time no causal link was identified.[18]

The short-term cognitive effects had been established prior with oxybutynin, which demonstrated impairment in memory, attention, reaction time, and sleep quality.[19,20] These trials demonstrated negative cognitive effects associated with short-term oxybutynin use that while significant, appeared to be reversible in the short term. Furthermore, the negative effects demonstrated in the oxybutynin patients were not noted in patients receiving alternative medications. For example, in the study by Kay and colleagues oxybutynin was found to cause memory impairment comparable to that seen with 10 years of normal aging, but there was no appreciable effect on memory in the comparison group who received darifenacin.[19] Likewise, sleep disruptions were noted on electroencephalography among patients treated with oxybutynin, but no appreciable changes were identified in the groups receiving trospium or tolterodine.[20] More recently, patients older than 65 years were given cognitive testing before and at week 12 of mirabegron therapy with no difference noted in cognitive function.[21]

These discrepancies between the apparent long-term effects of anticholinergics in observational studies compared to a lack of short-term effects (with the exception of oxybutynin) raised concern that the association may be a result of protopathic bias rather than true cause and effect. Studies have since aimed to address these limitations. For example, Welk and McArthur in 2020 performed a large retrospective matched-cohort study using Canadian health records. They evaluated around 47,000 patients who received anticholinergic medication for OAB and around 23,000 matched patients who received a beta-3 agonist. They identified a significantly increased risk of dementia in the anticholinergic group with a hazard ratio of 1.23. The most commonly used anticholinergic in this cohort was tolterodine in 40% of the patients.[22] A similar study of the Canadian database again identified increased risk of dementia with anticholinergic use compared to mirabegron. Interestingly, within this population solifenacin, darifenacin, tolterodine, and fesoterodine were associated with increased dementia but oxytbutynin and trospium were not. The authors attributed these discrepancies among the anticholinergics to protopathic bias. A study of the French national database identified an increased risk of dementia with oxybutynin and solifenacin but not with trospium.[23]

As evidenced by the studies outlined previously, all OAB anticholinergics are not created equal in terms of association with cognitive impairment and dementia. Based on the different distribution of muscarinic receptors throughout the body it follows that anticholinergics with affinity for muscarinic receptors within the central nervous system pose a higher risk for cognitive impairment. For example, darifenacin has high selectivity for the M3 receptor, which is highly concentrated in the urinary system. Recall the study by Kay and colleagues, which identified short-term cognitive decline in patients receiving oxybutynin but not those receiving darifenacin.[19] Additionally, pharmacologic properties of the different medications that facilitate entry and accumulation in the central nervous system are also more likely to be implicated in raising the risk of cognitive decline. Trospium is a large anionic compound, which in theory inhibits its ability to cross the blood-brain-barrier. In fact, a study by Staskin and colleagues found that trospium remained undetectable in cerebrospinal fluid samples despite steady-state plasma levels. Additionally, there was no demonstrable change in learning or recall within this group of patients receiving trospium.[24] A summary of various properties of common OAB medications can be found in **Table 1** with short-term adverse effects highlighted in **Table 2**.

FALLS, FRACTURES, AND ALL-CAUSE MORTALITY

While the association of anticholinergic use with cognitive decline and dementia has gained a lot of attention in recent years, it is important to note that anticholinergic use has been associated with other dangers in the elderly population as well. For example, falls and fractures have been associated with anticholinergic use in older adults.[25,26] OAB has also been identified as a risk factor for falls and fractures in elderly patients. OAB patients were noted to have an increased risk of any fall in the range of

Table 1
Illustrates pharamcologic properties of common overactive bladder medications including their receptor affinity and propensity to accumulate in the central nervous system

Drug Name	Specificity for Muscarinic Receptors	Accumulation in the CNS
Oxybutynin	Nonselective	High propensity to cross BBB
Tolterodine	Nonselective	Moderate propensity to cross BBB (less than oxybutynin, more than others below)
Fesoterodine	Nonselective	Low propensity to cross BBB
Solifenacin	Selective for M3 > M1, M2, M4, M5	Low propensity to cross BBB but can accumulate due to lack of efflux
Darifenacin	Selective for M3 > M1, M2, M4, M5	Low propensity to cross BBB and is substrate for efflux pump limiting accumulation
Trospium	Nonselective	Low propensity to cross BBB. Not detectable in CSF at steady-state plasma level
Mirabegron	Beta-3 receptor agonist	Not associated with cognitive decline
Vibegron	Beta-3 receptor agonist	Not associated with cognitive decline

Abbreviations: BBB, blood brain barrier; CNS, central nervous system; csf, cerebrospinal fluid.

Table 2
Highlights specific adverse effects of common overactive bladder medications

Oxybutynin	Memory impairment comparable to 10 y of aging with short-term use[19] Impairment of sleep quality characterized by electroencephalography changes with short-term use[20] Associated with increased fall risk[28]
Darifenacin	No memory impairment with short-term use[19] No significant change in heart rate[36]
Trospium	No memory impairment with short-term use[24] No impairment of sleep quality[20]
Tolterodine	No impairment of sleep quality[20] Noted to increase heart rate and decrease heart rate variability[36]
Mirabegron	No memory impairment with short-term use[21] No increased risk of falls[28] No significant change in heart rate. Can increase systolic blood pressure in a subset of patients[37]

1.3 - 2.3 and a 40% to 60% increased risk of recurrent or serious fall irrespective of treatment.[27] Similar to the initial data identifying correlation with dementia and cognitive decline, the early data regarding falls and fractures did not support causation. A 2022 study by Welk and colleagues identified that users of oxybutynin conferred an increased risk of falls compared to mirabegron use. These findings did not hold true for newer anticholinergic medications, however.[28] A retrospective study of patients in the United States identified that overall anticholinergic burden may be an important measure for stratifying a patient's fall risk. This study identified that low anticholinergic burden presented a hazard ratio for falls and fractures of 1.2 compared to 1.4 for those with high anticholinergic burden.[29] Furthermore, a large observational study concluded that higher levels of anticholinergic burden were associated with higher risk of falls and fractures.[30]

All-cause mortality has also been linked to anticholinergic use. Given the association with falls and fractures, which contribute to morbidity and mortality in older adults, this seems intuitive. However, when looking specifically at treatment targeting OAB the most likely causal mechanism of death is cardiovascular.[31] A large noninterventional multicenter study identified that users of OAB anticholinergics were at increased risk of mortality when compared to users of mirabegron.[32] This study did not specifically look at the frequency of use for each formulation of anticholinergic. Prior observational studies have evaluated specific anticholinergic medications. These studies identified increased mortality risk among users of oxybutynin (compared to tolterodine and solifenacin) and tolterodine (compared to solifenacin).[33,34] An additional study identified an increased risk with oxybutynin and tolterodine compared to other anticholinergics.[35]

Regarding cardiac mortality, it is believed that the anticholinergics have the potential to increase heart rate via blockade of the M2 receptors within the cardiac system. Indeed, tolterodine (nonselective) was compared to darifenacin (M3 selective) in a trial, which demonstrated increased heart rate and decreased heart rate variability with tolterodine compared to darifenacin.[36] In contrast, there was initial concern that use of beta-3 agonists could potentiate adverse cardiac events. Mirabegron was not found to increase heart rate significantly in young or old patients, although a subset of patients will experience an increase in systolic blood pressure.[37] Furthermore, mirabegron has been studied as a potential therapy for patients with terminal heart failure as it was noted to increase contractility and decrease pulmonary vascular resistance, further lessening surrounding concerns for adverse cardiac events.[38]

GUIDANCE

As outlined above, the risks of OAB anticholinergic medications range from the commonly discussed side effects of dry mouth, dry eyes, and constipation to more serious and potentially life-threatening adverse events. In contrast, the beta-3 agonist mirabegron has been noted to cause increases in blood pressure in a subset of patients with a higher frequency in the elderly.[37] The newer beta-3 agonist, vibegron, has not been associated with this risk. Taking these findings into account, the seemingly obvious solution would be to avoid OAB anticholinergics in favor of beta-3 agonists exclusively. Unfortunately, in clinical practice this is often not practical due to persistent high costs of the beta-3 agonists. Furthermore, it appears that while anticholinergics as a whole are associated with a host of adverse events, they are not all created equal. In response to these concerns there has been guidance from several societies in the management of elderly OAB patients.

In 2017 (and updated in 2021) the American Urogynecological Society issued a statement recommending that anticholinergics be avoided in women older than 70 years and in situations where they are used, they recommend usimg agents that do not cross the blood-brain-barrier and using the lowest possible dose. They also recommend using beta-3 agonists when able.[39] The Society of Urodynamics, Female Pelvic Medicine, and Urogenital Reconstruction echoed many of these statements in a 2022 white paper. In addition, they recommend considering advancing to therapies beyond medications sooner in the treatment approach.[40] The most recent EAU Guidelines for female lower urinary tract symptoms state that both OAB anticholinergics and beta-3 agonists are effective treatment options for elderly women. They do however recommend regular monitoring of cognitive function in those patients treated with an anticholinergic with long-term exposure posing the highest risk for cognitive decline. They specifically mention the association of oxybutynin with cognitive decline while citing that there is no association with cognitive decline for darifenacin, fesoterodine, solifenacin, and trospium.[3] Finally, the AUA in 2024 updated their guidance to eliminate step-wise therapy in OAB treatment and include a statement regarding the association of anticholinergics with the development of dementia.[1]

OAB is a common diagnosis and OAB medications commonly contribute to a patient's anticholinergic exposure. Recent evidence suggests cumulative anticholinergic burden is a modifiable risk factor for the development of cognitive impairment. Prescribers of anticholinergic medications for any indication should be aware of a patient's cumulative anticholinergic burden and should aim to use alternatives and otherwise use anticholinergics for the shortest duration possible.[41]

SUMMARY

OAB symptoms are common in the elderly population and elderly patients prioritize mitigating the risk of cognitive decline over other potential side effects. Beta-3 agonists should be used preferentially over anticholinergics due to their favorable side effect profile and equal efficacy. When beta-3 agonist use is not an option, preferential use of anticholinergics with a favorable neuropharmacological profile should be used for the shortest term possible.

CLINICS CARE POINTS

- Overactive bladder (OAB) is a common condition. Prevalence increases with age and over half of women over the age of 65 endorse OAB symptoms.

- Among the general OAB population, constipation has been found to be the most bothersome side effect of medical therapy. Elderly patients with OAB, however, state that the adverse effect that causes the greatest concern is the potential loss of cognitive function.
- Anticholinergic medications have been associated with cognitive decline and dementia, falls and fractures, and all-cause mortality. These associations do not hold true for beta-3 agonists.
- Different OAB anticholinergic medications have different pharmacologic profiles. As a result, the potential for adverse effects differs from medication to medication.
- Cumulative anticholinergic burden has been positively correlated with risk of cognitive decline. Minimizing duration of therapy should be prioritized when using these medications.

DISCLOSURE

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