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Lurasidone-Induced Urinary Retention: Options?

To the Editor: The report by Varghese and colleagues¹ provides further documentation (the second case report of its kind) that lurasidone appears to be implicated in drug-induced urinary retention. This is not surprising, since many psychotropics, including other atypical antipsychotics, also share this adverse event (AE).^{2–4} This observation by the authors¹ should sensitize clinicians to evaluate all patients who are prescribed lurasidone for this AE. As they state,¹ males are more vulnerable to this AE than females, but particular care should be taken for patients with a history of urinary obstruction or an enlarged prostate.^{2,3} Thus, elderly males may be particularly at risk, although females are still subject to this AE, as was the case in the patient presented by Varghese and colleagues.¹ In general, this AE rarely occurs and is mostly of mild severity.^{2–4} Occasionally, urinary retention can suddenly worsen, requiring a medical intervention to prevent kidney or bladder damage.^{5,6} Thus, urinary retention demands careful clinical attention. As demonstrated by the authors¹ and similarly presented by Asnis and colleagues,² this AE usually appears within days of a dose escalation and quickly dissipates upon dose reduction or discontinuation, thus being reversible.

One critical clinical issue to consider is what options are available to deal with a patient who develops lurasidone-induced urinary retention but also has had a significant clinical response, as occurred in the patient presented by Varghese and colleagues.¹ One option is to lower the dose of lurasidone, since psychotropic-induced urinary retention appears to be dose dependent.^{1,2} As demonstrated by Varghese and colleagues,¹ lowering the dose led to a full remission of the urinary retention problem, but, unfortunately, the lowered dose was inadequate to sustain the patient's clinical response. In their presentation, the authors¹ failed to explore other treatment strategies that might have allowed the lurasidone dose to be continued as is and thus not jeopardize the loss of the clinical response. One alternative strategy might be to continue drug treatment (eg, lurasidone) but add an α -1A-antagonist like tamsulosin 0.4 mg/d (given orally) to treat urinary retention. Since α -1 stimulation causes constriction of the urethral internal sphincter as well as the detrusor muscle of the bladder and smooth muscle of the prostate resulting in reduced micturition,³ antagonism of the α -1 receptor (one of the pharmacologic effects of tamsulosin) should relieve urinary retention. Tamsulosin is approved by the US Food and Drug Administration for urinary flow problems secondary to benign prostatic hypertrophy.⁷ Off

label, tamsulosin has been successfully used in levomilnacipran and other antidepressant-induced urinary retention cases with response occurring within hours and without inducing any additional side effects. Furthermore, the response was sustained for the duration of treatment (approximately 10 weeks). Although there have been no reports of the efficacy of tamsulosin in antipsychotic-induced urinary retention, it may be worth considering when a patient is having a good clinical response to antipsychotic treatment like lurasidone, particularly when alternative treatments for the patient's diagnosis (eg, bipolar depression) are limited, as was seen in the case presented by the Varghese and colleagues.¹

Dr Varghese and colleagues were shown this letter and declined to comment.

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