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## Venlafaxine induced urinary incontinence

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Venlafaxine is a serotonin-norepinephrine reuptake inhibitor (SNRI), antidepressant approved by Food and Drug Administration for the treatment of major depression, generalized anxiety disorder, social anxiety disorder, and panic disorder. We report a case of reemergence of urinary incontinence in a patient with benign prostate hyperplasia (BPH) after initiating treatment with venlafaxine, who was stabilized on tamsulosin and finasteride for 6 years. To best of our knowledge, there are only three published case reports of venlafaxine induced urinary incontinence (UI) and one possible association of UI with venlafaxine. Mr. M, a 66 year old Caucasian male diagnosed with BPH in 2004. At that time he had urinary frequency, urgency, urinary incontinence, nocturia, hesitancy and dribbling of urine. He was prescribed tamsulosin 0.4 mg QDay and finasteride 5mg OD. He was stabilized on these medications for several years and his urinary symptoms resolved. Patient also has prior history of major depressive disorder for which he was started on venlafaxine 75 mg per day, which was titrated to 225 mg /day over a period of three weeks, for his low mood and anxiety. He developed new onset UI within a week of starting venlafaxine. He described his UI in the form of involuntary leakage of urine both during the day and at night. His past medical history is significant for asbestosis, obstructive sleep apnea, hypertension, coronary artery disease, hyperlipidemia, peripheral neuropathy, arthritis, hiatal hernia, benign prostate hyperplasia and chronic low back pain. He is allergic to sulfa, meperidine and felodipine. His other medications were, citalopram 80mg QAM, venlafaxine 225 mg QAM, buspirone 15mg and mirtazapine 7.5mg QHS; acetaminophen 325mg QID, aspirin 81mg QDay, clonazepam 0.5mg QDay, docusate 100mg BID, furosemide 20mg BID, HCTZ 50mg, triamterene 75mg QDay, gabapentin 300mg TID, hyaluronate Na 10mg, morphine 15mg QID, omeprazole 20mg BID, propranolol 60mg QDay, sennosides 8.6 mg BID, simvastatin 40mg QDay. It was decided to discontinue the venlafaxine following which his UI improved. The temporal relationship of the urinary incontinence to the initiation of venlafaxine and the resolution of his UI with the discontinuation of venlafaxine supports our inference that the UI was induced by venlafaxine. Inghilleri, Maurizio et al studied the effects of venlafaxine on patients with spinal cord lesion. It was hypothesized that venlafaxine acts at the spinal cord level where it modulates the detrusor muscle contraction possibly by 5-HT<sub>1A</sub> receptor activation directly or indirectly on alpha1-adrenoreceptor, causing decrease in detrusor sphincter dysynergia (DSD). Buggard et al also supports this hypothesis that activation of 5-HT<sub>1A</sub> receptor induces detrusor muscle contraction. Another possible mechanism of action for UI is the indirect potentiation of cholinergic neurotransmission in the detrusor muscle of bladder. This is induced by serotonin activation of 5-HT<sub>4</sub> receptors, which further increases the bladder voiding efficiency resulting in urinary incontinence especially in conditions such as BPH, in this patient. His other medications citalopram and clonazepam may precipitate UI. However in this case it does not seem to be a factor as he was maintained on citalopram for 5 years, and his urinary symptoms resolved after stopping the venlafaxine. Another possible explanation is that venlafaxine level would be increased with medications inhibiting CYP2D6 such as citalopram in this patient. Clinicians should be aware of these rare adverse side effects while prescribing venlafaxine.

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