

Case Report

Urinary Incontinence During Sleep Associated With Buspirone: A Case Report



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ABSTRACT

Background: Buspirone is an anxiolytic medication that selectively alleviates anxiety without the anticonvulsant, sedative, or muscle-relaxant effects associated with benzodiazepine. To the best of our knowledge, case reports of nocturnal urinary incontinence in buspirone users have not yet been published.

Case Report: We report a 52-year-old female patient with urinary incontinence during sleep (once or twice a week) associated with buspirone (10 mg/day). The patient's nocturnal enuresis problem disappeared immediately after the discontinuation of buspirone.

Conclusion: The mechanism underlying this effect is known. These medications influence neurotransmitter systems that modulate bladder control, altering micturition patterns. Antidepressants affect central nervous system pathways that regulate bladder function, potentially decreasing inhibitory control and increasing detrusor muscle sensitivity. Reporting rare drug side effects is crucial for enhancing patient safety and improving pharmacovigilance.

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Introduction

Buspirone is an anxiolytic medication with a unique pharmacological profile that distinguishes it from traditional benzodiazepines. Buspirone is categorized as “anxiolytic,” meaning it selectively alleviates anxiety without the anticonvulsant, sedative, or muscle-relaxant effects associated with benzodiazepines [1]. It has been shown to lack the potential for abuse and physical dependence, distinguishing it from traditional anxiolytics [2]. Buspirone acts as a partial agonist at serotonin 5-HT_{1A} receptors and exhibits both agonist and antagonist properties at dopamine receptors [3]. Buspirone enhances dopaminergic activity by inhibiting dopamine autoreceptors, possibly contributing to its anxiolytic effect [4]. Buspirone does not interact with the benzodiazepine receptor complex, differentiating its action from benzodiazepines [5].

Although buspirone is effective in treating anxiety, it is associated with several side effects that warrant consideration. Some patients who used buspirone reported drowsiness, weakness, and faintness, particularly at higher doses (20 mg) [6]. Also, animal studies have indicated potential amnesic effects of high doses of buspirone, impairing performance in tasks related to memory and learning [7]. Buspirone increases sleep latency and decreases total sleep duration, affecting both non-rapid eye movement (non-REM) and rapid eye movement (REM) sleep [8]. Unlike many anxiolytics, buspirone does not exhibit significant sedative properties, making it a preferable option for those concerned about daytime alertness [9].

The effects of buspirone on sleep may indirectly influence urinary patterns, as it has been shown to disrupt sleep stages without sedative effects [10]. This disruption could exacerbate nocturnal incontinence. To our knowledge, no cases of buspirone-induced urinary incontinence have been reported. According to the medical literature, urinary incontinence is listed as infrequent adverse effect of buspirone, occurring in less than 1% of patients; however, detailed case reports describing this association are lacking [11, 12]. In the present study, we discuss the case of a 52-year-old woman who had been treated for depression for more than 26 years. She developed urinary incontinence during sleep after prescribing buspirone.

Case Presentation

We report a 52-year-old married female patient who was admitted to the Psychiatric Clinic with nocturnal enuresis 2 months ago. The patient has been suffering from depression for 26 years. At the time of referral to our clinic, the patient was undergoing antidepressant therapy but reported a gradual decline in mood stability over the preceding weeks, accompanied by reduced motivation, low energy, and diminished pleasure in daily routines. She also described a mild sleep quality and appetite deterioration, though without significant weight change. Her psychiatric history spanned more than two decades, during which several psychiatrists had treated her; however, detailed recollections of her initial symptom profile were limited due to the chronicity of her illness. Given the persistence of these residual depressive symptoms despite stable pharmacotherapy, buspirone 10 mg/day was initiated as an augmentation measure.

She had no history of chronic diseases such as thyroid disease, respiratory disease, hypertension, or diabetes. She had no history of drug or food allergies, urinary tract infection, kidney disease, pelvic surgery, or childhood enuresis. Physical examination findings were normal, and the rest of the clinical examination, including the gynecologic and neurologic examination, revealed no significant findings.

Laboratory evaluations, urinalysis, and renal function tests yielded results within normal limits, effectively excluding urinary tract infection, renal dysfunction, and metabolic disorders. Table 1 presents a summary of the laboratory tests.

At the time of presentation, the patient had a longstanding diagnosis of major depressive disorder in partial remission. She had been maintained for several months on a stable regimen of fluoxetine 10 mg twice daily (total daily dose 20 mg) and pregabalin 100 mg twice daily (total daily dose 200 mg). Approximately two months prior to the onset of symptoms, buspirone 10 mg/day was added (not for the treatment of anxiety symptoms), as part of a structured “step 2” augmentation strategy for partial response to antidepressant therapy, consistent with the approach evaluated in the sequenced treatment alternatives to relieve depression (STAR*D) trial. In that trial, buspirone, acting as a partial agonist at 5HT_{1A} receptors, was one of the recommended augmentation options to enhance selective serotonin reuptake inhibitor (SSRI) efficacy in patients with incomplete remission after initial monotherapy [13].

Table 1. Summary of laboratory test results

	Test	Result	Reference Range (Adult Female)
Urinalysis	Color/Clarity	Yellow /Clear	—
	Specific gravity	1015	1.005-1.03
	pH	6.0	4.5-8
	Glucose	Negative	Negative
	Protein	Negative	Negative
	RBC/WBC	0–2/hpf	0–2/hpf
	Blood urea nitrogen (BUN) (mg/dL)	16	7-20
	Creatinine (mg/dL)	1	0.6-1.1
	Fasting blood glucose (FBG) (mg/dL)	98	70-99
	Hemoglobin A1c (%)	5.4	<5.7
	Sodium (Na) (mmol/L)	140	135-145
	Potassium (K) (mmol/L)	4.2	3.5-5.
Complete blood count	Calcium (Ca) (mg/dL)	9.4	8.5-10.5
	Hemoglobin (Hb) (g/dL)	13.5	12-16
	WBC ($\times 10^9/L$)	6.5	4-11
	Platelets ($\times 10^9/L$)	245	150-400
	ESR (mm/hr)	12	<20
	CRP (mg/L)	<1	<3

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Approximately one week after starting buspirone, the patient developed nocturnal enuresis, occurring once or twice a week. This led her husband to sleep in a separate bed, causing significant embarrassment and emotional distress. She tolerated the situation for approximately one month without suspecting a potential link to the new medication. There were no daytime urinary symptoms, dysuria, hematuria, fever, pelvic pain, neurological deficits, or changes in bowel or fluid intake. At a subsequent psychiatric followup visit, she reported the problem, and buspirone was discontinued. No alternative augmentation agent was initiated, and her maintenance regimen of fluoxetine and pregabalin was continued.

The patient's nocturnal enuresis disappeared immediately after the discontinuation of buspirone. Over the ensuing six months, the nocturnal enuresis did not recur. Causality assessment using the Naranjo adverse drug reaction probability scale produced a total score of

7, indicating a probable association; detailed scoring is provided in [Supplementary Table S1](#).

Discussion

We present a case of nocturnal enuresis associated with buspirone. To the best of our knowledge, case reports of nocturnal urinary incontinence in buspirone users have not been published. Although medical literature reports [11, 12] urinary incontinence as a rare adverse effect (<1%) of buspirone, our report provides detailed clinical observations and a clear temporal relationship supporting this association. Causality assessment using the Naranjo algorithm yielded a score of 7, indicating a probable relationship. Rechallenge was not performed due to ethical considerations and the patient's refusal.

Studies have indicated that the use of SSRIs is associated with a 61% increased risk of urinary incontinence,

particularly in the elderly [14]. The incidence density ratio for urinary incontinence during SSRI exposure is notably high, with sertraline users facing even greater risk [14].

Individual patient characteristics and medical history can influence the incidence of nocturnal urinary incontinence as a side effect of antidepressants. The medical literature indicates that genetic factors, comorbid conditions, and demographic variables significantly predicts nocturnal urinary incontinence [15, 16]. Studies show that the female gender is associated with a higher incidence of urinary incontinence when treated with certain medications [17]. Age-related factors also contribute; elderly patients often experience higher rates of urinary incontinence due to a combination of medication effects and age-related physiological changes [18].

Antidepressants, particularly serotonin and norepinephrine reuptake inhibitors, such as duloxetine, can lead to nocturnal urinary incontinence through several neurophysiological mechanisms. These medications influence neurotransmitter systems that modulate bladder control, altering micturition patterns. Duloxetine enhances the activity of pudendal motor neurons, increasing urethral sphincter contractility, which can paradoxically lead to incontinence in some patients [19, 20]. Also, antidepressants affect central nervous system pathways that regulate bladder function, potentially decreasing inhibitory control and increasing detrusor muscle sensitivity [21, 22].

The use of serotonergic antidepressants has been linked to conditions like REM sleep behavior disorder, which may indirectly affect nocturnal urinary control. Adverse effects such as the syndrome of inappropriate antidiuretic hormone are more prevalent with serotonergic agents, potentially leading to increased nocturnal urination [23].

We hypothesize that buspirone-induced modulation of serotonergic and dopaminergic neurotransmission may impair micturition control during sleep. Specifically, partial agonism at presynaptic and postsynaptic 5HT_{1A} receptors may decrease inhibitory serotonergic tone on the pontine micturition center, leading to increased detrusor activity. Dopaminergic effects may further influence bladder reflexes. Moreover, buspirone's documented alterations in sleep architecture could diminish arousal thresholds, preventing awakening before bladder emptying. These factors can synergistically promote nocturnal urinary incontinence in predisposed patients.

While these medications can alleviate anxiety and improve quality of life, they may also influence urinary function, especially in individuals with serotonergic dysregulation. Reporting rare drug side effects is crucial for enhancing patient safety and improving pharmacovigilance. The lessons learned from case reports highlight the importance of effective monitoring, and data analysis in understanding the adverse effects of medications.

Ethical Considerations

Compliance with ethical guidelines

This study was designed based on the ethical principles of the Declaration of Helsinki (2008) for medical studies involving humans. This study was approved by the Ethics Committee of [Mazandaran University of Medical Sciences](#), Sari, Iran (Code: IR.MAZUMS.REC.1403.260). Informed consent was obtained from the patient before publication.

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Authors' contributions

Study design and project administration: Seyed Hamzeh Hosseini; Data collection: Raheleh Rafeiee; Data interpretation: Hamed Ghazvini, and Seyedeh Masoumeh Seyedhosseini Tamijani; Writing: Raheleh Rafeiee; Hamed Ghazvini, and Seyedeh Masoumeh Seyedhosseini Tamijani; Final approval: All authors.

Conflict of interest

The authors declared no conflict of interest.

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Supplementary Table S1. Naranjo Adverse Drug Reaction Probability Scale Assessment

Question	Answer	Score	Comment/Justification
1. Are there previous conclusive reports on this reaction?	Yes	+1	Similar urinary adverse effects reported with other serotonergic agents; no prior buspirone-specific cases found.
2. Did the adverse event occur after the suspected drug was administered?	Yes	+2	Nocturnal enuresis developed approximately one week after starting Buspirone (10 mg/day).
3. Did the adverse reactions improve after discontinuation of the drug?	Yes	+1	The symptom resolved immediately after withdrawal of Buspirone.
4. Did the adverse reaction reappear when the drug was readministered?	Do not know	0	Re challenge not performed due to ethical concerns and patient refusal.
5. Are there alternative causes (other than the drug) that could have caused this reaction?	No	+2	Comprehensive diagnostic work-up (laboratory and clinical) excluded other causes.
6. Did the reaction reappear when a placebo was administered?	Do not know	0	Not tested.
7. Was the drug detected in the blood (or other fluids) in concentrations known to be toxic?	No	0	Serum levels not measured; dose was within therapeutic range.
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	Do not know	0	Only one dose level was used throughout treatment.
9. Did the patient have a similar reaction to the same or similar drug after any previous exposure?	No	0	No history of similar adverse effects.
10. Was the adverse event confirmed by objective evidence?	Yes	+1	Detailed patient history and corroboration by family members are documented in the clinical records.
Total Score		7	Probable association according to Naranjo scale classification.