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CASE REPORT

Double Incontinence as Adverse Effect During Brief Usage of Clonazepam

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ABSTRACT

Clonazepam, a benzodiazepine used for treating seizures, anxiety disorders, and movement disorders, is known for its central nervous system depressant side effects, such as drowsiness and urinary incontinence. However, double incontinence is rarely reported. We present the case of a 41-year-old woman with multilevel degenerative disc disease and right gastrocnemius-soleus muscle spasticity secondary to an Achilles tendon rupture, who developed who experienced dual incontinence after brief clonazepam use. Within three days of commencing clonazepam, she experienced nausea, fatigue, drowsiness, loose stools, and urinary frequency, progressing to urinary and faecal incontinence. Investigations, including blood tests, urine analysis, and abdominal imaging, were unremarkable, ruling out differential diagnoses such as infections, spinal pathology, and metabolic disorders. The patient responded positively to the antidote and supportive medical care, with total symptom relief two days after cessation of the medication. We discussed the causes of double incontinence, potential drug-induced incontinence, and the possible mechanisms by which clonazepam could cause double incontinence, given the limited evidence on it. Clonazepam's sedative effects can lead to decreased muscle tone and coordination, potentially resulting in incontinence. Additionally, its impact on GABAergic pathways and anticholinergic effects could influence bladder and bowel function indirectly, but this

is just a theory, and actual evidence is difficult to find. underscores the need for clinicians to recognize double incontinence as a potential adverse effect of clonazepam, even with short-term use, and to consider dose adjustments or alternative therapies when indicated. Further research into the mechanisms of clonazepam-induced double incontinence is warranted given the limited existing evidence.

INTRODUCTION

Clonazepam is a benzodiazepine commonly prescribed for its anticonvulsant and anxiolytic properties. Emerging evidence supports its efficacy in managing myoclonus, particularly spinal segmental myoclonus(Edinoff et al., 2021). Primary adverse reactions include drowsiness, fatigue, sedation, and motor impairment(Lappasetal.,2023).Lesscommonly, it may cause blurred vision, psychomotor agitation, confusion, and irritability(Lappas et al., 2023). While some medications, especially antipsychotics (clozapine, olanzapine, risperidone, olanzapine and aripripazole) have been systematically reviewed as causing double incontinence, clonazepam has been underrecognized (Arasteh et al., 2021a). Studies in frail older adults indicate a 45% increased risk of urinary incontinence among benzodiazepine users, but evidence in middle-aged adults remains limited (Landi et al., 2002) The benzodiazepine class has frequently been associated with urinary incontinence; however, only occasional reports of urinary incontinence specifically implicating clonazepam (Landi et al., 2002; Tsakiris et al., 2008). Aside from an isolated report of urinary incontinence linked to clonazepam in 1979, only a single case report describing bowel incontinence related to lorazepam was published in 2018 (Güngören et al., 2018; Williams & Gillespie, 1979). (Williams & Gillespie, 1979) To date, no documented case has reported concurrent bowel and urinary incontinence associated with benzodiazepine use-particularly clonazepam.. Herein, we present what is, to our knowledge, the first reported case of simultaneous bowel and bladder incontinence following short-term clonazepam use, with complete resolution upon discontinuation of the medication.

CASE PRESENTATION

A 41-year-old female with underlying hypertension, diagnosed in 2016 and currently on Tab Losartan 50 mg once daily, was referred to the rehabilitation clinic for persistent right calf spasm. She also has multilevel degenerative disc disease, diagnosed in 2018, specifically affecting L4/L5 and L5/S1 with right exiting nerve root involvement at L4, diagnosed in 2019. Additionally, she sustained a left-sided Achilles tendon rupture of the right lower limb in 2017 after trauma from a falling fire extinguisher. The Achilles tendon rupture responded well to conservative treatment, but over a year, she developed progressively worsening right calf spasms. The gastrocnemius-soleus muscle is likely due to muscle compensation and altered biomechanics during the healing process. Despite multiple treatments with analgesics, including 300 mg of gabapentin twice daily and 200 mg of celecoxib as needed for the past six months, her symptoms persist.

Therefore, we initiated a nightly regimen of 0.5 mg clonazepam before bedtime during the clinic visit, aiming to alleviate the ongoing right calf spasms. A day after taking her first dose of clonazepam, she started to feel nausea, fatigue, loss of appetite, and drowsiness. She also had two episodes of loose stools and an increase in urinary frequency, urinating almost every one to two hours, with three episodes of nocturia Despite these symptoms, she continued to take her second dose of clonazepam on the following night. The next day, she had reduced consciousness and could not recall the number of urinary frequency, and the frequency of stool passed. Her caregiver had to put her in diapers due urinary and bowel incontinence. There were no dietary changes, sick contact, or history of

taking supplements that could have explained the incontinence. No history of trauma or neurological deficit prior to the symptoms.

Upon arrival at the emergency department, she was drowsy and mildly dehydrated. However, her vital signs were within the normal range. Her Glasgow Coma Scale (GCS) score was 12/15, with a score of 3 for eye-opening response (opens eyes to verbal command), 4 for verbal response (confused speech but able to communicate), and 5 for motor response (localized pain but reduced overall motor function). The abdominal examination revealed a soft and non-tender abdomen, with hyperactive bowel sounds. A full neurological examination was not performed as the patient was drowsy and fatigued. A per rectal examination showed an empty rectum and intact anal sphincter.

Blood samples were sent for a complete blood count, renal and liver function, random glucose, and electrolyte analysis. A urine sample was sent for culture, and an abdominal x-ray and ultrasound were done. All these investigation results were normal. At this point, an adverse drug reaction to clonazepam was suspected, and hence, clonazepam was stopped. A single 0.2 mg intravenous (IV) dose of flumazenil was administered, resulting in noticeable improvement in drowsiness within minutes. She was closely monitored with IV fluids, stool, and fluid charting. On the first day of admission, her loose stools reduced to two episodes, and by the following day, both bowel and urinary incontinence had completely resolved. With no recurrence of symptoms, she remained hemodynamically stable and was discharged in good condition.

During subsequent follow-up at the rehabilitation clinic, the patient then received an injection of botulinum toxin into her right calf muscles. Each gastrocnemius muscle head received 75 units of Dysport® intramuscular injection. Two weeks post-injection, the patient's reassessment revealed complete resolution of pain and spasm, as well as marked improvement in gait and function.

DISCUSSION

Clonazepam is a potent benzodiazepine tranquilizer with a half-life of of 20 hours and takes about 20 to 60 minutes for its onset of action (Ghit et al., 2021; Wu & King, 2024). This intermediate-to-long-acting GABA-A receptor agonist drug was widely endorsed for managing epilepsy and anxiety disorders (Dokkedal-Silva et al., 2019; Edinoff et al., 2022). Researchers also report its effective off-label use for sleep disorders, multi-infarct dementia, tinnitus, depression, withdrawal from other benzodiazepines, neuropathic pain management, myoclonus, and movement disorders (Edinoff et al., 2021; Ghit et al., 2021).

A comprehensive evaluation is essential to rule out all the potential causes of double incontinence. Faecal incontinence can arise from structural abnormalities such as sphincter injury or rectal prolapse, as well as neurological dysfunction including spinal cord injury and diabetic neuropathy (Knowles et al., 2022). It may also result from altered rectal sensitivity, inflammatory conditions, or disrupted colonic motility, reflecting its complex and multifactorial nature (Knowles et al., 2022). Urinary incontinence may result from urinary tract infections, overactive bladder, pelvic floor dysfunction, or neurological impairments affecting bladder control (Dobrek, 2023; Tsakiris et al., 2008).

For double incontinence, concurrent spinal pathology (e.g., cauda equina syndrome) or systemic conditions (e.g., autonomic neuropathy) should be considered. Drug-induced incontinence, though less common, is a critical differential, particularly with medications possessing anticholinergic, sedative, or muscle-relaxant properties, such as antipsychotics, opioids, or benzodiazepines (Dobrek, 2023; Knowles et al., 2022). In this case, the temporal association between

clonazepam initiation and symptom onset, along with the exclusion of structural, infectious, and metabolic causes, strongly supports drug-induced double incontinence as the primary diagnosis. This underscores the importance of medication review in patients presenting with unexplained incontinence.

Recent literature explains that drugs with sedatives and hypnotics mainly cause overflow or functional incontinence (Arasteh et al., 2021b). Contrarily, evidence on drug-induced bowel incontinence is sparse. Few cases reported double incontinence secondary to antipsychotic medications (Hergüner & Mukaddes, 2008; Incecik et al., 2015), but none related to the benzodiazepine group. The tranquilizer of the potent benzodiazepine takes about one to eight hours to reach its peak blood concentration, and its effect lasts around six to twelve hours (Edinoff et al., 2022). Toxicity by clonazepam alone is rarely lethal. Unlike in this case report, severe consequences due to CNS depressants often seen when taken in combination with ethanol, barbiturates, or opioids (Arasteh et al., 2021b; Hieger et al., 2024). Nevertheless, the patient in this case is not utilizing any of the aforementioned combination drugs.

The fundamental mechanism of how clonazepam can induce double (urinary and bowel) incontinence has been poorly documented in literature reviews. However, the potential pharmacokinetic explaination is that it crosses the blood-brain barrier, affecting brain regions involved in motor control and autonomic functions (mainly in the cortex and limbic region), which has an inhibitory neurotransmitter called gamma amino-butyric acid (GABA) (Ghit et al., 2021). The serotonergic effects increase GABA activity and depress the central nervous system (CNS) (Dokkedal-Silva et al., 2019; Lappas et al., 2023). This action leads to increased inhibition in the CNS, which can affect the neural pathways controlling bladder and bowel function.

Additionally, the sedative effects of clonazepam can lead to decreased muscle tone and coordination, potentially resulting in incontinence (Dokkedal-Silva et al., 2019). Inhibition of the limbic system may alter emotional responses to bladder and bowel sensations, contributing to incontinence (Edinoff et al., 2021, 2022). While clonazepam does not directly target α1 receptors, its impact on GABAergic pathways and anticholinergic effects could influence bladder and bowel function indirectly (Arasteh et al., 2021). The synergistic effects of these mechanisms potentially explain the cause of incontinence in this patient.

The fundamental principle in the management of clonazepam toxicity is supportive medical care tailored to the severity of the patient's condition. Flumazenil should not be used routinely in the management of benzodiazepine toxicity(Hieger et al., 2024). Recent evidence has shown that its usage is significantly related to serious adverse events such as seizures and arrhythmias (Hieger et al., 2024).

CONCLUSION

This report emphasizes the risk of clonazepam-induced both bowel and urinary incontinence, especially with short-term use. The patient's symptoms improved through non-pharmacological measures and targeted medications. This inaugural case highlights the urgent need for fundamental research into the mechanisms by which clonazepam may cause dual incontinence. This case underscores the necessity for clinicians to monitor patients closely for rare but significant adverse effects when prescribing clonazepam in their daily practice.

CONFLICT OF INTEREST

All of the authors have no conflict of interest.

CONSENT

Informed consent was obtained from the patient, and a copy of the written consent is submitted to the journal.

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