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Lorazepam-Induced Orthostatic Intolerance and Secondary Headache in a Low Blood Pressure Phenotype

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INTRODUCTION & AIM

Background

Constitutional low blood pressure (BP) phenotypes are frequently encountered in clinical practice but remain poorly characterized in terms of pharmacologic vulnerability. While hypotension in elderly or critically ill populations has been extensively studied, young individuals with lifelong low baseline BP have received comparatively little attention in the literature.

Benzodiazepines, including lorazepam, are widely prescribed for insomnia and anxiety disorders. Their central nervous system depressant effects are well established; however, their influence on autonomic regulation and orthostatic hemodynamics in low BP phenotypes has not been systematically evaluated. Sedative-induced reductions in sympathetic tone may theoretically impair compensatory cardiovascular responses upon standing, especially in individuals with already reduced vascular tone.

Orthostatic intolerance (OI) represents a spectrum of symptoms—lightheadedness, blurred vision, chest discomfort, and headache—that may occur even in the absence of classic orthostatic hypotension (OH) criteria. This distinction is clinically important, as symptom burden may not correlate strictly with blood pressure thresholds.

Clinical Gap

Existing data largely focus on geriatric populations or patients with polypharmacy. There is limited documentation of reproducible, dose-dependent orthostatic phenomena in otherwise healthy young adults with constitutional low BP.

Aim

This case report aims to:

1.Describe a reproducible, dose-dependent pattern of orthostatic intolerance associated with nightly lorazepam use.

2.Characterize the hemodynamic profile (BP vs HR changes) underlying symptom development.

3.Identify potential modulating factors, including anticholinergic burden and hydration status.

4.Highlight implications for prescribing practices in low BP phenotypes.

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METHOD

◆ **Study Design**

Prospective single-patient observational case study with repeated orthostatic measurements across multiple medication conditions, allowing intra-individual comparison and dechallenge–rechallenge analysis.

◆ **Patient Profile**

•30-year-old male
•Lifelong constitutional low BP (~90–95/50–55 mmHg), asymptomatic at baseline
•Chronic insomnia treated intermittently with lorazepam (1–2 mg nightly)
•No history of cardiovascular disease, diabetes, or endocrine disorders

◆ **Baseline Evaluation**

Laboratory and diagnostic workup included:
•Complete blood count (CBC)
•Comprehensive metabolic panel (CMP)
•Thyroid function testing
•Hemoglobin A1c
•Morning cortisol
•Electrocardiogram

All findings were within normal limits, excluding structural, endocrine, or arrhythmic causes of orthostatic symptoms.

◆ **Monitoring Protocol**

Orthostatic measurements were obtained using a standardized approach:
•Supine BP and HR after 5 minutes of rest
•Standing BP and HR at 1 minute and 3 minutes
•Symptom severity graded on a 0–3 scale
•Comparison across:
- 1 mg lorazepam nights
- 2 mg lorazepam nights
- Off-medication nights

Additional factors recorded:
•Total anticholinergic burden (ACB) when co-exposed to imipramine or diphenhydramine
•Urine specific gravity as a proxy for hydration status

RESULTS & DISCUSSION

◆ **[1] Dose-Dependent Orthostatic Pattern**

A consistent pattern emerged:
•2 mg lorazepam nights were associated with significantly higher symptom scores (grade 3), including lightheadedness, chest tightness, and evening headache.
•1 mg nights demonstrated mild symptoms.
•Off-medication nights showed marked symptom improvement.

This dechallenge–rechallenge phenomenon strengthens the causal association between lorazepam dose and orthostatic symptom burden.

◆ **[2] Hemodynamic Characteristics**

The orthostatic response revealed:
•Modest systolic BP reductions (not consistently meeting ≥20 mmHg criteria for classic OH).
•Disproportionately elevated heart rate responses upon standing.
•Strong correlation between HR increase and symptom severity.

These findings are more consistent with **orthostatic intolerance** rather than strict orthostatic hypotension. The case highlights that symptomatology may arise even when conventional OH thresholds are not met.

◆ **[3] Modulating Factors**

Anticholinergic Burden

Episodes of higher total anticholinergic burden coincided with:
•Increased mucosal dryness
•Ocular discomfort
•Amplified orthostatic symptoms

This suggests additive autonomic suppression.

Hydration Status

Urinalysis on high-symptom days demonstrated:
•Elevated urine specific gravity
•Intermittent ketonuria and hyaline casts

These findings indicate relative dehydration, which likely exacerbated reduced preload and autonomic instability.

◆ **Pathophysiologic Considerations**

Several mechanisms may explain the observed phenomenon:
•Central GABAergic suppression reducing sympathetic outflow
•Impaired baroreceptor-mediated compensation
•Reduced baseline vascular tone in constitutional low BP phenotype
•Combined sedative and anticholinergic autonomic inhibition

Even subtle reductions in compensatory sympathetic activation may be clinically significant in individuals with chronically low systemic vascular resistance.

Night	Lorazepam Dose	ΔSBP (3 min)	ΔHR (3 min)	Symptom Score (0–3)	ACB Score
A	1 mg	–3 mmHg	+6 bpm	1 (mild)	3
B	2 mg	–9 mmHg	+18 bpm	3 (severe)	6
C	1 mg (off TCA/AH)	–4 mmHg	+9 bpm	1 (mild)	0
D	2 mg (+ TCA/AH)	–8 mmHg	+20 bpm	3 (severe)	6

Table: Orthostatic Hemodynamics by Lorazepam Dose

CONCLUSION

This case demonstrates reproducible, dose-dependent orthostatic intolerance associated with lorazepam use in a young patient with constitutional low BP.

Key observations include:
•Symptom severity correlated more strongly with heart rate response than absolute BP decline.
•Evening headaches were likely secondary to transient cerebral hypoperfusion.
•Anticholinergic burden and hydration status modulated symptom intensity.

These findings suggest that constitutional low BP may represent a clinically relevant prescribing risk phenotype requiring individualized dosing and monitoring strategies.

FUTURE WORK / REFERENCES

Future research directions include:
•Prospective cohort studies evaluating sedative tolerance in low BP phenotypes
•Tilt-table testing with continuous beat-to-beat hemodynamic monitoring
•Autonomic function profiling in constitutional hypotension
•Development of prescribing guidelines for benzodiazepines in vulnerable autonomic subgroups
•Greater emphasis on non-pharmacologic insomnia interventions (e.g., CBT-I) in this population

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