Abstract Form

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Project Title:	Challenging Dilemma on the Management of Orthostatic Hypotension and Supine Hypertension

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction
Orthostatic hypotension and supine hypertension when presenting together are manifestations of autonomic dysfunction. Neurogenic orthostatic hypotension occurs with impaired synaptic release of norepinephrine and insufficient vasomotor response. Supine hypertension, defined as systolic blood pressure >140 mmHg or a diastolic blood pressure >90 mmHg after five minutes lying supine, may be present in up to 50% of cases of orthostatic hypotension. Supine hypertension occurs due to baroreceptor denervation with norepinephrine deficiency. We present a case of neurogenic orthostatic hypotension and supine hypertension that was managed with specific tailored interventions.

Case Description
37 year-old male with history of type 2 diabetes mellitus with A1c 10.7%, hypertension, and alcohol use disorder presented with anxiety and right hand swelling and pain. The patient also reported dizziness and bilateral feet pain for 4 months prior to admission. Vitals signs were temperature 37 C, HR 103, BP 186/106, SpO2 99% on room air. Physical exam was notable for tongue fasciculations, tremulous hands, and erythema and swelling of right first digit. Patient had persistent episodes of severe hypertension despite treatment for alcohol withdrawal, pain control, antibiotic therapy for right hand cellulitis, and resuming home antihypertensives. It became apparent that patient was experiencing positional hypertensive episodes when lying supine and postural dizziness with systolic and diastolic BP drops of 40-60 mmHg and 20-30 mmHg when standing. Despite large orthostatic fluctuations in BP, the patient's HR remained in 80-90s. Several therapies were trialed in attempt to address orthostatic hypotension, including IV fluids, cold water intake, use of abdominal binder, and escalating doses of fludrocortisone and midodrine but without success. Antihypertensives were dosed for the evening to mitigate supine hypertension but unsuccessful in controlling supine BP. Management was complicated by acute infarct of the right posterior limb of the internal capsule and corona radiata infarct with patient experiencing left-sided numbness and weakness. Given suspicion for autonomic neuropathy, additional diagnostics were obtained resulting with negative HIV, RPR, and Chagas titers, unremarkable SPEP/UPEP, and normal kappa/lambda light chain ratio. Patient's essential hypertension was controlled on nifedipine 30 mg and losartan 100 mg. Transdermal nitroglycerin patch 0.2mg/hr was trialed at night which controlled supine hypertension. Pyridostigmine 60 mg was trialed during the day with marked improvement of postural dizziness and orthostatic blood pressure changes to 20-30/10-20 mmHg. Duloxetine 60 mg was added for peripheral neuropathy. Patient's supine and standing norepinephrine levels resulted low, 62 and 133 pg/mL respectively. Droxidopa was started, which led to resolution of orthostatic hypotension with titrating doses. Patient continued inpatient rehabilitation with physical therapy and was referred for autonomic testing at tertiary care center.

Conclusion
Autonomic neuropathy may manifest as orthostatic hypotension and supine hypertension in patients with risk factors like diabetes mellitus and alcohol use. Together, there is increased risk for end organ damage, cardiovascular events, and all-cause mortality. Early recognition of diurnal and nocturnal discrepancies in blood pressure and heart rate in the inpatient hospital setting may be crucial to determine necessary tailored treatments. Medical therapy targeting synaptic transmission can assist diagnosis and replacement of low catecholamine levels is therapeutic.