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CLINICAL VIGNETTE

Management of Orthostatic Hypotension with Supine Hypertension

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Case Summary

An 88-year-old male with diabetes mellitus complicated by diabetic foot ulcers, orthostatic hypotension with supine hypertension was brought in by family to the Emergency Department (ED) with generalized weakness, dizziness, and cough. The patient had been feeling weaker than usual and per family seemed more lethargic. His past medical history also included mild cognitive impairing BPH and drug use with recent falls. He denied dyspnea but did have a non-productive cough for the last three days. He lived with family and had remote history of tobacco use. His home medications included midodrine 5 mg TID, amlodipine and irbesartan, glargine with sliding scale lispro, aspirin, lovastatin, and finasteride.

In the ED, he had temperature of 38.2 C, blood pressure (BP) 127/56, heart rate (HR) 85/min, and he was hypoxic to 85% on room air, but recovered to 96% on 2L nasal cannula. His physical exam was generally unremarkable with lungs clear to auscultation. Laboratory testing included hemoglobin of 11.8 g/dL, white blood cell count 9.5 K/uL, lactic acid 2.8 mmol/L, procalcitonin 0.20 ng/mL. Chest X-Ray was remarkable for patchy bilateral densities most prominent in the left lung base. His COVID-19 antigen and PCR tests returned positive. He was started on dexamethasone and remdesivir and admitted for COVID-19 pneumonia. The patient was weaned off oxygen without issues and continued to tolerate COVID-19 supportive care.

However, during hospitalization course the patient developed significant worsening of his orthostatic intolerance compared to his baseline. Orthostatic blood pressures showed severe supine hypertension (systolic blood pressure (SBP) > 180 mmHg), with greater than 70 mmHg drop in BP with sitting (SBP 110 mmHg) and over 100 mmHg drop in BP standing (SBP of 60-70 mmHg). Exacerbating factors included the patient's general confinement to bed, acute illness with COVID-19, as well as frequent holding of midodrine and anti-hypertensives based on pre-set hold parameters.

In consultation with cardiology, both non-pharmacologic and pharmacological strategies were used to stabilize the patient. First, being in an isolation room with severely limited mobility and fluid intake, which was treated with intravenous hydration. Second, the head of the bed was elevated to 30 degrees at night to reduce supine hypertension. Third, an abdominal binder and lower extremity compression stockings were utilized to increase venous return.

His home midodrine was continued three times a day, however, the administration times were adjusted earlier to ensure the greatest effect during waking periods and reduce hypertension while sleeping. Long-acting chronic anti-hypertensives were stopped including irbesartan and amlodipine, and replaced with shorter acting nighttime captopril to be given as needed for elevated blood pressure that was measured at 30 degrees head of bed elevation. Collectively, these strategies led to the resolution of his orthostatic intolerance, allowing him to participate in physical therapy and safely discharge home.

Discussion

Orthostatic hypotension is defined as a reduction of systolic BP \geq 20mm Hg or diastolic BP \geq 10 mmHg produced by upright posture that may lead to dizziness or syncope if the cardiovascular system is unable to maintain adequate brain perfusion.¹ Either a reduction in intravascular volume or dysfunctional autonomic reflexes are the pathophysiologic cause behind the phenomenon.

Intravascular volume depletion may occur from an imbalance of fluid intake versus output. Losing a disproportionate amount of fluids can occur with vomiting, diarrhea, hemorrhage, profuse sweating, and iatrogenic diuretic use. Similarly, poor fluid intake due to poor appetite or lack of access can create a volume imbalance that can cause hypovolemia leading to OH. Alcohol and hot temperatures also worsen OH both from dehydration effects as well as peripheral vasodilation.

Autonomic dysfunction can also play a pivotal role in OH pathophysiology. With position change, the baroreceptors trigger the sympathetic nervous system to induce peripheral constriction leading to increased venous return and maintenance of cardiac output. Many different disease processes affect this baroreceptor response including diabetes mellitus, multiple sclerosis, Parkinson's disease, Alzheimer's dementia, and other neurodegenerative diseases.^{1,2} Typical age-related changes can also affect baroreflexes and OH through increased vessel wall stiffness, decreasing cardio-vagal autonomic control, decreased muscle mass and reduced physical activity. It is also important to remember the large capacity splanchnic-mesenteric capillary bed is highly sensitive to arterial and venous baroreflexes and can lead to post-prandial worsening of symptoms.³

Neuropathies affecting the baroreceptor reflex can often be triggered by autoimmune diseases as well. Guillain-Barre Syndrome, Sjogren's, paraneoplastic syndromes, and viral infections generate antibodies that can affect autonomic nervous system function. This has been of particular interest recently with some patients recovering after COVID-19 infections having new onset or significant worsening of prior autonomic disorders such as postural orthostatic tachycardia syndrome orthostatic hypotension.⁴

Finally, medications are a major contributor to onset or worsening of orthostatic hypotension. Diuretics and SGLT2 inhibitors can lead to hypovolemia, and agents such as ACE inhibitors, angiotensin receptor blockers, alpha-1 antagonists, and nitric oxide-mediated vasodilators commonly can precipitate orthostatic hypotension by peripheral vasodilatation effects. Beta-adrenergic and calcium-channel blockers can unmask struggling electrical systems ability to respond to position changes, and drugs that work on the nervous system such as carbidopa-levodopa or anti-depressants can cause central inhibition that leads to OH. While stopping anti-hypertensive medications seems like a strategy that could prevent orthostatic hypotension, the longstanding hypertension that results can further weaken the baroreceptor reflex and exacerbate OH in the future. A meta-analysis of nearly 18,500 patients in nine blood pressure treatment trials over 6 months involving more than 18,000 patients showed intensive blood pressure treatment actually lowered the risk of orthostatic hypotension (odds ratio 0.93, 95% CI 0.86-0.99).⁵

Treatment of OH

Many patients with orthostatic hypotension have complicated treatment due to concurrent supine hypertension, however, leaving both untreated can lead to significant morbidity and mortality.⁶⁻⁷ Untreated orthostatic hypotension can lead to weakness, cognitive difficulties, lightheadedness and syncope with falls and fall related injuries. Conversely untreated supine hypertension can lead to acute cardiovascular complications, increased risk for hemorrhagic stroke and renal failure.⁸ Furthermore, supine hypertension induces nocturnal pressure natriuresis with subsequent volume depletion which can increase fall risk after increased nocturia and exacerbation of morning orthostatic hypotension.

Although large trials of different treatment strategies with hard clinical endpoints do not exist, expert and consensus opinions and results from small studies have created treatment recommendations.⁹ These should be tailored to individual patients' etiology, severity, and comorbidities, and often involve a combination of pharmacologic and non-pharmacologic or lifestyle interventions. The goals of treatment should be to improve orthostatic blood pressure in a way that reduces symptoms and increases the patient's ability to stand and perform activities of daily living without inducing unacceptable supine hypertension.³ A detailed history should include identification of medications and additional medical conditions that can exacerbate OH, oral intake (including alcohol, fluids and sodium

intake), the use of durable medical equipment like hospital beds and walkers, and an assessment of frailty and the ability to exercise or perform activities of daily living.

Nonpharmacological strategies help increase circulating blood volume and include adequate hydration and sodium intake; which should be relatively higher during exercise and in hot weather. Compression stockings, abdominal binders, lower extremity counter maneuvers, and leg elevation to improve venous return. Prolonged bed rest and supine positioning during the day can cause or worsen symptoms, so postural training and increasing aerobic conditioning can improve overall symptoms and quality of life. Severe cases may need to start with activities in the supine position, with gradual progression to recumbent, seated, and finally upright exercises.

Management of the concurrent supine hypertension should also be addressed with elevation of the head at least 4 inches using pillows, wedge, or mechanical bed. A bedtime snack can help induce postprandial hypotension, which can further mitigate supine hypertension.^{3,10}

Pharmacologic options include midodrine, droxidopa, fludrocortisone and pyridostigmine. Midodrine is an oral vasopressor with a short duration of 2-4 hours so it may be dosed multiple times during the day, starting 30-60 minutes before first getting out of bed. Droxidopa is a prodrug converted to norepinephrine and is approved for neurogenic orthostatic hypotension, also dosed three times a day. Since both of these medications can significantly worsen supine hypertension, additional dosing should be avoided after 6 pm. Fludrocortisone expands plasma volume and may be needed if adequate fluid and solute intake cannot be achieved. Finally, pyridostigmine is a cholinesterase inhibitor that has a modest effect on OH without causing supine hypertension.³ For supine hypertension, evening dosing of short acting, non-diuretic and anti-natriuretic hypertensive medications such as clonidine, captopril, hydralazine, or labetalol can be used.

Managing hospitalized patients with orthostatic hypotension and supine hypertension relies on the same strategies. Along with the non-pharmacologic interventions mentioned, early bed mobility and physical therapy is essential to prevent deconditioning that can worsen orthostatic symptoms. If pharmacologic intervention is needed, education for both the patient and hospital staff is essential as both blood pressure raising and lowering agents are often needed at specific times of day.⁷ While guidelines exist, there is no universal treatment plan for patients with both orthostatic hypotension and supine hypertension. Ultimately an individualized approach is necessary as most drugs are not universally effective, symptoms vary and therapy regimens need to be reproducible at home.

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