

Conference

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Title

Acute Orthostatic Hypertension following a Cerebrovascular Accident: A Case Report

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Case Diagnosis

Acute Orthostatic Hypertension following Cerebrovascular Accident (CVA)

Case Description

A 53-year-old male with hypertension and diabetes was admitted to acute inpatient rehabilitation (AIR) following a central midbrain infarct with impulsivity and persistent hypertension. The patient required 24/7 observation due to restlessness and frequent attempts to leave bed. Antihypertensive therapy with lisinopril and nifedipine was initiated. Blood pressure normalized at rest but became markedly elevated with standing and activity, limiting participation in therapy, consistent with orthostatic hypertension (OHT). Propranolol 20mg three times daily was trialed for impulsivity and OHT without clinical improvement. Clonidine 0.1mg twice daily was added for its inhibitory effect on central sympathetic outflow.

Setting

Inpatient cerebrovascular accident and neurorehabilitation ward

Results

The patient's positional blood pressure and impulsivity improved. However, functional performance declined despite being normotensive, raising concern for relative hypotension and decreased cerebral perfusion during therapy. Nifedipine and propranolol were discontinued while lisinopril and clonidine were continued. The patient demonstrated improved therapy participation, blood pressure remained stable, and 24/7 observation was no longer necessary. He was successfully discharged to subacute rehabilitation.

Discussion

OHT is an understudied condition associated with autonomic dysregulation. It is frequently overlooked in acute care settings, likely because patients are frequently bedbound. Physiatrists may encounter OHT more often than other disciplines because rehabilitation requires routine upright positioning and activity. Risk factors for OHT include conditions associated with autonomic dysfunction, including hypertension and diabetes. In this case, CVA likely compounded preexisting autonomic

vulnerability, resulting in OHT. Although no established first-line pharmacologic therapies exist, the addition of clonidine, an alpha-2 agonist, demonstrated significant benefit for this patient.

Conclusions

OHT is an underrecognized cause of hemodynamic instability during AIR. Upright positioning and therapeutic activity may reveal blood pressure abnormalities not apparent at rest. Increased awareness among physiatrists may improve recognition and guide appropriate management in patients with autonomic dysfunction following cerebrovascular accidents.