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Levodopa-induced orthostatic hypotension in parkinsonism: a red flag of autonomic failure

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Introduction: Levodopa (LD) is the main treatment for parkinsonism, but its use may be limited by a potential hypotensive effect.

Objective: To evaluate the cardiovascular and hemodynamic effect of LD in patients with parkinsonism on chronic LD treatment.

Methods: Head-up tilt test were performed before and 60 minutes after 100/25mg LD/dopa-decarboxylase inhibitor (pre-LD vs post-LD HUTT) in 164 patients. Features predictive of LD-induced orthostatic hypotension (OH) were assessed by logistic regression analysis.

Results: Basal supine blood pressure (BP) and heart rate (HR) decreased after LD. During post-LD HUTT, BP drop and HR increase were significantly greater than at pre-LD HUTT. A proportion of 37% of patients had OH at post-LD HUTT compared to 22% of patients presenting OH at pre-LD HUTT ($p<0.001$). Risk factors for LD-induced/worsened OH were pre-LD OH [odds ratio (OR): 36; 95% confidence interval (CI):10-131], absence of overshoot at Valsalva maneuver [OR: 9; CI:4-20] and pathological Valsalva ratio [OR: 6; CI:2-15]. Hemodynamic data showed a steady decrease in HR and left ventricular contractility after LD administration.

Conclusion: The 100/25mg LD/dopa-decarboxylase inhibitor administration caused hypotension in both supine and orthostatic conditions primarily mediated by a cardioinhibitory mechanism. Patients with cardiovascular autonomic failure had a higher risk of developing LD-induced OH. In clinical practice, LD-induced OH could represent a red flag for cardiovascular autonomic failure.