

Levodopa-carbidopa intestinal gel efficacy on freezing of gait and other features of gait: preliminary results from a 6-months prospective study

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Introduction: Levodopa-carbidopa intestinal gel (LCIG) showed some benefit on Freezing of Gait (FoG) and gait difficulties in advanced Parkinson's disease (PD), also in cases refractory to oral dopaminergic therapy [1,2]. As these assessments were primarily conducted by means of expert-delivered rating scales and patient judgment of severity of symptoms, there is a need of implementation by use of objective, observer-independent outcome measures.

Objective: To assess the efficacy of LCIG on FoG and spatiotemporal gait parameters by means of New Freezing of Gait Questionnaire (NFOG -Q), confirmed by technology objective measurement with APDM Mobility Lab™ motion sensors.

Methods: This is an observational open-label study enrolling patients screened as candidates for LCIG therapy, currently including 9 patients with several episodes of FoG a day in the month preceding the baseline evaluation. Assessment by sensors was first performed at baseline, before percutaneous endoscopic gastrojejunostomy implant, in the OFF and best-ON oral antiparkinsonian therapy condition, and repeated 3 and 6 months after starting LCIG therapy, during daily-ON condition.

Results: 5 of 9 patients have currently completed the 6-month follow-up, with a significant improvement at the NFOG-Q (from 15,8±4,2 to 10,6±6,1, p=0,013). The motion sensors confirm the improvement from baseline best-ON to 6 months daily-ON: a trend is valuable at the Two-minute walking test for gait speed and step duration (increase) and double support (reduction), at the Timed up and go test for duration (reduction), at the 360 degrees Turn Test for Turn angle (increase), and at the Sway test for Sway area (reduction).

Conclusion: Preliminary results from motion sensors suggest an improvement of gait features after LCIG start, confirmed by patients' subjective impression. The lack of response at three months could indicate the need for a longer time to induce synaptic plasticity processes within neuronal networks implicated in the genesis of the symptom [3].

References:

[1] Rispoli et al., 2018, Mov Disord Clin Pract 5(5):542-545.

[2] Zibetti et al., 2018, J Neurol Sci. 385:105-108.

[3] Nonnekes et al., 2020, JAMA Neurol. 77(3):287-288.