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Long-Term Subcutaneous Foslevodopa/Foscarbidopa Infusion Restores Oral Intake and Ambulation in Advanced Parkinson's Disease After Deep Brain Stimulation: A Case Report

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Abstract

Several clinical trials have shown that a 24-hour continuous subcutaneous infusion of the foslevodopa/foscarbidopa (LD/CD) hydrate combination reduces "off" time and extends "on" time without causing marked dyskinesia in patients with advanced Parkinson's disease (aPD; Hoehn & Yahr stage V). However, whether this treatment is effective in patients with severe chronic disease progression remains unclear. Moreover, to our knowledge, no studies have addressed the subsequent functional outcomes or described detailed clinical courses specifically in patients who received such formulations of LD/CD after deep brain stimulation (DBS). A 74-year-old woman with aPD (Hoehn & Yahr V) and prior bilateral subthalamic nucleus DBS presented with severe motor fluctuations, akinesia, gait disturbance, and progressive dysphagia requiring percutaneous endoscopic gastrostomy (PEG). Previous pharmacotherapy and DBS adjustments had not improved the severe symptoms. The patient was bedridden and required tube feeding. She was transitioned to home care and subsequently experienced severe "off" periods with life-threatening non-motor symptoms, including systolic hypertension (180-200 mmHg) and severe hypoxia (SpO2 < 40%) from breath-holding spells. Although intermittent subcutaneous apomorphine resolved the acute "off" episodes, concerns over risks of sudden death prompted the initiation of continuous subcutaneous levodopa/carbidopa prodrug (LDp/CDp) infusion to stabilize the levodopa levels. Before the treatment change, the patient received 450mg of oral levodopa, 32mg of transdermal ropinirole (200 mg per day of levodopa equivalent dose [LED]), 100mg of safinamide (150 mg per day of LED), and 25mg of opicapone (225mg per day of LED), yielding a total daily LED of 1,025 mg/day. To minimize outpatient burden, LDp/CDp was initiated at a dose of 0.15 mL/hr (26 mg/hr and 624mg/day of LED) upon admission without a preliminary levodopa challenge test. Daily oral levodopa dose was reduced from 450 to 350 mg, while safinamide was continued at 100 mg/day.

The LDp/CDp rate was adjusted to 0.17 mL/hr (708 mg of daily LED) following the development of transient auditory hallucinations, and total daily LED finally increased from 1,025 to 1,208 mg/day. The non-motor fluctuations and symptoms immediately resolved, including severe respiratory dysfunction. Motor symptoms, orthostatic regulation, and gait also improved, enabling walker-assisted gait during rehabilitation sessions. Remarkably, the oral intake resumed despite long-term PEG dependence. As of the two-year follow-up, ongoing LDp/CDp therapy at a consistent dosage has successfully maintained the patient's improved quality of life. LDp/CDp may be useful for improving the quality of life in select patients with aPD, showing its potential as a therapy for secondary DBS failure.

Categories: Neurology, Internal Medicine, Neurosurgery
Keywords: advanced parkinson's disease, deep brain stimulation, device aided therapy, foslevodopa/foscarbidopa, motor complication

Introduction

Patients with advanced Parkinson's disease (aPD) may experience worsening pre-existing motor and non-motor symptoms. Moreover, the emergence of motor complications such as diurnal fluctuations (wearing-off) and dyskinesia can become clinically problematic. In the course of medication adjustments, patients also experience an increasing pill burden, together with impaired drug absorption and an attenuated symptomatic response associated with gastrointestinal dysmotility [1]. For these challenges, consideration of device-aided therapy (DAT) is recommended, and the "5-2-1" criterion has been proposed as one practical indicator for the initiation of DAT [2].

Additional to deep brain stimulation (DBS), which is a well-established treatment, and continuous subcutaneous apomorphine infusion used as rescue therapy, novel DATs that deliver levodopa replacement via alternative absorption routes have recently become available, including levodopa-carbidopa intestinal

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gel (LCIG) and a 24-hour continuous subcutaneous infusion of the foslevodopa/foscarbidopa hydrate combination (LD/CD) or the prodrug version (LDp/CDp) [3]. These approaches are anticipated to overcome the challenges of impaired absorption and pharmacokinetic variability that arise from differences in drug delivery mechanisms. International multicenter trials have reported a reduction in the "off" time and an extension of the "on" time without marked dyskinesia [1,4,5]. However, these trials were conducted in moderate cases (Hoehn & Yahr (H&Y) stage III or IV) [6], and reports of initiation in severely affected patients such as those at H&Y stage V remain limited. The use of LDp/CDp has been reported in severely affected patients as "off-label rescue" for reversible acute dopaminergic depletion states. However, the baseline severity of these cases was H&Y stage III or IV [7,8]. Most of the research regarding the initiation of LDp/CDp in patients with aPD and a history of DBS has included only subgroup analyses of phase 3 trials and single-center real-world data reports [2,9]. Furthermore, to our knowledge, no studies have specifically addressed the functional recovery of an individual patient.

Therefore, in this report, we describe a case of long-term aPD in which chronic progression led to an inability to consume food or drink orally and significantly limited mobility, including a review of the relevant literature. We initiated LDp/CDp as long-term therapy with the aim of treating non-motor fluctuations, which resulted in the resumption of swallowing function and improved mobility. This report also follows the CAse REport (CARE) guidelines [10].

Case Presentation

A 74-year-old woman with PD underwent bilateral subthalamic nucleus (STN) DBS implication for "wearing-off" and dyskinesia at an outside hospital eight years prior to the current admission. Dysphagia emerged and gradually progressed, necessitating percutaneous endoscopic gastrostomy (PEG) placement. The patient was referred to our hospital for a consultation for LCIG adaptation. Notably, the gastrostomy could not be used exclusively for drug delivery because of its necessary role for nutrition owing to the patient’s dysphagia. Considering the severity of the PD (H&Y stage V), LCIG was considered inappropriate. Despite adjustment of the DBS stimulation current, with the left-sided current increased from 1.5 to 1.7 mA, and levodopa replacement therapy (Table *1*), the patient developed akinesia, severe dysphagia, and gait disturbances. These motor disturbances had shown no improvement, possibly because of insufficient drug delivery. The patient was ultimately transitioned to home-based care.

Item	Pre-LDp/CDp	Post-LDp/CDp
Age at onset (years)	56	56
Age at DBS (years)	68	68
Age at LDp/CDp (years)	74	74
DBS target	Bilateral STN	Bilateral STN
Reason for LDp/CDp (1)	Non-motor fluctuations (respiratory failure)	Non-motor fluctuations (respiratory failure)
Reason for LDp/CDp (2)	Dysphagia	Dysphagia
Reason for LDp/CDp (3)	Gait disturbance	Gait disturbance
UPDRS part III scores (on state)	46	25
UPDRS part IV	12	5
LED (mg)	1025	1208
DBS settings (Lt)	1.5mA, 90μs, 130Hz	1.7mA, 90μs, 130Hz
DBS settings (Rt)	3.0mA, 90μs, 130Hz	3.0mA, 90μs, 130Hz
Oral medications (1)	Levodopa/carbidopa 100/10 mg (1/2 tablet × nine)	Levodopa/carbidopa 100/10 mg (1/2 tablet ×seven)
Oral medications (2)	Ropinirole patch 32 mg	Safinamide 100mg
Oral medications (3)	Safinamide 100mg	None (Not applicable)
Oral medications (4)	Opicapone 25 mg	None (Not applicable)

TABLE 1: Patient Characteristics and Changes in Treatment

Abbreviations: LDp/CDp, foslevodopa/foscarbidopa; LED, levodopa equivalent dose; DBS, deep brain stimulation; Lt, left; Rt, right; STN, subthalamic nucleus; UPDRS, Unified Parkinson's Disease Rating Scale

While in home-based care, apomorphine subcutaneous injection therapy was attempted as a rescue therapy for sudden "off state" manifestations and as additional rescue therapy for dysphagia during the "off" episodes. Although the patient's "on" state could be maintained for a certain period after having rescue therapy, she occasionally exhibited severe hypertension (over 180-200 mmHg systolic). Respiratory dysfunction (< 40% SpO2 accompanied by dyspnea and cyanosis) and breath-holding spells (Video *1*) also occurred as additional non-motor symptoms during the "off" states. Owing to the extent of severe respiratory dysfunction and dyspnea requiring rescue therapy and the risk of sudden death, we determined that a more stabilized serum levodopa concentration and continuous dopaminergic stimulation were required for LD/CD therapy. Thus, we chose the LDp/CDp version as the most appropriate option. Although the rescue therapy had shown some efficacy in the patient, several motor and non-motor symptoms and motor complications had responded only partially to LD/CD. Given the severity of the patient's condition, it remained uncertain whether further clinical improvement and an associated improvement in quality of life (QOL) could be achieved. Generally, a levodopa challenge test is required before introducing LDp/CDp. However, in this case, the LDp/CDp was immediately initiated upon admission to decrease the burden to the patient and family associated with visiting and staying in the outpatient clinic for the testing.

VIDEO 1: Respiratory dysfunction (< 40% SpO2) and breath-holding spells occurred as additional non-motor symptoms during the "off" states.

View video here: https://vimeo.com/1236695130?share=copy&fl=sv&fe=ci

Before the treatment change, the patient received 450mg of oral levodopa, 32mg of transdermal ropinirole (200 mg per day of levodopa equivalent dose [LED]), 100mg of safinamide (150 mg per day of LED), and 25mg of opicapone (225mg per day of LED). The LED contribution of opicapone was calculated as 0.5 times a daily oral levodopa dose, yielding a total daily LED of 1,025 mg/day. LDp/CDp was started at a lower dosage of a

0.15 ml/hr infusion rate (26 mg/hr and 624 mg/day of daily LED) to prevent complications associated with LDp/CDp, such as psychiatric and dermatologic manifestations. Daily oral levodopa dose was reduced from 450 to 350 mg, while safinamide was continued at 100 mg/day. The non-motor fluctuations, particularly in respiratory dysfunction, disappeared immediately after LDp/CDp was initiated. Although oral administration was required, the LDp/CDp dosage was finally maintained at up to 0.17 ml/hr (29.5 mg/hr and 708 mg /day of daily LED; Table *1*) after necessary adjustments for auditory hallucinations, and total daily LED finally increased from 1,025 to 1,208 mg/day. The clinical course and drug adjustments are shown in Figure *1*. The patient showed continued improvement in the non-motor symptom fluctuations, including respiratory dysfunction, and in some of the motor symptoms, achieving an improved QOL. In particular, respiratory dysfunction accompanied by dyspnea and cyanosis did not occur from immediately after initiation of LDp/CDp onward. The previously severe dysphagia had required the long-term administration of continuous tube feeding via the PEG; however, a limited amount of oral intake and spontaneous eating behaviors were noted after LDp/CDp was initiated (Figure *2*, Videos *2*, *3*). Swallowing function and aspiration risk were evaluated under speech-language pathologist assistance and under supervision, and food texture was progressively modified. After flow-rate adjustment, the diet was modified from a soft-textured diet to a minced diet, and ultimately the patient was able to chew and swallow solid food. Hence, food texture adjustment was completed without performing the videofluoroscopic swallowing study that had been planned during hospitalization. The severe hypertension and mobility showed some improvement with this therapy; therefore, the patient was able to begin rehabilitation which included walker ambulation (Figure *3*, Video *4*) during periodic rehabilitation hospitalization periods. As of the two-year follow-up, the patient has continued to undergo LDp/CDp therapy under the same dosage and has maintained the improvements in the QOL.

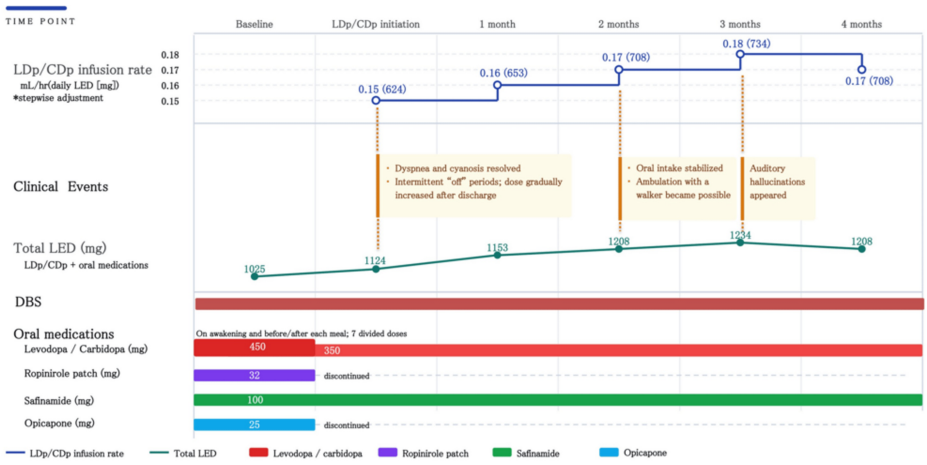


FIGURE 1: Clinical course of the case. Initiation of foslevodopa/foscarbidopa (LDp/CDp) infusion and four-month follow-up. Dyspnea and cyanosis resolved after LDp/CDp initiation. Intermittent "off" periods persisted; therefore, the infusion rate was gradually increased after discharge (0.15–0.18 mL/hr), then reduced to 0.17 mL/hr at four months when auditory hallucinations appeared.

DBS: deep brain stimulation, LED: levodopa equivalent dose, "off", off period. Dashed grey bars indicate a drug that was discontinued at LDp/CDp initiation. Dose labels are in mg (oral agents) or mL/hr (LDp/CDp infusion).



FIGURE 2: Oral intake before (A) and after (B) the initiation of continuous subcutaneous foslevodopa/foscarbidopa prodrug (LDp/CDp) infusion. Before initiation, the patient was unable to orally consume food or drink and was dependent on enteral nutrition via gastrostomy (A); after initiation, oral intake improved and proceeded without adverse effects (B).

VIDEO 2: Oral intake before the initiation of continuous subcutaneous foslevodopa/foscarbidopa prodrug infusion. Before initiation, the patient was unable to orally consume food or drink and was dependent on enteral nutrition via gastrostomy.

View video here: https://vimeo.com/1236697372?share=copy&fl=sv&fe=ci

VIDEO 3: Oral intake after the initiation of continuous subcutaneous foslevodopa/foscarbidopa prodrug infusion. After initiation, oral intake improved and proceeded without adverse effects.

View video here: https://vimeo.com/1236698109?share=copy&fl=sv&fe=ci



FIGURE 3: Gait training with the assistance of a physical therapist and a front-wheeled walker four months after the initiation of continuous subcutaneous foslevodopa/foscarbidopa prodrug (LDp/CDp) infusion. Before initiation, the patient was significantly impaired motorically and bedridden (Hoehn & Yahr stage V).

VIDEO 4: Gait training with the assistance of a physical therapist and a front-wheeled walker four months after the initiation of continuous subcutaneous foslevodopa/foscarbidopa prodrug infusion.

View video here: https://vimeo.com/1236698438?share=copy&fl=sv&fe=ci

Discussion

Herein, we presented a case of aPD and eight-year prior DBS in which long-term subcutaneous foslevodopa/foscarbidopa infusion improved oral intake and mobility. The case highlights the following key points. LDp/CDp was initiated not as 'off-label' rescue therapy for an acute depletion state, but as planned long-term maintenance therapy. This decision was challenged by the severity of the presentation: chronic progression to PEG-dependent dysphagia and a bedridden state years after DBS. However, the planned long-term maintenance therapy in a chronic progressive aPD patient (H&Y stage **V**) was challenged by the development of severe dysphagia requiring tube feeding and the patient's bedridden state due to gait/mobility impairment years after DBS treatment. Second, the LDp/CDp was effective regardless of its extended use, adverse effects, and the high degree of severity of the case (H&Y stage **V**). After the LDp/CDp was initiated, the oral intake improved, and the patient was ultimately able to ambulate with a walker. Furthermore, the non-motor manifestation fluctuations, such as high blood pressure and respiratory failure, stabilized. Laryngeal dysfunction can cause potentially fatal upper airway obstruction in PD [11]. In this patient, severe respiratory events during "off" periods raised concern for life-threatening respiratory

compromise and possible sudden death, prompting the initiation of LDp/CDp. These improvements have been maintained as of the two-year follow-up; therefore, these results are not considered as only short-term effects.

Previous case reports have described LDp/CDp usage as "off-label rescue" for reversible acute dopaminergic depletion states, with symptoms reported such as dysphagia and akinetic crisis associated with aspiration pneumonia or parkinsonism-hyperpyrexia syndrome during perioperative periods [7,8]. The primary goal of rescue therapy using LDp/CDp was established for early transitions back to oral intake. Although this rescue therapy has shown efficacy for moderate PD cases (H&Y stage **III** or **IV**), whether maintenance therapy using LDp/CDp is beneficial for PD patients with chronic clinical courses and/or severe symptoms (H&Y stage **V**) remains unclear. Other reports have described improvements in "freezing" gait or dysphagia (based on videofluoroscopy) after DAT initiation including LCIG and LDp/CDp; however, these cases have also been restricted to moderate PD [12,13].

To our knowledge, no report has described planned long-term initiation of LDp/CDp in a patient severely affected by chronic disease progression (H&Y stage **V**), together with the subsequent functional outcomes. Therefore, this may be the first reported case from this perspective. Based on our experience with this case, a trial of LDp/CDp initiation targeted to improve dysphagia and gait/motor disturbance may be recommended depending on a patient's levodopa responsiveness.

Although the effectiveness of LDp/CDp in patients with such advanced disease remains uncertain, this case suggests that its potential role in addressing dysphagia and gait/motor disturbances warrants further investigation, taking individual levodopa responsiveness into account.

A 52-week, open-label phase 3 trial of244 patients with PD included 24 patients (9.8%) who had received prior DBS and 220 who had not. The results showed that LDp/CDp achieved improvements in "off" and "on" times without significant dyskinesia with or without prior DBS [1]. After approvals were obtained for the formulations for these indications, several reports have described the efficacy of additional therapy with LDp/CDp after DBS using real-world data [9,14]. In contrast, the background in our case was different from that of the recruited patients in these clinical trials, which were targeted to address severe residual aftereffects through the chronic progressive phase of PD. DBS is expected to improve motor complications and is targeted initially as a treatment option in younger patients over LCIG or LDp/CDp therapies; however, its efficacy gradually decreases with disease progression. LCIG or LDp/CDp have been initiated as "secondary DAT" when DBS efficacy weakens and the patients redevelop motor/non-motor symptoms and motor complications [15]. Upon initiation of LCIG or LDp/CDp, re-optimization of stimulation settings has been required in some cases [9]; However, this optimization was not considered necessary in other cases because of the poor responses to DBS. When considering "secondary DAT" initiation, the clinician should be mindful of poor responses to prior pharmacotherapy or DBS adjustments. Notably, the patient in the present case showed no improvement in the motor symptoms and complications after undergoing prior pharmacotherapy and DBS adjustments. In particular, the DBS efficacy was significantly decreased as seen by the patient's symptoms because of the amount of time that had elapsed after the initial DBS. This was considered to be associated with the lack of re-optimization after the LD/CD was initiated. However, the temporary response to non-motor fluctuations after apomorphine subcutaneous injection therapy led us to initiate LDp/CDp. Hence, verification of a patient's responsiveness to any rescue therapy or levodopa-challenging test is necessary for patients with a poor response to prior pharmacotherapy or DBS adjustment before initiating "secondary DAT". Furthermore, responsiveness to rescue therapy using apomorphine subcutaneous injection could potentially improve not only non-motor fluctuations but also symptoms such as dysphagia and gait/motor disturbance.

Subcutaneous delivery of the water-soluble prodrug bypasses the variable gastrointestinal absorption that underlies levodopa resistance in aPD; more stable plasma levodopa concentrations may in turn stabilize pharyngeal and laryngeal motor control, a mechanism consistent with the videofluoroscopic improvements reported by Hirano et al. [13]. The improvement in swallowing ability observed in the present case is consistent with that study. In patients with aPD, impaired drug absorption via gastrointestinal dysmotility is a major factor in levodopa treatment resistance. Considering the perspective of administration routes, LDp/CDp delivers a water-soluble prodrug subcutaneously, thereby circumventing the variable of gastrointestinal absorption. This is of particular therapeutic significance in situations such as the present case, in which oral intake was insufficient to sustain nutritional requirements. LCIG or LDp/CDp has also been reported to improve gait disturbances [12]; thus, similar to dysphagia, this finding is strongly associated with a differential drug delivery system compared to that of conventional therapies.

Regarding safety, infusion-site reactions and neuropsychiatric vulnerability are the most frequently reported adverse events of LDp/CDp [4]. These adverse events have been associated with infusion dose, poor nutritional status, higher LED, older onset patients, or cognitively impaired patients [16,17]. Furthermore, the post hoc analysis in the previously mentioned 52-week trial reported a higher frequency of severe treatment-emergent adverse events in patients with prior DBS (45.8% vs. 23.6%), suggesting that the threshold for adverse-event surveillance should be lowered in patients with prior DBS or high disease severity [1]. Several notable features were observed in the present case, and careful dosage adjustment was required. Starting from a low initial dose and transitioning to small incremental doses when using

dopaminergic drugs can lead to successful initiation, maintenance, and management of adverse events in LDp/CDp therapy. This case highlights that considering the patient's entire clinical course is important for LDp/CDp management in high-risk cases.

However, this report has several limitations that affect its applicability and generalizability. First, this was a single, unblinded case report. Second, the observation period was insufficient to assess the long-term effectiveness of LDp/CDp. Finally, the contributions of concomitant therapies, including DBS, other medications, and rehabilitation, were not separately evaluated. Previous case reports have described improvements in gait or postural alignment following multimodal non-pharmacological interventions in patients with PD [18-20]. The walker-assisted ambulation achieved in the present case therefore cannot be attributed to LDp/CDp infusion alone and may reflect the combined effects of stabilized dopaminergic stimulation, rehabilitative training, and other concomitant therapies. Further case reports and prospective studies with adequate sample sizes and longer follow-up periods are needed to evaluate the effectiveness of LDp/CDp in patients with aPD many years after DBS and to distinguish its effects from those of concomitant therapies.

Conclusions

Herein, we report a patient with aPD eight years after initial DBS, in whom LDp/CDp was effective for dysphagia and gait/motor disturbance parallel with motor and non-motor fluctuations. LDp/CDp therapy may be useful for improving QOL and could serve as a potential treatment option for patients with aPD (H&Y stage V) with secondary DBS failure. Accumulating reports and prospective studies with large sample sizes and longer observation periods are required to validate these findings, which will ultimately lead to improved patient care and outcomes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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