of 7 cases.[9] Blink reflexes are reported to be abnormal in cases of cranial dystonia and blepharospasm.[10,11] Autopsy and neurophysiological/imaging findings coupled with consistent reports demonstrating successful treatment of dystonia with DBS would suggest that indeed the brainstem-basal ganglia-thalamocortical circuits are impaired in Meige syndrome. The biophysics of DBS are also unclear. Our patient responded much better to monopolar stimulation than to bipolar. One might speculate that the broader current diffusion of monopolar stimulation may affect circuitry outside the GPi itself leading to beneficial clinical results, at least in this case.

Bilateral GPi DBS may be an effective and safe treatment for select patients with primary Meige syndrome who obtain disappointing results from conventional treatment. This is the second report in the literature showing substantial improvement in a patient with isolated Meige syndrome. Further studies and long-term follow-up are needed. It would also be beneficial to study DBS in other dyskinetic disorders involving the lower face such as tardive dyskinesia and orobuccal lingual dystonia, because even botulinum toxin is often of limited use in these conditions.

LEGENDS TO THE VIDEO

Segment 1. Preoperative: baseline Meige's syndrome.
Segment 2. Postoperative: after programming, 4 weeks. Facial dysknesia is almost completely resolved.

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Open-Label Pilot Study of Levetiracetam (Keppra) for the Treatment of Levodopa-Induced Dyskinesias in Parkinson's Disease

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Abstract: **We evaluated the tolerability and preliminary efficacy of levetiracetam (LEV; Keppra) in reducing levodopa-induced dyskinesias in Parkinson's disease (PD) in an open-label pilot study. Nine PD patients who were experiencing peak-dose dyskinesias for at least 25% of the awake day and were at least moderately disabling were treated with LEV in doses up to 3,000 mg for up to 60 days. The primary outcome measure was the percent of the awake day that patients spent *on* without dyskinesia or with nontroublesome dyskinesia (good *on* time). The mean dose of LEV at endpoint was 625 ± 277 mg/day. LEV significantly improved percent of the awake day *on* without dyskinesia or with nontroublesome dyskinesia at endpoint compared to baseline (43% ± 12% vs. 61% ± 17%; $P = 0.02$). Percent *on* time with troublesome dyskinesia decreased from 23% ± 10% at baseline to 11% ± 6% at endpoint, although not significantly. There was no significant increase in *off* time from baseline to endpoint. There was a 56%**

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dropout rate, mostly due to somnolence. In PD patients who experienced peak-dose dyskinesia for at least 25% of the awake day, LEV significantly improved *on* time without dyskinesia or with nontroublesome dyskinesia. © 2005 Movement Disorder Society

Key words: levetiracetam; Keppra; dyskinesia; Parkinson's disease; levodopa

Parkinson's disease (PD) is a neurodegenerative disorder that is caused by a loss of dopamine-producing neurons in the nigrostriatal pathway.[1] Although motor symptoms of the disease respond to dopaminergic medication, long-term therapy with levodopa often causes dyskinesia.[2] Dyskinesia may cause disability, be uncomfortable or embarrassing, or limit the physician's ability to increase medications to improve symptoms or reduce *off* time.[3]

Levetiracetam [LEV; (S)-alpha-ethyl-2-oxo-1-pyrrolidine acetamide; Keppra; UCB Pharma, Smyrna, GA] is an antiepileptic drug that is approved as add-on therapy in the treatment of partial-onset seizures.[4] LEV reduced levodopa-induced dyskinesias in preclinical studies of animal models of PD.[5,6] Case reports and small open-label studies indicate that LEV may reduce various abnormal involuntary movements, including myoclonus,[7] paroxysmal kinesiogenic choreoathetosis,[8] and Meige's syndrome.[9] We sought to evaluate the tolerability and preliminary efficacy of LEV in reducing levodopa-induced dyskinesias (LID) in PD patients in a prospective open-label pilot study.

PATIENTS AND METHODS

Idiopathic PD patients who were followed at a university movement disorders center were invited to participate in the study during a 3-month recruitment period. The inclusion criteria were idiopathic PD, age 30 to 80 years, at least two of three cardinal signs of PD (bradykinesia, rigidity, or tremor), responsiveness to levodopa, and the presence of levodopa-induced dyskinesia. Further inclusion criteria included patients' use of 2 to 10 doses of levodopa daily at time of study entry, the presence of motor fluctuations, the presence of peak-dose dyskinesia for at least an average of 25% of the awake day as indicated on two diaries performed in the 4 weeks prior to study entry, and dyskinesia that was at least moderately disabling according to item 33 on the UPDRS.[10] Patients needed to take stable doses of antiparkinsonian medications for 4 weeks prior to study entry and for the duration of the study. Patients had to achieve at least a 70% concordance rate with the principal investigator (PI; T.A.Z.) for diary ratings of *on* and *off* time. Additional antiparkinsonian medications, including amantadine, dopamine agonists, selegiline, and anticholinergics were permitted during the study, as were prior surgeries for PD. Exclusion criteria included the concurrent or prior use of dopamine receptor antagonists; pregnancy or lactation; dementia (MMSE score less than 20); the presence of severe renal disease or blood urea nitrogen (BUN) 50% greater than normal; and major neurological, psychiatric, or medical disorders that were judged by the PI to disqualify a patient from entering the study. Patient diaries included five categories: asleep, *off*, *on* without dyskinesia, *on* with nontroublesome dyskinesia, and *on* with troublesome dyskinesia.[11,12] Patients who met inclusion and exclusion criteria provided informed consent to enter the study. Institutional review board (IRB) approval for the study was granted through the University of South Florida (USF).

Patients who entered the study were administered oral LEV in tablet form, beginning at a dose of 250 mg per day and increasing by 250 mg every 5 days to a maximum of 3,000 mg over 60 days. Study medication was titrated upward using a three times a day (t.i.d.) schedule to reduce the likelihood of somnolence from higher individual doses. Patients underwent evaluations at baseline and every 10 days thereafter during their optimal *on* time (best On time, approximately 1 hr following medication intake) and recorded their motor status with diaries completed each of the 2 days immediately prior to each follow-up evaluation. Dose adjustments were permitted at each office visit and further LEV titration could be halted with a patient report of satisfactory dyskinesia control or emergence of side effects. The primary endpoint measure was the change from baseline to endpoint in the percent of the awake day *on* without dyskinesia or with nontroublesome dyskinesia. These categories were analyzed together, as they represent good *on* time.[11,12] Secondary measures included percent of the awake day *on* with troublesome dyskinesia, percent of the awake day *off*, the Abnormal Involuntary Movements Scale (AIMS),[13] Unified Parkinson's Disease Rating Scale (UPDRS) total and motor subscore in the *on* state, the Clinical Global Impression of severity (CGI) Scale,[14] and the Epworth Sleepiness Scale (ESS).[15] The AIMS, UPDRS total and motor subscores, ESS, and CGI were obtained during patients' optimal *on* time or the best *on* time after the first daily dose of LEV. The CGI assessed the effects of LEV on levodopa-induced dyskinesia and on PD symptoms. One assessment was made for each scale at each visit. Data were analyzed using nonparametric paired samples analysis. The mean $\pm$ standard deviation (SD) is reported. Last observation carried forward (LOCF) was used for analyses for patients who prematurely withdrew from the study.

TABLE 1. *Patient characteristics*

	All subjects (n = 9)	Subjects who withdrew due to somnolence (n = 4)	Completers or dropouts due to reasons other than somnolence (n = 5)
Mean age	65	66	64
Gender (% male)	6/9 (67%)	2/4 (50%)	4/5 (80%)
Hoehn and Yahr stage (mean ± SD)	3 ± 0.87	3 ± 0.82	3 ± 1
MMSE (mean ± SD)	29 ± 2	29 ± 2	29 ± 2.3
UPDRS (total; mean ± SD)	54.7 ± 14.8	56 ± 8.3	53.6 ± 19.6
UPDRS (motor; mean ± SD)	30 ± 9.3	30.3 ± 5.9	29.8 ± 12.1
Duration of disease, yr (mean ± SD)	15.7 ± 10	23.0 ± 10.1	9.8 ± 5.1
Levodopa daily dose, mg (mean ± SD)	844 ± 444.0	963 ± 568	750 ± 357
ESS baseline score (mean ± SD)	10.4 ± 5.2	8.5 ± 4.1	12.0 ± 5.8
Other PD medications, n (mean daily dose, mg)			
Pramipexole	2 (1.9)	1 (2)	1 (1.9)
Ropinirole	4 (10.6)	2 (7.8)	2 (13.5)
Selegiline	1 (500)	1 (500)	0 (0)
Entacapone	6 (800)	2 (800)	4 (800)
Tolcapone	1 (600)	0 (0)	1 (600)
Amantadine	1 (100)	1 (100)	0 (0)
Trihexyphenidyl	0 (0)	0 (0)	0 (0)
Prior surgeries for PD (N)	0	1-pallidotomy	0

RESULTS

Three women and six men with PD were enrolled in the study (mean age, 65 ± 11 years; mean Hoehn and Yahr stage, 3 ± 1; Table 1). There was a 56% dropout rate by study endpoint. Two patients withdrew from the study before completing postbaseline diaries and are therefore not included in the primary efficacy analysis. However, ESS data were obtained from these patients prior to dropout and are included in the safety analysis. One patient withdrew due to somnolence and the other due to obtundation (dose = 250 mg/day each). Two additional patients dropped out of the study at days 11 and 39 due to somnolence, and one patient discontinued the study at day 14 because of dizziness and confusion (Table 2).

The mean dose of LEV at endpoint was 625 ± 277 mg/day (range, 375–1,000 mg/day). None of the patients who reached study endpoint (n = 4) reached the maximum allowable dose of 3,000 mg/day. Two patients experienced somnolence that prevented them from titrating LEV upward (one patient experienced somnolence at day 30 while taking 1,000 mg/day; the other patient experienced somnolence at day 40 while taking 500 mg/day). Two patients reached satisfactory dyskinesia control while taking 750 mg/day and 1,000 mg/day, respectively. Mean percent *on* time without dyskinesia or with nontroublesome dyskinesia increased from 43% ± 12% at baseline to 61% ± 17% at endpoint ($P = 0.02$). Percent *on* time with troublesome dyskinesia decreased from 23% ± 10% at baseline to 11% ± 6% at endpoint, although not significantly ($P = 0.13$). There was no increase in *off* time (31% ± 18% at baseline vs. 27% ± 17% at endpoint; $P = 1.0$) and no significant change in the AIMS score from baseline to endpoint (9.7 ± 4.5 vs. 6.7 ± 5.7; $P = 0.45$; Table 3). There was a trend for improvement in total UPDRS scores from baseline to endpoint (56 ± 16 vs. 47 ± 20; $P = 0.13$). Motor subscores on the UPDRS did not change significantly (29 ± 10 at baseline vs. 24 ± 10 at endpoint; $P = 0.22$). Dyskinesia improved as assessed by the CGI scale in six patients (marked improvement in one patient, moderate improvement in two patients, minimal improvement in three patients, and no change or worsening of dyskinesia in one patient). PD symptoms were much improved in two patients, unchanged in three patients, minimally worse in one patient, and much worse in one patient.

TABLE 2. *Dropouts*

Patient no.	Day	Reason	Daily LEV dose, mg
1	2	Somnolence	250
2	2	Obtundation	250
3	11	Somnolence	500
6	39	Somnolence	1,250
9	14	Dizziness and confusion	250

The mean ESS score did not change significantly from baseline to endpoint for the entire group (10.4 ± 5.2 vs. 13.1 ± 7.4; $P = 1.0$; n = 9). However, patients who dropped out of the trial due to somnolence or obtundation had a mean baseline ESS score of 8.5 ± 4.1 and a mean endpoint score of 14.3 ± 8.0 (n = 4; $P = 0.13$). Patients who completed the trial or dropped out for reasons other than somnolence had a mean baseline ESS score of 12.0 ± 5.8 and a mean endpoint score of 12.2 ±

TABLE 3. *Changes from baseline to endpoint with levetiracetam treatment*

	Baseline (mean ± SD)	Endpoint (mean ± SD)	*P*
ON time without dyskinesia or with nontroublesome dyskinesia (n = 7)	43% ± 12%	61% ± 17%	0.02
ON time with troublesome dyskinesia (n = 7)	23% ± 10%	11% ± 6%	0.13
OFF time (n = 7)	31% ± 18%	27% ± 17%	1.0
AIMS (n = 7)	9.7 ± 4.5	6.7 ± 5.7	0.45
UPDRS (total; n = 7)	56 ± 16	47 ± 20	0.13
UPDRS (motor subscale; n = 7)	29 ± 10	24 ± 10	0.22
ESS (n = 9)	10.4 ± 5.2	13.1 ± 7.4	0.29

7.7 ($P = 1.0$). Patients who withdrew from the study due to somnolence or obtundation tended to have a longer mean disease duration (23.0 ± 10.1 vs. 9.8 ± 5.1 years; $P = 0.10$) than those who did not drop out of the study or who dropped out for another reason. The mean daily levodopa dose was not significantly different between the groups (963 ± 568 mg for patients who withdrew due to somnolence vs. 750 ± 357 mg for patients who completed the study or who withdrew due to another reason; $P = 0.72$). The number of hours asleep per 24-hr period did not change significantly from baseline to endpoint for the seven patients who completed postbaseline diaries (9.5 ± 1.7 vs. 9.5 ± 2.3; $P = 1$). Two of the nine patients withdrew from the study before completing postbaseline diaries and are not included in this analysis.

DISCUSSION

In this open-label pilot study of LEV for the treatment of levodopa-induced dyskinesia, LEV increased percent good *on* time (*on* time without dyskinesia or with nontroublesome dyskinesia) by 42% without significantly changing *off* time. Percent *on* time with troublesome dyskinesia decreased by 52% and AIMS scores decreased by 45%, but these changes were not significant. There was an improvement in dyskinesia as assessed by the CGI in six of nine patients and a trend for improvement in total UPDRS scores.

There was an overall dropout rate of 56% with somnolence occurring in 44% of patients despite a slow titration schedule. The recommended dosing for LEV initiation in epilepsy patients is 500 mg twice daily, with titration by 1,000 mg every 2 weeks up to a maximum daily dose of 3,000 mg.[16] Although patients in our study received only one-quarter of the recommended initial dose and were titrated over a much longer period of time, they experienced a high drop rate due to somnolence. The mean baseline ESS score for all patients was elevated, but there was no significant change in the mean ESS score at endpoint. Of note, ESS scores were normal at baseline in the subgroup of patients who dropped out of the study due to somnolence and worsened by 5.75 points at study termination ($P = 0.13$). This finding suggests that patients who were not sleepy at baseline may have been more sensitive to the soporific effects of LEV than those patients who had baseline somnolence and who may have been more acclimated to it. Somnolence is associated with PD itself[17–19] and antiparkinsonian medications.[20,21] These factors may make PD patients more susceptible to LEV-induced somnolence than the epileptic population. The use of dopamine agonists did not appear to affect the dropout rate, although the numbers of patients taking these medications was low.

In a small open-label pilot study such as this one, any positive change could potentially be due to placebo effect, and this study is limited by the small sample size, a substantial dropout rate, and the subjective nature of the primary outcome measure. In addition, two patients had prior interventions (a pallidotomy in one and amantadine use in the other) that could have influenced dyskinesia and confounded the results. We also cannot exclude the possibility that somnolence caused the observed decrease in dyskinesia.

The pathophysiology of levodopa-induced dyskinesia is unknown, but may involve abnormal striatal glutamatergic receptors.[22] *N*-methyl-D-aspartate (NMDA) receptor antagonism improves levodopa-induced motor complications in animal models of PD,[23] and amantadine, a noncompetitive NMDA receptor antagonist, reduces levodopa-induced dyskinesia in PD patients and animal models of PD.[24] One preclinical animal model found that LEV potentiated the antidyskinetic action of amantadine in MPTP-lesioned marmosets.[25] The antidyskinetic benefit of amantadine in humans has not been found to be long-lasting.[26] Only one patient in our study took amantadine concomitantly with LEV, and patients who were not taking it had not tried it previously.

Our open-label pilot study indicates that LEV may be effective in treating levodopa-induced dyskinesia, although the poor tolerability of the dosing regimen limited study results. There is a need for further tolerability studies using LEV in the PD population. Future studies

may start with lower initial doses and utilize slower titration schedules to reduce somnolence associated with LEV use.

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Frequency of Movement Disorders in an Ethiopian University Practice

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Abstract: **There is little information on the frequency of movement disorders seen by physicians in the continent of Africa. We performed a medical record review of all patients seen in a university-based neurology clinic in Addis Ababa, Ethiopia, over 1 year to determine the frequency of movement disorders seen, disease characteristics, diagnostic evaluations, and treatment. A total of 15.1% of the neurological patients were seen for movement disorders. Of these, most were for parkinsonism (47.7%), followed by ataxia (16.5%), dystonia (8.3%), essential tremor (8.3%), chorea (7.3%), and miscellaneous (11.9%). Diagnostic eval-**

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