UNIVERSITY MOVEMENT DISORDERS CLINIC

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REQUEST FOR PRIOR AUTHORIZATION

Deep Brain Stimulation Surgery

ADMINISTRATIVE INFORMATION

Submission Date:	October 8, 2025
Procedure Requested:	Bilateral Subthalamic Nucleus (STN) Deep Brain Stimulation
CPT Codes:	61863 (bilateral leads), 61868 (IPG insertion)

PATIENT IDENTIFICATION

Name:	DAVIS, Sandra Marie
Date of Birth:	November 23, 1958 (Age 66 years)
Sex:	Female
Medical Record Number:	SMC-884562
Insurance:	Medicare Advantage - UnitedHealthcare
Member ID:	UHC998877665

DIAGNOSIS

Primary Diagnosis:	G90.3 - Multiple System Atrophy, Parkinsonian type (MSA-P)
Alternative consideration:	G20 - Parkinsonism, atypical features
Duration of Symptoms:	6 years (onset 2019)

CLINICAL HISTORY AND PRESENTATION

Ms. Davis is a 66-year-old female presenting with a 6-year history of progressive parkinsonism. Symptoms began in 2019 with bilateral hand tremor and slowness of movement. Initial evaluation by community neurologist resulted in diagnosis of Parkinson's disease and treatment with carbidopa-levodopa was initiated.

Over the subsequent 6 years, patient has experienced rapid progression of symptoms despite escalating medication doses. She has developed additional neurologic symptoms beyond typical Parkinson's disease, raising concern for atypical parkinsonian syndrome.

Parkinsonian Motor Features Present:

- Bradykinesia: Severe generalized slowing, symmetric bilaterally
- Rigidity: Lead-pipe type rigidity (not cogwheel), bilateral upper and lower extremities
- Postural instability: Severe, with multiple falls (15-20 falls in past year)
- Minimal tremor: Only mild postural tremor, no classic resting tremor

Atypical Features Suggesting MSA:

1. Autonomic Dysfunction (Prominent and Early):

- **Orthostatic Hypotension:** Severe symptomatic orthostatic hypotension documented on autonomic testing. Supine BP: 145/85, Standing BP (3 min): 95/60 (drop of 50/25 mmHg). Patient experiences frequent presyncope, lightheadedness, and has had 3 syncopal episodes in past year.
- **Urinary Dysfunction:** Severe urinary urgency, frequency, and incontinence beginning within first 2 years of motor symptoms. Urodynamic studies show detrusor hyperreflexia. Patient requires absorbent undergarments. Post-void residual volumes elevated (150-200cc).
- **Erectile Dysfunction:** Patient reports complete loss of erectile function (relevant as autonomic marker, though patient is female - documentation error, should be bladder dysfunction emphasis)
- **Constipation:** Severe, requiring daily laxatives

2. Cerebellar Signs:

- Gait ataxia: Wide-based, unsteady gait beyond what would be expected from parkinsonism alone
- Limb ataxia: Mild dysmetria on finger-to-nose testing bilaterally
- Scanning dysarthria noted in speech

3. Other Red Flags for Atypical Parkinsonism:

- Rapid progression: Hoehn & Yahr Stage 4 within 6 years (faster than typical idiopathic PD)
- Symmetric onset and progression (idiopathic PD typically asymmetric)
- Early and severe postural instability with falls
- Minimal tremor (tremor-dominant PD not present)
- Dystonia: Anterocollis (forward neck flexion) and camptocormia (forward trunk flexion)

MOTOR EXAMINATION

Examination Date: September 28, 2025

General Appearance: Patient seated in wheelchair. Notable anterocollis and camptocormia (forward flexion of neck and trunk). Facial expression shows mild hypomimia but less than typical for PD severity.

UPDRS Part III Motor Score:

State	Score
OFF medications (>12 hours)	58 / 132
ON medications (60 min after C/L 25/250 x 3 tabs)	52 / 132
Improvement	**6 points (10% improvement only)**

Hoehn and Yahr Stage: Stage 4 (severe disability; still able to walk or stand unassisted, but severely disabled; requires walker for short distances) **Detailed Motor Findings:**

- **Bradykinesia:** Severe, symmetric bilaterally. Finger tapping shows marked slowing and decrement, similar on both sides.
- **Rigidity:** Lead-pipe rigidity (not cogwheel pattern) throughout all extremities. Symmetric. Score 3/4.
- **Tremor:** Minimal. Mild postural tremor only. No classic resting tremor of PD.
- **Postural Instability:** Severe. Pull test: patient unable to recover, would fall if not caught. Retropulsion prominent.
- **Gait:** Wide-based, short-stepped, unsteady. Requires walker. Freezing episodes present but also has ataxic quality suggesting cerebellar involvement.
- **Speech:** Hypophonic with scanning, irregular quality (cerebellar component)
- **Axial Posture:** Marked anterocollis and camptocormia

Cerebellar Examination:

- Finger-to-nose: Mild bilateral dysmetria
- Heel-to-shin: Ataxic bilaterally
- Rapid alternating movements: Slow (parkinsonian) but also irregular (cerebellar)

Cranial Nerves: No supranuclear gaze palsy. No apraxia of eyelid opening. Facial expression mildly reduced but not severely masked. Voice quality has scanning component.

LEVODOPA RESPONSIVENESS ASSESSMENT

Acute Levodopa Challenge Test (September 28, 2025):

Protocol: Patient held all antiparkinsonian medications for 12 hours overnight. Baseline UPDRS Part III performed in OFF state. Patient then given carbidopa-levodopa 25/250 mg x 3 tablets (750 mg levodopa supratherapeutic dose). Serial examinations performed at 30, 60, 90, and 120 minutes.

Results:

Time Point	UPDRS Part III Score	Change from Baseline
Baseline (OFF)	58	--
30 minutes	56	-2 (3% improvement)
60 minutes	52	-6 (10% improvement) - PEAK

90 minutes	54	-4 (7% improvement)
120 minutes	56	-2 (3% improvement)

Clinical Observations During Challenge: Patient reported feeling "slightly less stiff" at peak but no dramatic improvement in mobility. Bradykinesia minimally improved. Rigidity decreased slightly. Postural instability unchanged. Gait ataxia unchanged. Autonomic symptoms unchanged. No clear, robust "ON" period observed.

Interpretation: POOR LEVODOPA RESPONSE. Only 10% improvement in UPDRS at peak dose - well below the threshold typically seen in idiopathic Parkinson's disease (typically >30-50% improvement). No clearly defined "ON" periods. This poor levodopa response is highly suggestive of atypical parkinsonism rather than idiopathic PD.

MEDICATION HISTORY

Medication	Maximum Dose Attempted	Duration	Response
Carbidopa-Levodopa	25/250 mg QID (1000 mg/day levodopa)	6 years	Minimal benefit; no robust response even at high doses
Pramipexole	4.5 mg daily	5 years	No significant benefit; caused worsening orthostatic hypotension
Rasagiline	1 mg daily	4 years	No appreciable benefit
Amantadine	100 mg TID	3 years	Minimal to no benefit
Midodrine	10 mg TID	2 years	For orthostatic hypotension; partial benefit
Fludrocortisone	0.1 mg daily	2 years	For orthostatic hypotension; partial benefit

Medication Summary: Despite trials of multiple dopaminergic medications at appropriate doses, patient has demonstrated consistently poor response. The lack of robust levodopa benefit, even at high doses, strongly suggests this is not idiopathic Parkinson's disease. Additionally, patient requires medications specifically for autonomic dysfunction (midodrine, fludrocortisone), which is uncommon in typical PD at this stage.

DIAGNOSTIC STUDIES

Brain MRI with and without contrast (August 15, 2025):

T2/FLAIR sequences show atrophy of the pons and middle cerebellar peduncles. The putamen demonstrates T2 hypointensity bilaterally with a hyperintense rim laterally ("putaminal rim sign"). Moderate cerebellar atrophy present, particularly affecting the vermis and cerebellar hemispheres. Midbrain appears relatively preserved without significant atrophy. These findings are consistent with Multiple System Atrophy.

No acute stroke, hemorrhage, or mass lesion. No basal ganglia stroke or vascular malformation.

DaTscan (March 2023):

Reduced striatal dopamine transporter uptake bilaterally and symmetrically. Pattern shows decreased uptake in both caudate and putamen with relative preservation of caudate:putamen ratio (differs from typical PD pattern). Findings consistent with presynaptic dopaminergic deficit but does not distinguish between idiopathic PD and atypical parkinsonian syndromes.

Autonomic Function Testing (July 2025):

Formal tilt table testing documented severe orthostatic hypotension (supine BP 150/90, upright BP after 3 min: 90/55). Abnormal heart rate variability. Quantitative sudomotor axon reflex test (QSART) showed reduced sweating response. Findings confirm severe autonomic dysfunction.

Urodynamic Studies (May 2025):

Detrusor hyperreflexia with incomplete bladder emptying. Post-void residual volumes 150-250 cc. Findings consistent with neurogenic bladder dysfunction.

NEUROPSYCHOLOGICAL ASSESSMENT

Evaluation by Dr. Jennifer Martinez, PhD (September 15, 2025):

MOCA: 26/30 (within normal limits)

Comprehensive neuropsychological battery shows mild frontal-executive dysfunction but overall cognition preserved. No dementia. Patient demonstrates adequate understanding of medical information and decisionmaking capacity.

Psychiatric Evaluation: No active depression or psychosis. Mood appropriately low given disease burden. No substance abuse.

MULTIDISCIPLINARY EVALUATION

Movement Disorder Specialty Team Assessment:

Patient case extensively reviewed by multidisciplinary team including movement disorder neurologists, neurosurgeon, and autonomics specialist. Team consensus based on clinical features:

- Parkinsonism with poor levodopa response (<15% improvement)
- Prominent, early autonomic failure (orthostatic hypotension, urinary dysfunction)
- Cerebellar signs (ataxia, dysmetria, scanning speech)
- Rapid progression to severe disability (Stage 4 within 6 years)
- Symmetric presentation
- MRI findings: putaminal rim sign, pontocerebellar atrophy

Clinical Diagnosis: Multiple System Atrophy, Parkinsonian type (MSA-P)

Patient meets diagnostic criteria for probable MSA-P based on consensus criteria (Gilman et al., 2008). This is an atypical parkinsonian syndrome classified as a "Parkinson-Plus" syndrome.